

Vertigo – a practical approach

Vertigo is a relatively common presentation in general practice.

J Grobbelaar, MB ChB, MMed, FCS Otorhinolaryngology

ENT Private Practitioner, Medi Clinic, Bloemfontein

Dr Grobbelaar completed his undergraduate studies at the University of the Free State (UFS) in 1997. He did his internship in Bloemfontein and community service in Potchefstroom. He completed his ENT studies in 2006 under the great Professor A J Claassen at UFS. Dr Grobbelaar stayed on as a consultant for three years, of which one year was in Cork, Ireland, and went into private practice in 2009. His interests include all three major branches of ENT, although head and neck surgery is his favourite.

Correspondence to: J Grobbelaar (jgrobbelaar1@gmail.com)

Part I. History and examination

‘There can be few physicians so dedicated to their art that they do not experience a slight decline in spirits when they learn that their patient’s complaint is giddiness. This frequently means that after exhaustive enquiry it will still not be entirely clear what it is that the patient feels wrong and even less so why he feels it.’¹

This is especially true in a busy practice. My aim is to provide some knowledge of basic anatomy and physiology, but, more importantly, a stepwise approach to a dizzy patient. Of course it is very easy to over-complicate or over-simplify this topic. However, by the end of this article you should be able to distinguish vertigo from other forms of dizziness. You will also be able to differentiate vertigo further into peripheral and central pathologies and advise the correct treatment

options. It is estimated that approximately 90% of individuals over 65 years of age have visited their physician at least once with vertigo as their primary complaint. Complex cases should, however, be identified and referred to a specialised unit.

Anatomy and physiology

Each labyrinth consists of the vestibular apparatus and the cochlea. The vestibular apparatus can be divided into three semi-circular canals and the otolith organ, which is composed of the utricle and saccule. (Fig. 1.)

The semi-circular canals register angular rotations, and the otolith organs register linear changes and gravitational direction. The information from the ears, along with visual and proprioceptive

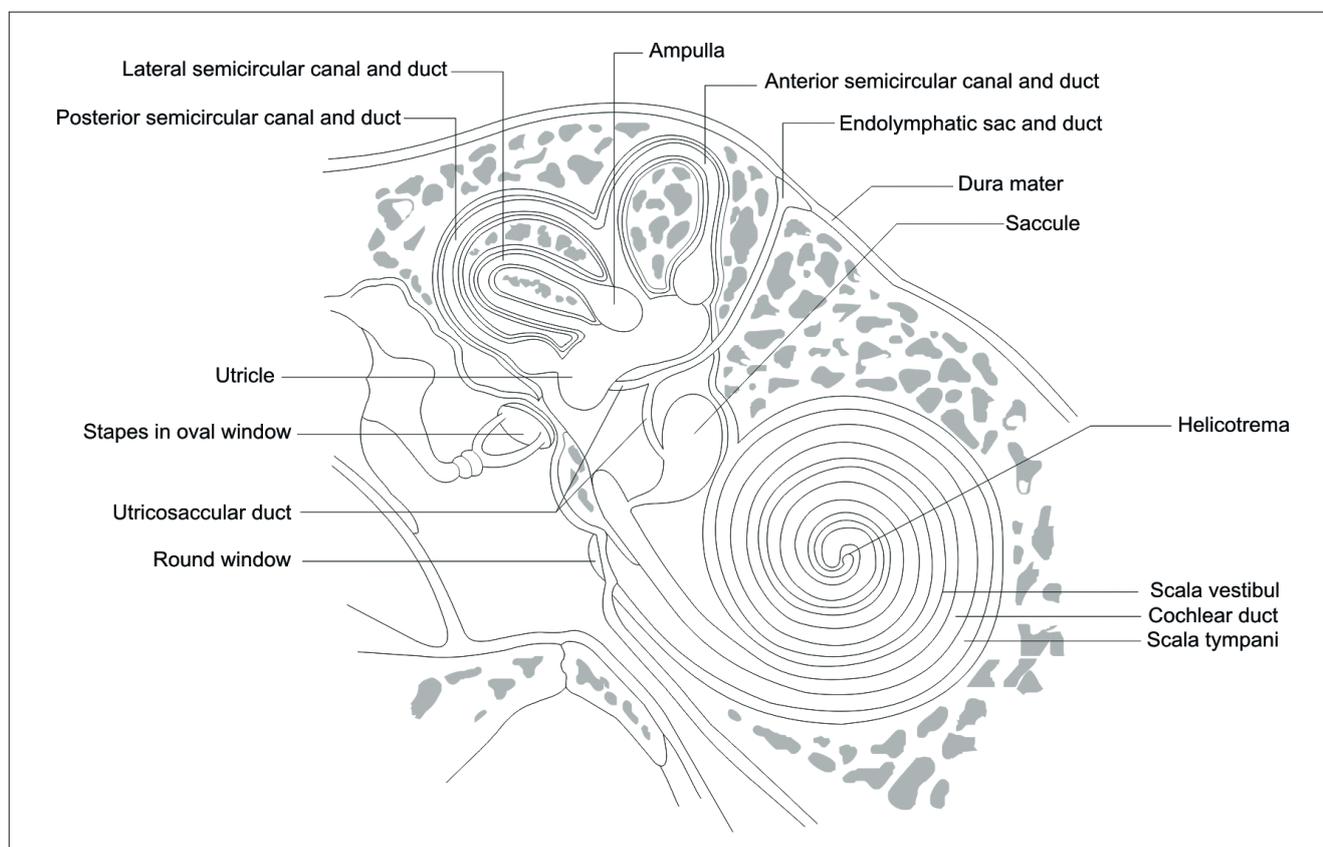


Fig. 1. Inner-ear anatomy.

Vertigo

information, forms the major input to the balance system, as depicted in Fig. 2.

