Central vs. peripheral vertigo – a guide to diagnosis

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Diagnosis of vertigo is complex. When a patient enters your practice with a complaint of vertigo or dizziness, it is imperative that you establish whether the vertigo is peripheral or central in origin. The former is generally benign and the treatment of peripheral conditions is on the whole within an osteopath's scope of practice. The latter can be potentially serious and warrants immediate referral. This article outlines an initial approach to diagnosis, and highlights the key issues practitioners need to consider when assessing patients. Further reading is advised for a more comprehensive account of individual tests and their interpretation to cover all possible peripheral and central vestibular conditions.

ANATOMY

Understanding central and peripheral vertigo requires a review of the anatomy of the vestibular system and its related structures to demonstrate what encompasses peripheral and central components.

Anatomy of the peripheral vestibular system

The peripheral vestibular system comprises:

- The inner ear consisting of the cochlea, the organ of hearing;
- The vestibular apparatus comprising:
  - The three semicircular canals that sense angular movements, such as rotations in all planes;
  - The otolith organs composed of the utricle and saccule that react to linear acceleration and deceleration;
- The eighth cranial nerve, the vestibulocochlear nerve, is also considered part of the peripheral nervous system.

Anatomy of the central vestibular system

The central vestibular system includes the brainstem and cerebellum. Ten of the 12 cranial nerves contain their nuclei in the brainstem. The focus here is on a portion of the eighth cranial nerve nuclei, the vestibular nuclei, which are situated in the pontomedullary junction. The vestibular nuclei, in turn, make connections with the oculomotor, abducens and trochlear nuclei via the medial longitudinal fasciculus (MLF). The vestibular system also sends information to the cerebellum, which coordinates the motor movements needed to maintain posture and balance. The vestibular nuclei and cerebellum connect via the thalamus to the vestibular cortex, but that doesn't concern us too much in this context.

Posterior circulation

The focus is on the posterior portion of the 'Circle of Willis' consisting of the vertebrobasilar system, particularly the labyrinthian or internal auditory artery, a branch of the anterior inferior cerebellar artery (AICA), which supplies all structures of the inner ear and can be compromised by any ischaemic event such as arteriosclerosis, haemorrhage or infarct (see Figure 1).

CASE HISTORY

The case history of a dizzy patient is of the utmost importance and contributes profoundly to the diagnosis. ***In 69% to 76% of dizzy patients, the diagnosis is primarily based on history*** (Büki & Tarnutzer, 2014). Additional tailored questions must therefore be added to the customary history:

- **Description and characteristic of the spell.** A patient often finds it difficult to describe his/her symptoms so it is important not to place too much emphasis on the description. Nevertheless, it can add information towards the diagnosis;
- **Onset of symptoms.** A description of the onset of symptoms is important and can add valid clues to achieving a diagnosis. For example:
  - Any preceding illness? (e.g. herpes simplex virus can cause vestibular neuritis);
  - Any preceding trauma? (e.g. falls, motor vehicle accident causing concussion or whiplash);
  - Acute vestibular syndrome (AVS) such as vestibular neuritis;
  - An abrupt onset favours a vascular cause;