The vestibular end organs are dynamic structures. They are not silent until stimulated but constantly discharge at a resting pattern of signals to the brain. The two sets (right and left) of vestibular apparatus are mirror images of one another. With any acceleration/deceleration movement, opposite but equal change in firing rate takes place on the contralateral side. The cerebral cortex interprets the change in firing rate as movement in a specific direction and of a specific speed. The vestibular nuclei have important connections to various regions in the brain, but three of the most important are to the ocular nuclei, cerebellum and spinal tracts. In response to vestibular stimulation, the eyes move in the opposite direction to retain the field of last gaze, otherwise known as a reduction in retinal slip, which prevents blurred vision (Fig. 3). This allows for movement without becoming dizzy or feeling off-balance while still maintaining clear vision, and is the basis of the vestibular-ocular reflex (VOR). Examination of the VOR is very important in the dizzy patient.

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The vestibular-spinal tracts adjust trunk and limb muscles and the cerebellum adjusts muscle tone to meet the new situation. Over time the brain has learned exactly what to expect from the vestibular organs. This is partly instinctive but is mostly learned as a baby learns to walk and the specific sections of the nervous and musculoskeletal system mature. The vestibular organs are, therefore, in constant dynamic balance, one checking the other and working as a team.

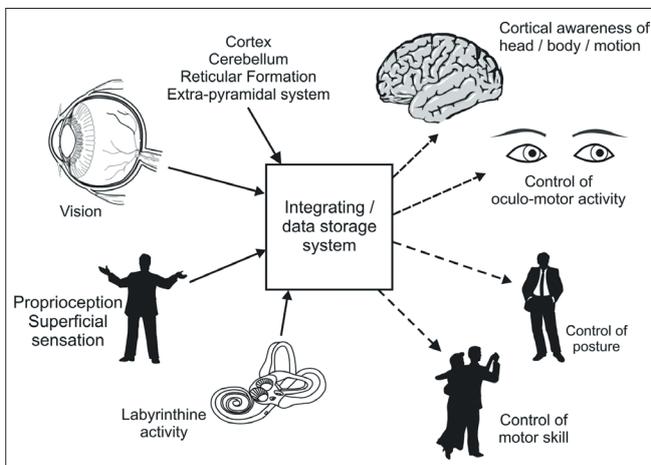


Fig. 2. Input and output of vestibular system.

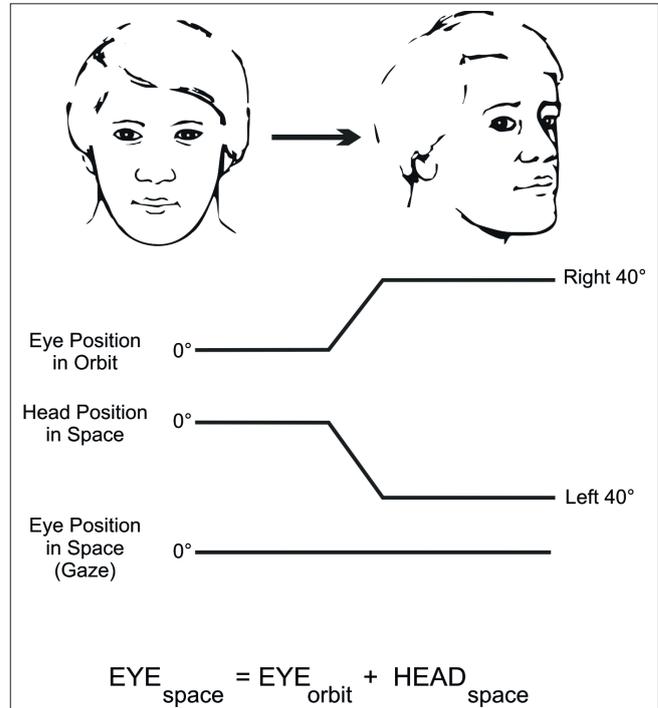


Fig. 3. Vestibular-ocular reflex.

Disease strikes

With a sudden pathological dysfunction of one vestibular organ (mostly hypofunction), the two sides discharge at unequal rates. This unbalanced information is interpreted by the brain as a condition of constant motion. This is the basis of our definition of vertigo, i.e. an illusion of movement which will be rotatory in most cases, but can be described as pitching, yawing or rolling in character. The same unbalanced information arrives at the ocular nuclei and spinal tract. The eyes move in response to the stimulation to the last field of gaze and the slow phase of nystagmus is seen. Because the eyeball cannot turn 360°, it reaches a point of maximal deviation and a signal from the reticular formation returns the eyes to their starting position, which forms the fast phase of nystagmus. This explains the basis of vestibular nystagmus. The same unbalanced response in the spinal tract causes staggering and ataxia.

In a matter of minutes the cerebellum imposes a shutdown of the unbalanced information from the vestibular nuclei. This alleviates, but certainly does not eliminate, the immediate problem. Fortunately, over a few days the spontaneous nystagmus and vertigo abate because of plasticity and adaptation within the central nervous system, but the asymmetric VOR persists as long as the impaired labyrinth remains depressed. Using this explanation there are two important rules: (i) a vestibular crisis of any severity will cause vestibular nystagmus; and (ii) if the symptoms last continuously for more than 2 - 3 weeks, the cause is not vestibular.

Bilateral vestibular loss will cause severe oscillopsia with minimal vertigo. Oscillopsia refers to a sensation of bobbing up and down

Vertigo

and blurred vision when moving. These patients tend to make slow head movements, turning head and body together to avoid this sensation.

In contrast, unilateral loss of otolith input causes postural instability and deviations in upright stance. During acute loss, the head tilts and the body leans towards the impaired side because of loss of extensor tone on the involved side. Because proprioceptive input is intact, the patient can still stand. Over time the deviation lessens and the patient shows little sign of injury. With bilateral loss of otolith input, the patient is deprived of his/her internal sense of gravity. He or she becomes more dependent on proprioceptive and visual cues and has trouble walking in the dark or on unpredictable surfaces (sand, grass, inclines). This disability persists, although most patients learn to ambulate quite well under most conditions.

The ability to maintain a quiet stance is driven mainly by proprioceptive input. Pressure receptors in the soles of the feet and joint stretch receptor information in the feet, legs, trunk, and neck, all combine to create a rich network of multilevel subcortical and cortical reflex pathways designed to maintain the body's centre of gravity over its base of support at the feet. During vestibular dysfunction, this system becomes more important to make up for loss of labyrinthine information regarding gravity. Diseases such as peripheral neuropathies and corticospinal degeneration interrupt these pathways and create difficulties with posture control and ambulation.

Approach to a patient

There are some excellent articles on the examination of dizzy patients.^{2,4} I would urge all general practitioners to read through these, as they explain specific tests in more detail.

Many specialised units include a questionnaire, or a standardised sheet to complete (Appendix 1). A basic outline is as follows:

- Questionnaire or standardised sheet
- History
- Examination
 - general
 - head and neck
 - ear, nose and throat (ENT)
 - neuro-otological
 - nystagmus
 - fixation – gaze straight
 - fixation – gaze in different positions
 - without fixation
 - central oculo-motor signs
 - smooth pursuit
 - saccade
 - vergence
 - visual fixation/gaze holding
 - optokinetic nystagmus
 - VOR battery
 - Dix Hallpike and lateral semi-circular canal testing
 - dynamic visual acuity test (DVAT)

- head thrust/head impulse test
- head shake test
- caloric test
- cranial nerves
- co-ordination
- posture
- gait
- special examinations.

History

A diagnosis can be made on history alone in 70% of patients. Physical examination and special tests will contribute 10 - 20%, respectively.

The key history points are:

- Is it vertigo?
- What is the time course?
- What are the precipitating/exacerbating factors?
- Are there accompanying symptoms?

Vertigo can be an illusion that the external world is moving relative to an individual or the individual relative to space. Rotational vertigo or other illusionary sensations of motion indicate vertigo (vestibular symptoms), whereas a sensation of light-headedness, giddiness, drowsiness, or impending fainting implies dizziness of non-vestibular origin. Non-spinning dizziness when standing or walking usually indicates a neurological gait problem rather than vestibular vertigo.

Vertigo onset is usually sudden and comes in spells varying from seconds or minutes to hours. The offset is less clear, with patients feeling unwell for a variable time. Some peripheral vertigo is brought on by a change in position and most patients will improve by lying still. Tinnitus, hearing loss, and aural fullness frequently accompany peripheral disease.

Unlike peripheral vertigo, central causes of dizziness produce a more variable picture. Patients may describe it as spinning, tilting, forced to one side, light-headedness, clumsiness or blacking out. If loss of consciousness is documented, a peripheral aetiology for dizziness is rarely – if ever – the reason. The following symptoms also point to a central cause, i.e. dysarthria, dysphagia, diplopia, hemiparesis, severe localised cephalgia, seizures or memory loss.

Some specific questions in the history can point to diagnosis:

- Do you become dizzy just rolling over in bed?
 - Benign paroxysmal positional vertigo (BPPV)
- Are you light sensitive during your dizzy spell?
 - Vestibular migraine
- Does one ear feel full before or during an attack?
 - Meniere's disease
- Does a loud sound make you dizzy or make your world jiggle?
 - Superior semi-circular canal dehiscence
- Did your first attack of severe vertigo last hours, with nausea and vomiting?

- Vestibular neuritis
- Are you light-headed when you get up from a bed or chair for a few seconds?
 - Blood pressure/cardiovascular disease (CVS)
- Do you pass out completely with your dizziness?
 - CVS

Examination

As mentioned above, general, ENT and neuro-otological examinations are done. This article focuses mainly on the neuro-otological examination.

First look for nystagmus with fixation with the gaze straight and then in different positions. Repeat the same without fixation, usually using Frenzel glasses (20+ dioptre). Lastly, look for nystagmus in positional testing (discussed below). Peripheral nystagmus will cause a uni-directional fixed nystagmus, which will increase when looking in the direction of the fast phase and with loss of fixation. In almost all cases it will be horonto-rotatory with the eye in the neutral position. All other forms point to a central problem such as direction changing, disconjugate, seesaw, pendular, congenital and gaze nystagmus. Purely vertical and/or torsional nystagmus always points to a central problem.

With a sudden pathological dysfunction of one vestibular organ (mostly hypofunction), the two sides discharge at unequal rates.

Next, examine specific eye movements – oculo-motor testing. In smooth pursuit testing ask the patient to track your finger/a light in the horizontal and vertical planes. Make sure not to exceed 40°/s and more than a 60° arc. As a general rule horizontal smooth pursuit is better than vertical and both will diminish with age. Next ask the patient to track back and forth between two fingers about 20 cm apart without moving their head. This is known as saccade testing. Observe the eyes for either under- or over-correction and also conjugate movement. Abnormalities in smooth pursuit or saccade testing point to a central problem. The other oculo-motor tests are not discussed in this article.

The VOR battery of testing then follows. Dix Hallpike and lateral semi-circular canal testing are tests specifically aimed at diagnosing the different forms of BPPV. Because posterior canal BPPV causes more than 90% of problems, the Dix Hallpike test is discussed in more detail (Fig. 4). With the patient sitting up in bed the head is turned 45° to the side and the patient is brought into a supine position with the head just hanging over the edge of the bed. This is not a simultaneous movement and there is also no need to do this quickly. The patient is instructed to keep the eyes in the neutral position and, on instruction by the physician, to the left and