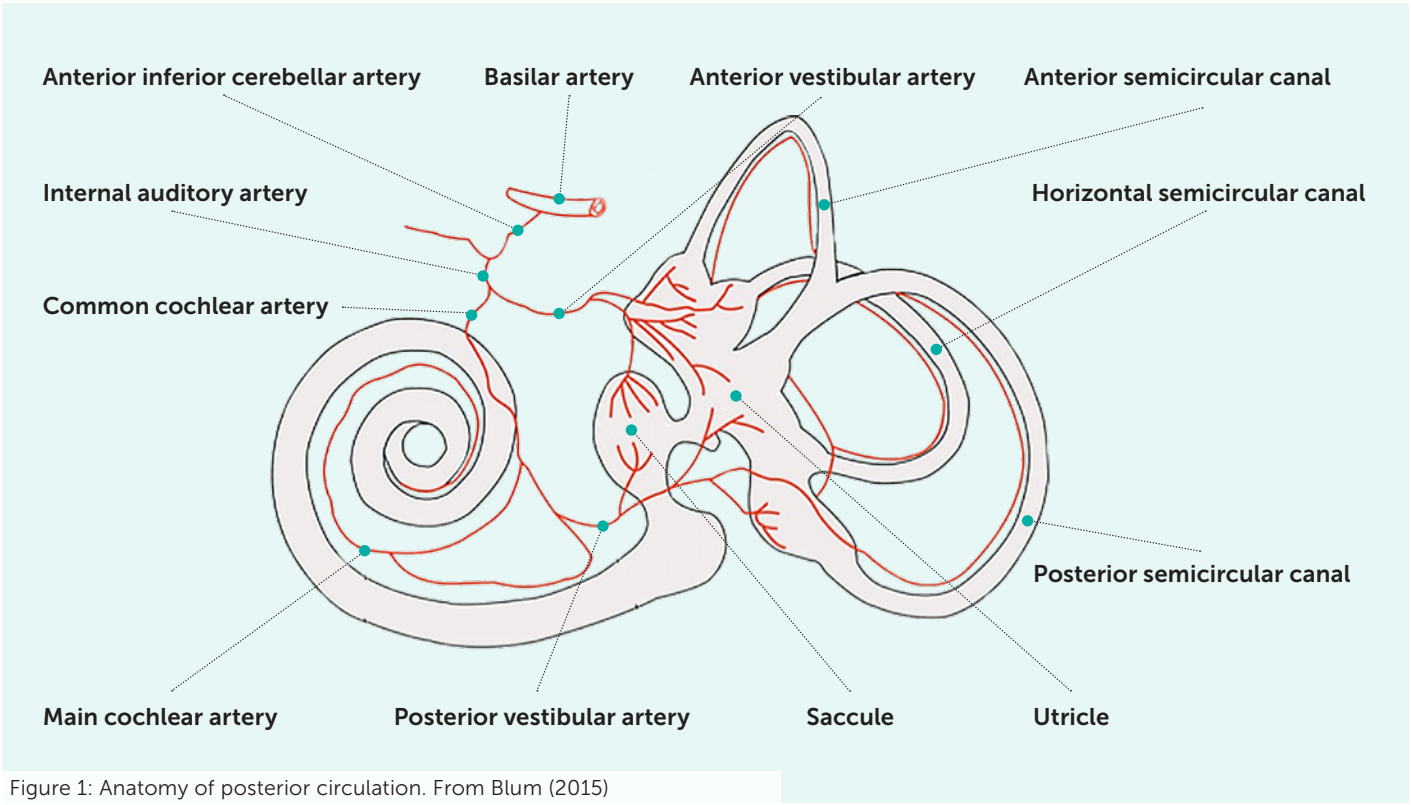

Figure 1: Anatomy of posterior circulation. From Blum (2015)

- More slowly evolving symptoms are encountered in the case of vestibular migraine;

Duration of symptoms. The duration of symptoms can add important clues to the history and diagnosis because most disorders present with a characteristic duration of episodes (see Table 1). For example, if the dizziness comes in spells, how long do the spells usually last?

Associated events and symptoms. The presence of associated symptoms additional to vertigo adds valuable information to the history. Specific events or symptoms and common conditions associated with vertigo (Table 2) are:

- Tinnitus;
- Hearing loss. ***An acute, sudden hearing loss always warrants further investigation and referral!***;
- Aural fullness;
- Visual changes;
- Falling (pulsion, tripping, poor balance);
- Headaches, migraine;
- Autonomic: nausea, vomiting, perspiration;
- Lightheadedness with body gravity changes;
- Psychological (fear, anxiety, depression).

TABLE 1: DURATION OF SYMPTOMS

DISORDER	DURATION
Benign paroxysmal positional vertigo (BPPV)	15 to 30 seconds
Panic attacks	10 to 20 minutes
Ménière's disease	2 to 3 hours maximum
Vestibular neuritis	24 to 48 hours
Vestibular migraine	Minutes to hours

Exacerbating factors and triggers. Finding out what exacerbates or triggers a patient's symptoms adds crucial information to the history because they are often characteristic for certain conditions (Table 3).