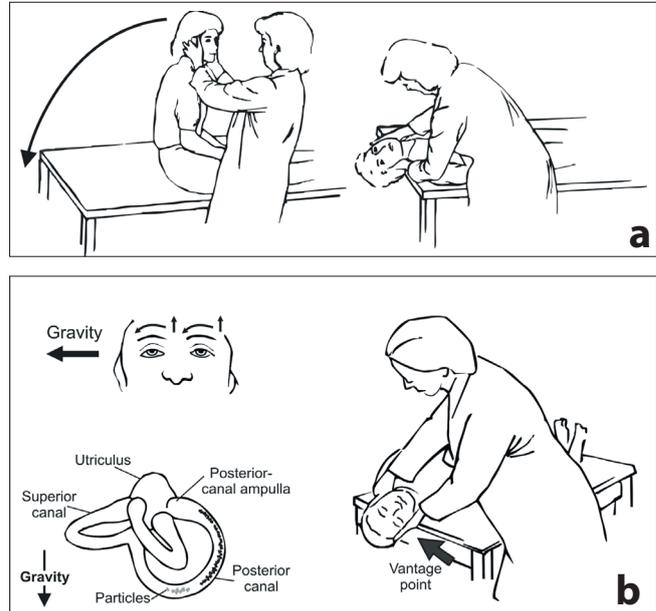


Fig. 4. Dix Hallpike test for BPPV.

right. Observe the eyes using Frenzel glasses and note any type of nystagmus. In posterior canal BPPV there is usually a brief latency, followed by a geotropic (beating towards the ground) vertical-rotatory nystagmus lasting less than 60 seconds. Sometimes there is a reversal of the nystagmus pattern when coming up again. Lateral canal testing is done with the patient lying prone and then turning the head to the left and right. Again observe for any nystagmus using Frenzel glasses. Because of the different variations associated with lateral canal BPPV, it is best to refer these patients.

DVAT is an extremely useful test to confirm a peripheral lesion. With the best corrected vision the patient reads the smallest line possible on a Snellen eye chart (handheld is acceptable). The procedure is repeated, with the examiner shaking the patient's head at 2 Hz and recording the number of lines lost during headshake. A drop of more than two lines indicates a bilateral vestibular loss or a poorly compensated unilateral loss.

The head thrust test is also known as the head impulse test (HIT). This test has become one of the most important bedside tests for the evaluation of VOR. The patient is instructed to look at the examiner's nose while he quickly turns the patient's head randomly around the horizontal axis between 20° and 30°. Normally, patients will have no problem keeping their gaze on the nose. With unilateral vestibular loss the VOR will fail to keep the gaze on the nose and there will be a catch-up saccade. A patient who presents with acute vertigo and has a normal HIT always points to a central problem. The HIT is illustrated in Figs 5 and 6.

In the head shake test tilt the patient's head 30° forward and shake at 2 Hz for 20 seconds. Observe the eyes with Frenzel glasses for any abnormal eye movements. A peripheral problem will cause post head

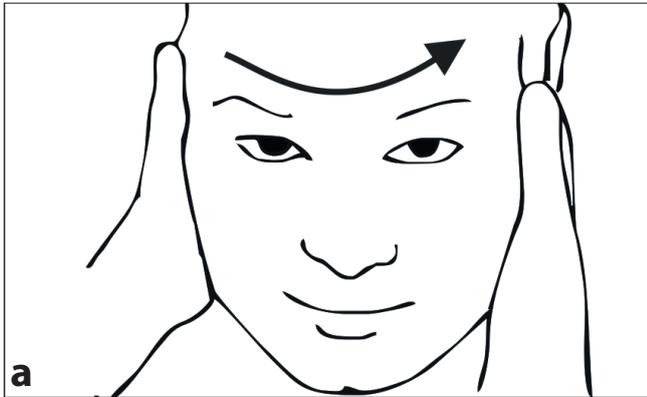


Fig. 5. Normal HIT. When turning the head to the left, the eyes stay focused on the examiner's nose.

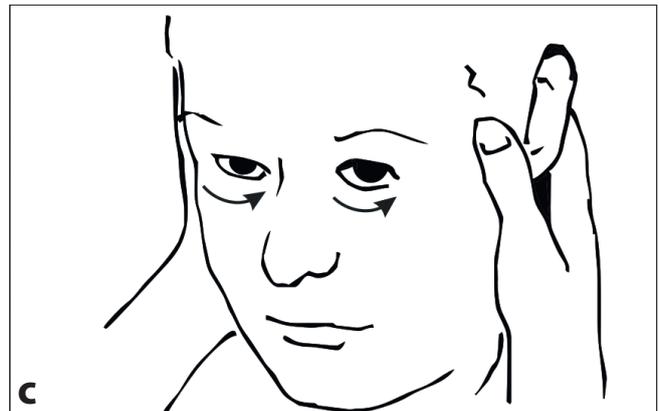
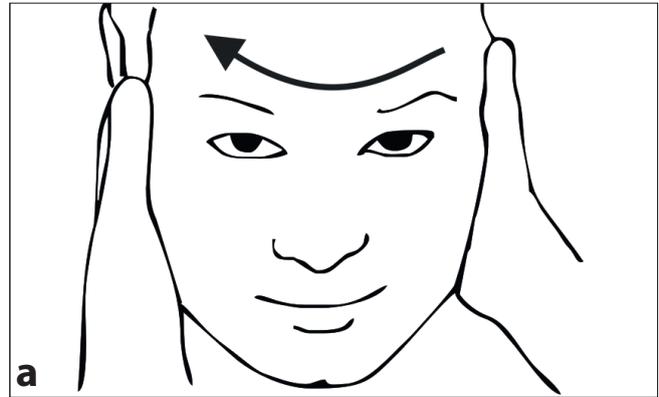


Fig. 6. Abnormal HIT. When turning the head to the right, the eyes follow the movement of the head, and a corrective saccade is necessary to fixate on the examiner's nose.

shake nystagmus with a small reversal component. Central problems may cause prolonged, disconjugate or cross coupling nystagmus (vertical nystagmus following horizontal shaking).