Focal neurological symptoms. Enquire about neurological symptoms other than vertigo; these may closely mimic benign vestibular disorders. ***However, take care because these symptoms can be missing in more than 50% of patients presenting with acute vestibular syndrome (AVS) due to ischaemic stroke*** (Büki & Tarnutzer, 2014). Symptoms may include:

- Double vision, difficulty swallowing, slurred speech, limb weakness, or numbness strongly support a central cause of dizziness/vertigo;

"The case history of a dizzy patient is of the utmost importance and contributes profoundly to the diagnosis"

TABLE 2: ASSOCIATED EVENTS AND SYMPTOMS

HEARING LOSS	AURAL FULLNESS	DROP ATTACKS	TINNITUS	AMPLIFICATION OF SOUND	HEADACHES
Ménière's disease (fluctuating)	Ménière's disease	Ménière's disease	Ménière's disease		Vestibular migraine
Labyrinthitis			Labyrinthitis		
Superior semicircular canal dehiscence		Superior semicircular canal dehiscence		Superior semicircular canal dehiscence	
Anterior inferior cerebellar artery infarction (acute loss)		Orthostatic hypotension			
		Vasovagal syncope			
		Cardiovascular			

- Brainstem signs, such as long tracts, cranial nerves and oculomotor abnormalities;
- Cerebellar signs such as nystagmus (which may be up/downbeating, direction changing or rebound) – see box below.

COMMON VESTIBULAR SYNDROMES AND FREQUENTLY ASSOCIATED AETIOLOGIES

A limited number of vestibular syndromes and underlying conditions cause most of the vestibular symptoms seen in patients (see Figure 2). Prioritising the recognition and treatment of urgent conditions, chiefly stroke, is imperative.

A key red flag symptom is a first-ever attack of acute spontaneous vertigo – AVS. This requires urgent referral to check for posterior circulation stroke.

If the vertiginous presentation is recurrent in nature, the aetiology is more commonly benign, albeit debilitating.

> "A key red flag symptom is a first-ever attack of acute spontaneous vertigo"

TABLE 3: EXACERBATING FACTORS AND TRIGGERS

TRIGGERS	CONDITION
Head or body positioning	Characteristic for benign paroxysmal positional vertigo (BPPV)
Standing up	Orthostatic hypotension
Loud sounds, increased pressure (Valsalva)	Superior semicircular canal dehiscence
Persistent positional nystagmus	Arnold-Chiari malformation, posterior fossa tumour
Triggers are typically absent	Ménière's disease, vestibular migraine
Quick head movements, busy environment	Oscillopsia in vestibular hypofunction
Intolerance of head movements	Acute vestibular syndrome (AVS)

Nystagmus

A key factor differentiating between central and peripheral vertigo is observing the nystagmus! Observing nystagmus in the vertiginous patient is paramount and adds invaluable diagnostic information in differentiating between central and peripheral vestibular conditions (see Table 4). This, therefore, warrants further attention. Nystagmus has a slow and a fast phase, and the direction of the nystagmus is determined by the fast phase. See Table 7 on page 21 for how to provoke or test and observe the nystagmus.

TABLE 4: FEATURES OF NYSTAGMUS OF CENTRAL AND PERIPHERAL ORIGIN

CENTRAL NYSTAGMUS	PERIPHERAL NYSTAGMUS
Changes direction	Never changes direction (unless positionally provoked)
Pure vertical nystagmus (downbeating)	Decreases with fixation
Does not decrease with fixation	Increases when looking toward the direction of the fast phase
Rebound nystagmus	Decreases when looking toward the direction of the slow phase

Recurrent vertigo can be further categorised by the presence or absence of positional provocation. Recurrent positional vertigo is often caused by benign paroxysmal positional vertigo (BPPV), while recurrent spontaneous vertigo is often secondary to vestibular migraine or Ménière's disease.

A presentation of generalised imbalance often points towards vestibular hypofunction (especially bilateral hypofunction) or ataxia. For those with chronic or recurrent symptoms, a change in pattern should prompt consideration of a new pathology, rather than simply an atypical presentation of a recurrent condition.