Caloric testing stimulates mainly the lateral semi-circular canal. Various irrigating regimens are described, but I suggest using the mini-caloric test. In this test, tap water is used to irrigate the ear for 10 seconds in summer and 8 seconds in winter. The most important aspect of caloric tests is to visualise the tympanic membrane when irrigating. Frenzel glasses are used and the duration and amplitude of nystagmus are recorded. The caloric test is very important because this is the only vestibular test that can test one side at a time. The acronym COWS stands for cold water opposite, warm water same side, indicating the direction of nystagmus produced with water.

Lastly, the Romberg test is primarily for somatosensory function and proprioception, not vestibular input. There are, however, two variations to make it more sensitive for vestibular input. First instruct the patient to do a tandem stance Romberg and secondly to do a Romberg test while standing on 10 cm foam. Observe for any sway with the patient's eyes open and then closed.

The other neurological tests such as cranial nerves examination, cerebellar function, posture and gait are not discussed here, but are

very important. Specialised tests include other vestibular testing, radiology, audiology and blood tests.

After the examination one can distinguish three main groups of patients. Firstly, the group with definite peripheral vertigo will have positive signs with some of the following tests: Dix Hallpike test, lateral semi-circular canal (SCC) test, caloric test, HIT, and DVAT. Patients with central vertigo will have positive signs with some of the

Vertigo

Table 1. Difference between peripheral and central vertigo

	Peripheral	Central
Hallucinations of movement	Definite	Less definite
Onset	Usually paroxysmal	Seldom paroxysmal
Intensity	Usually severe	Less severe
Duration	Seconds to hours	Weeks to months
Induced by head position	Frequently	Seldom
Nystagmus	Present	Present or absent
Nystagmus pattern	Uni-directional, increased by loss of fixation	Direction changing, no change with fixation, other forms of nystagmus
Autonomous nervous system symptoms	Definite	Less definite or absent
Tinnitus	Frequently present	Seldom present
Hearing loss	Frequently present	Seldom present
Disturbance of consciousness	Absent	More frequently present
Other neurological signs	Usually absent	Frequently present

Table 2. Differential diagnosis of vertigo

Peripheral	Central
<p>Common</p> <ul style="list-style-type: none"> • BPPV • Vestibular neuritis • Meniere's disease • Bilateral vestibulopathy • Vestibular schwannoma/acoustic neuroma <p>Rare</p> <ul style="list-style-type: none"> • Superior semi-circular canal dehiscence (SSCD) • Vestibular paroxysmia (vascular loop compression) • Perilymph fistula • Labyrinthitis • Auto-immune inner-ear diseases <p>Other problems</p> <p>Physiological/pathological stimulation</p> <ul style="list-style-type: none"> • Motion disease • Caloric stimulation <ul style="list-style-type: none"> • Water exposure • Wind exposure • Rotational stimulation <ul style="list-style-type: none"> • Flying • Driving • Pressure changes • Changes in specific gravity <ul style="list-style-type: none"> • Alcohol-induced vertigo 	<p>Common</p> <ul style="list-style-type: none"> • Phobic postural vertigo • Vestibular migraine • Pathological forms of nystagmus, e.g. <ul style="list-style-type: none"> • Down-beat nystagmus • Up-beat nystagmus • Gaze nystagmus <p>Rare</p> <ul style="list-style-type: none"> • Central positioning vertigo • Dizziness syndromes of unclear aetiology/familial • Episodic ataxia type II • Arnold-Chiari malformation • Psychogenic dizziness <p>Central nervous system diseases/causes</p> <ul style="list-style-type: none"> • Multiple sclerosis • Vascular disease • Tumours • Epilepsy • Infections • Medications

Vertigo

following tests: smooth pursuit, saccades, and cerebellar tests. Lastly patients with dizziness will have none of the above or sometimes bizarre combinations.

The difference between peripheral and central vertigo is summarised in Table 1.

Part II. Clinical diseases Differential diagnosis

Note: vertigo does not imply peripheral disease; it can also be from a central cause. Vertigo does imply some form of illusion of movement, mostly spinning. All other symptoms are grouped under dizziness.

After taking a history and doing an examination the clinician needs to decide the following:

- Is it peripheral labyrinthine, eighth nerve, central vestibular disease, or diffuse?
 - If labyrinthine, is it unilateral or bilateral?
- Is this acute isolated, acute recurrent, or chronic disease?
 - If central, is it brainstem or cerebellar disease?

Three typical forms of peripheral vestibular dysfunction can be identified, based on their characteristic symptoms and signs:

1. Acute or subacute unilateral vestibular failure, characterised by rotational vertigo, oscillopsia, and a tendency to fall toward the affected ear – usually acute vestibular neuritis.
2. Bilateral peripheral vestibular failure (bilateral vestibulopathy), characterised by instability of gait and posture, and oscillopsia induced by head movement.
3. Paroxysmal peripheral vestibular stimulation or inhibition, characterised by attacks of vertigo and oscillopsia, e.g. in BPPV, Meniere's disease, and vestibular paroxysmia.

The patient who is having a first attack of acute spontaneous vertigo may have acute vestibular neuritis or a cerebellar infarction. These patients are commonly misdiagnosed in the casualty department as having 'labyrinthitis' or 'middle-ear infection'. Differentiating between the two can be difficult. Acute vestibular neuritis will cause a uni-directional nystagmus pattern that improves with fixation, produces a positive head impulse test and mostly the patient will be able to stand alone, in contrast to cerebellar infarction in which they tend to fall when walking. Brainstem ischaemia symptoms such as diplopia, reduced vision, dysarthria, and dysphagia will be present in less than 50% of patients with cerebellar infarction.

A differential diagnosis for the most common causes of vertigo is presented in Table 2.

A differential diagnosis for dizziness is presented in Table 3.

Looking at a clinical correlation it is easy to follow the schematic description below.

- Is it vertigo?
 - Yes
 - Rotatory
 - Vestibular neuritis
 - BPPV
 - Meniere's disease
 - Vestibular migraine
 - Sensation of boat
 - Bilateral vestibulopathy
 - No
 - Dizziness
- Duration
 - Seconds to minutes
 - BPPV
 - Vestibular paroxysmia
 - Minutes to hours
 - Meniere's disease
 - Vestibular migraine
 - Hours to days
 - Vestibular neuritis
 - Varies
 - Fistula
 - SSCD
- Precipitating/exacerbating factors
 - Present in rest
 - Vestibular neuritis
 - Worse when walking
 - Bilateral vestibulopathy
 - Precipitated by turning the head to the left and right
 - Vestibular paroxysmia
 - Turning in bed to one side
 - BPPV
 - Coughing/pressing/sounds
 - Fistula
 - SSCD
 - Social or environmental condition
 - Phobic postural vertigo
- Accompanying symptoms
 - Inner ear
 - Tinnitus, hearing loss, aural fullness
 - Central nervous system
 - Diplopia, dysphagia, sensory disturbances, dysarthria, paralysis of arms or legs
 - Headache
 - Vestibular migraine

BPPV

BPPV is the most common cause of vertigo and affects mainly older women. It can follow after vestibular neuritis, head trauma, prolonged bed rest and Meniere's disease, but more than 90% of cases are idiopathic. It is caused by otolith crystals becoming dislodged and floating in a semi-circular canal. As the name implies this is a benign condition with spontaneous resolution in weeks; however,

Table 3. Differential diagnosis of dizziness

Dizziness

Condition	Causes
Pre-syncope/syncope	<ul style="list-style-type: none"> • Arrhythmias • Reduced cardiac output • Hypovolaemia • Pericarditis • Orthostatic hypotension • Autonomic dysfunction • Vasovagal syncope • Hyperventilation • Hypoglycaemia
Central nervous system	<ul style="list-style-type: none"> • Normal pressure hydrocephalus • Posterior fossa tumours • Primary orthostatic tremor
Cardiovascular system	<ul style="list-style-type: none"> • Blood pressure • Vascular diseases
Metabolic	<ul style="list-style-type: none"> • Glucose metabolism • Thyroid hormone production
Medication	<ul style="list-style-type: none"> • Blood pressure medication • Antibiotics – aminoglycoside • Chemotherapy • Psychotropic medication • Tranquillisers
Proprioceptive/somatosensory	<ul style="list-style-type: none"> • Neck disease • Peripheral neuropathy
Eye	<ul style="list-style-type: none"> • Poor vision • Double vision
Psychogenic	

30% of cases will persist. As mentioned above, posterior canal BPPV is the most common variant (90%) and is discussed in more detail. It is characterised by brief attacks of vertigo after turning in bed, lying down in bed, looking up or bending down. The vertigo usually lasts for less than 1 - 2 minutes and patients will typically lie still. Some patients confuse the time frame of the vertigo versus feeling unwell following the vertigo, but a careful history should clear this up.

It is diagnosed by doing a Dix Hallpike test as described above. A corrective turning manoeuvre is done to move the crystals out of the affected canal. There are two manoeuvres, i.e. the Semont or Epley. Both are equally effective with cure rates of more than 95%. The Epley manoeuvre (Fig. 7) is a turning manoeuvre with 1 - 2-minute intervals at certain positions. The patient's head is kept in the same position that the nystagmus was seen after the Dix Hallpike test for 2 minutes. The head is turned 90° towards the opposite side; wait another 2 minutes. The patient is then turned on their side, with head turning a further 90° in the same direction. The patient will be facing the ground, and typically will experience vertigo again (which is a good sign). After 2 minutes instruct the patient to keep

their chin as far as possible on their shoulder and bring them into an upright position.

Afterwards the patient must not make any movements that can trigger the BPPV again, such as lying down, or any up/down movements, for 10 days. They must not lie on the affected side or lie completely flat for 3 days. Some centres instruct their patients to do the manoeuvres at home. Unfortunately the recurrence rate of BPPV is 15 - 30% per year.

Tinnitus, hearing loss, and aural fullness frequently accompany peripheral disease.

Vestibular neuritis

Vestibular neuritis is the second most common cause of peripheral vertigo after BPPV. It is characterised by acute/subacute sustained horizo-rotatory nystagmus with oscillopsia, imbalance with falls, and nausea. The nystagmus is suppressed by visual fixation and there is a pathological HIT. An otherwise normal neurological examination and the ability to stand unassisted without lateropulsion are important in distinguishing vestibular neuritis from a brainstem bleed or infarction. There is no hearing loss associated with vestibular neuritis, in contrast to labyrinthitis, which is extremely rare.

There is sometimes a history of upper airway infection and evidence suggests that vestibular neuritis is caused by reactivation of a latent herpes simplex virus type 1.⁵⁷

Although the onset of vestibular neuritis is more predictable, the chronic course of the disease is more variable. In most cases, the initial episode will resolve, and the patient experiences disequilibrium while ambulating and momentary dizziness with rapid head turns, which can last up to three months. Some patients (10%) experience repeated episodes of severe vertigo much like the initial episode. BPPV may follow vestibular neuritis.