Acute vestibular syndrome or acute transient vestibular syndrome

A new significant headache or neck pain is concerning for cerebellar haemorrhage or vertebral artery dissection, respectively. Check for stroke symptoms such as facial/limb weakness or numbness, as well as posterior circulation stroke (PCS) symptoms, such as dysarthria, diplopia, dysmetria, dysphonia and dysphagia. Spontaneous vertical nystagmus, if seen, would indicate a central cause. An inability to walk unaided is also concerning for stroke.

While many conditions may lead to permanent disability over months or years, a disruption to the posterior circulation may cause immediate irreparable damage. The first-ever attack of acute spontaneous vertigo may represent PCS, especially in those with cardiovascular risk factors. When symptoms are present for more than 24 hours, it is termed AVS; if symptoms last for less than 24 hours, it is referred to as acute transient vestibular syndrome (ATVS). Despite this categorisation, a short duration of symptoms should not be inferred as low risk for an ischaemic cause, as PCS can frequently present with symptoms lasting only minutes. Associated symptoms can include nausea, vomiting, autonomic symptoms and head motion intolerance, although these are not specific to PCS.

PCS can be effectively and rapidly differentiated from an acute vestibular neuritis, the two most common aetiologies of AVS, using the HiNTS+ exam (see box on page 20), while transient PCS can be evaluated using the ABCD2 score (see Table 5).

"A new significant headache or neck pain is concerning for cerebellar haemorrhage or vertebral artery dissection, respectively"

Recurrent spontaneous vertigo

- Ménière's disease
- Vestibular migraine

Acute vestibular syndrome (AVS)/Acute transient vestibular syndrome (ATVS)

- Posterior circulation stroke (PCS)
- Vestibular neuritis/labyrinthitis

Recurrent positional vertigo

- Benign paroxysmal positional vertigo (BPPV)
- Central positional vertigo

Imbalance

- Vestibular hypofunction
- Ataxias cerebellar/sensory

Figure 2: Four common vestibular syndromes and their frequently associated aetiologies

HiNTS+ plus exam

The HiNTS+ plus exam is 99% sensitive and 97% specific for identifying a central cause of vertigo in patients with constant vertigo and nystagmus but should not be applied in patients unless they have both significant constant vertigo and spontaneous nystagmus. If the patient's symptoms are transient, ABCD2 scoring should be carried out (see Table 5).

The HiNTS+ exam comprises a series of four bedside tests:

- **H**ead **i**mpulse test (HIT). This involves carrying out a brief but rapid 15˚ to 20˚ lateral head turn. The HIT is performed by asking the patient to fix their gaze on your nose and then rapidly move the patient's head 20˚ off the midline and then back to the midline;
- **N**ystagmus. Gaze-evoked nystagmus (GEN);
- **T**est of **s**kew. This checks for vertical ocular misalignment by alternately covering each eye. In lesions involving the brainstem, vertical misalignment of the eyes may present (skew deviation). The alternate cover test can demonstrate this. This test involves asking the patient to look straight ahead and then alternately covering each eye at a time. If a skew deviation exists, a corrective vertical or oblique saccadic movement is observed;
- **+**Finger rub. Bedside test for hearing.

A unidirectional nystagmus, no vertical skew, an abnormal HIT and no new hearing loss suggests a 'peripheral' HiNTS+ result and confirms a diagnosis of vestibular neuritis.

In the case of vestibular neuritis, one side will demonstrate an abnormal 'catch-up saccade', where the eyes will overshoot your nose and then re-fixate on your nose. This is an abnormal HIT and not seen in cerebellar strokes. The counterintuitive finding with a normal HIT is therefore concerning for a stroke in a patient with constant vertigo and nystagmus.

If any of the four tests point to a central cause, the likelihood of a central aetiology such as PCS should be strongly considered.

HiNTS+ exam indicating a central cause of vertigo

- HIT is normal
- Direction-changing nystagmus GEN
- Test of skew reveals vertical ocular misalignment
- Finger rub reveals acute hearing loss

Features of a posterior circulation stroke

- Absence of a catch-up saccade on HIT (normal HIT)
- Central nystagmus patterns, including (but not limited to):
  - Spontaneous vertical
  - Pure torsional
  - Direction-changing – where the fast beat changes towards the direction of gaze
  - Skew deviation (a vertical misalignment of the eyes)

TABLE 5: ABCD2 STROKE RISK SCORING

An ABCD2 stroke risk score of