Treatment is based on the following:

- supportive treatment
 - IV fluids
 - anti-emetic agents
- vestibular sedatives for 3 days
 - diazepam
- causative treatment
 - methylprednisolone 100 mg/d and reduced every fourth day by 20 mg
 - valacyclovir 1 g tds po for 7 days
 - Unfortunately the administration of antivirals alone or in combination with steroids has no additional benefits if given after 48 hours.
- improved vestibular compensation

Vertigo

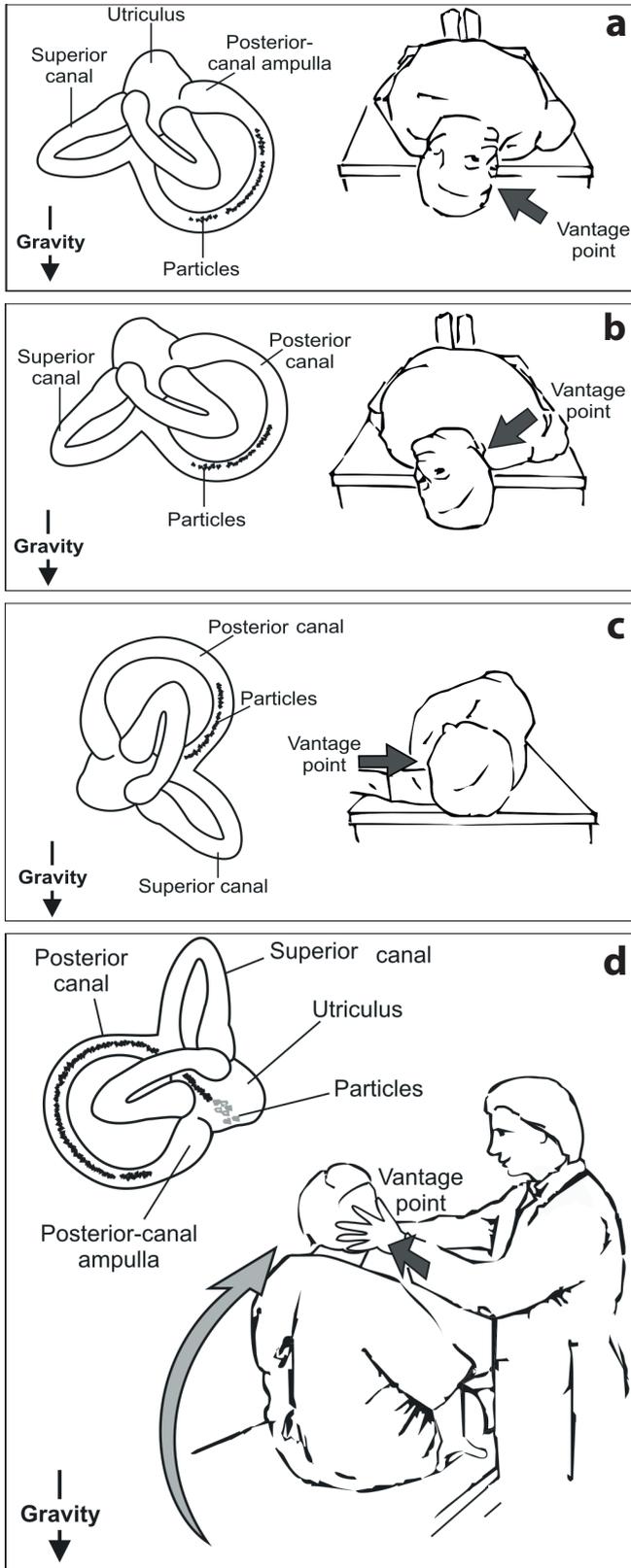


Fig. 7. Epley manoeuvre for a right-sided posterior semi-circular canal BPPV.

- There is some evidence to support the use of high-dose betahistidine (Serc®). Unfortunately the dose of 48 mg tds po is impractical and expensive in South Africa.^{5,8}
- vestibular exercises (30 minutes 3 times per day).

The question always arises whether or not to scan a patient. In a straightforward case there is no need to do a scan. However, more often than not one is confronted with an atypical case or a possible cerebellar infarction. These patients should be scanned, but also referred to a specialised unit.

Meniere's disease

The diagnosis of Meniere's disease is based on a combination of symptoms, i.e. episodic vertigo lasting minutes to hours, fluctuating low-tone hearing loss which becomes permanent, tinnitus, and aural fullness.

The diagnosis may be difficult to make as the classic picture may take years to develop. In contrast to the classic picture, there are also two variants, i.e.:

- cochlear variant
 - fluctuating hearing loss, tinnitus and fullness with no vertigo
- vestibular variant
 - episodic vertigo with no hearing loss, tinnitus or fullness.

Treatment consists of medical, surgical and lifestyle modifications. Because of the difficulty in diagnosing the patients, and the various options available, it is best to refer these patients.

Bilateral vestibulopathy

As mentioned above, these patients have minimal vertigo, but present with postural imbalance, broad-based gait, increased gait variability, and oscillopsia. In fact, it is the most common cause of postural imbalance in the elderly. The symptoms will always worsen when the patient needs to rely on the vestibular organs such as closing the eyes, dark environment or walking on uneven ground. On examination there will be an abnormal HIT and Romberg test with the eyes closed. In three-quarters of patients one is unable to identify a cause. Known causes are antibiotics, Meniere's disease, meningitis, and encephalitis. The treatment consists of balance training.

Vestibular schwannoma/acoustic neuroma

This is a rare tumour of the vestibular nerve. In most cases it is sporadic, but there is a genetic component in 5% of cases. It presents with hearing loss (95%), tinnitus (70%), imbalance (50%), and vertigo (20%). In severe cases it can cause brainstem and cranial nerve V and VII symptoms. Any patient who presents with all three inner-ear symptoms, i.e. hearing loss, vertigo, and tinnitus, should be referred to an ear, nose and throat specialist.

Superior semi-circular canal dehiscence syndrome (SSCD)

As the name implies this syndrome is caused by exposure of the superior semi-circular canal to the cerebrospinal fluid (CSF)

surrounding the brain. SSCD is characterised by vertigo induced by noise/pressure, pulsatile tinnitus, autophony, and unilateral hearing loss/fullness. Sometimes a patient gives a history of chronic disequilibrium following minor head trauma. Examination and special examinations are complex, and it is best to refer these patients.

Vestibular paroxysmia

This is characterised by spontaneous attacks of vertigo lasting seconds to minutes. Attacks are mainly spontaneous but can be induced by hyperventilation, exercise, and head turn. It is assumed that vascular compression of the 8th nerve is the cause (as in trigeminal neuralgia, hemifacial spasm and superior oblique myokymia). The condition is treated with carbamazepine.

Phobic postural vertigo (PPV)

PPV is common, being the second most common diagnosis in a tertiary referral balance unit.⁵ The patients complain of swaying vertigo, light-headedness, and gait unsteadiness that are continually present but fluctuate in severity. Symptoms are often accompanied by anxiety and fear of falling, but without actually falling. This is usually followed by increasing avoidance behaviour, especially of large open spaces. Typically these patients have obsessive compulsive disorder (OCD) personalities.

In general the symptoms are worse during the day and are improved by taking moderate amounts of alcohol and exercise. These patients typically have symptoms when standing or walking, but as the balance task gets more difficult they improve.

A decoupling hypothesis explains the underlying mechanism. Patients are more aware of normal body movements/sway and interpret this as abnormal. Treatment consists of explanation, desensitisation, and selective serotonin re-uptake inhibitors (SSRIs) will help in a third of cases. In general three-quarters of patients will improve.

Vestibular migraine

Vestibular migraine is the most common cause of central vertigo.⁶ Up to one-third of all migraine patients have experienced vertigo. It presents with a combination of vertigo and ataxia of stance or gait. The vertigo usually lasts minutes to hours and rarely days. Sixty per

cent of patients have auras during vertigo, accompanied or followed by head pressure, pain, nausea, and vomiting. In some patients there is no correlation with a headache. Treatment consists of regular migraine medications.

Episodic ataxia type II

Episodic ataxia type II is characterised by vertigo attacks lasting hours and ataxia. It is often provoked by stress or exercise. It is an autosomal dominant disorder and treated with acetazolamide.

Down-beat nystagmus

Down-beat nystagmus is the most common form of central nystagmus. The leading symptoms are postural imbalance and oscillopsia, usually worse in the morning and improving during the day. On examination the patient presents with fixation nystagmus with an increase in intensity during lateral and downward gaze and when lying prone with the nose down.

Down-beat nystagmus is usually due to bilateral dysfunction of the flocculus of the cerebellum. Its three most common causes are cerebellar atrophy, ischaemia, and Arnold-Chiari malformation.

Gaze nystagmus

These patients are unable to sustain eccentric gaze. It is probably the most common form of acquired nystagmus. Known causes are a range of drugs such as anticonvulsants, benzodiazepines, alcohol, and midline cerebellar diseases.

Psychogenic forms of vertigo

The patient describes experiencing frequent postural imbalance or a diffuse feeling of dizziness (a feeling of numbness, light-headedness, unsteadiness when walking, toppling over) or very rarely, rotatory vertigo.⁹ Depending on the underlying psychiatric illness, the following additional symptoms may be present: disorders of motivation and concentration, decline in performance, restriction of daily and professional activities, vegetative symptoms (accelerated heart beat, nausea, sweats, apnoea, fear of suffocating, loss of appetite, weight loss), emotional and mood disorders, sleep disturbances and symptoms of anxiety. These patients are best referred to the appropriate discipline.

References available at www.cmej.org.za

IN A NUTSHELL

- The word vertigo implies a hallucination of movement, mostly a spinning sensation.
- Vertigo does not imply only peripheral disease, and the problem can be in the central connecting pathways.
- After you have confirmed the presence of vertigo, there are two important steps. Firstly try to determine the time period that the patient experiences the vertigo and not the unwell feeling. Secondly ask leading questions regarding the potential trigger factors such as rolling over in bed.
- During the examination look for spontaneous or induced nystagmus and always check for central oculo-motor signs.
- You need to be able to perform a Dix Hallpike manoeuvre, as this is used to diagnose BPPV.
- In the acute setting try to differentiate vestibular neuritis from a brainstem infarction.

Appendix 1

VERTIGO

History:

Main complaint:

First attack:

Specific questioning:

1. Is it vertigo?

Spinning: Direction of head turn:

Up/down: Direction of fall:

Sideways:

2. Other imbalance:

Unsteadiness: Unsure:

Floating: Other:

Pre-syncope:

3. Time:

Seconds: Minutes:

Hours: Days:

Varies: Other:

4. Induced by:

Position: Sound:

Pressure: Other:

Walking in the dark:

5. Otological localisation:

Tinnitus: Hearing loss:

Fullness: Pressure:

6. Autonomic symptoms:

Nausea: Vomiting:

7. Fixation:

Improves: Worsens:

No influence:

8. Severity:

No influence on activity: Impairment:

Lie still in bed: Function:

Other organ systems:

1. CNS

TIA: Arms: Legs: Dysphasia:

Diplopia: Stroke:

2. Headache:

Migraine: Other:

3. Eyes:

Vision: Surgery: Glasses: Other:

4. Neck:

Injuries: Surgery: Problems: Other:

5. CVS:

Arrhythmias: Hypertension: Orthostatic hypotension:

Other:

6. Respiratory

Hyperventilation: Other:

Vertigo

7. GI

Reflux disease:

Other:

8. Endocrine:

DM:

Hypoglycaemia:

9. Other:

Multisensory:

Bed rest:

10. Medications:

11. Other diseases:

12. Familial diseases:

Examination:

General:

Ears:

Nose:

Throat:

CNS:

Higher functions:

Motor:

Sensory:

Reflexes:

Gait:

Cranial nerves:

I:

II:

III, IV, VI:

V:

VII:

VIII:

IX, X:

XI:

XII:

Cerebellar functions

Dysdiadochokinesis:

Finger-nose:

Heel-shin:

Fast tongue movements:

Ataxia:

Dysmetria:

Pendular reflexes:

Romberg:

Nystagmus:

Vestibular:

Dix Hallpike:

Lateral SCC:

Caloric:

Head thrust:

Fistula:

Head shake:

Other:

Eye movements:

Nystagmus:

Spontaneous:

Induced:

Characteristics:

Saccades:

Smooth pursuit:

Fixation:

DVAT:

Other:

Audiology:

Diagnosis:

Special investigations:

Vestibular laboratory: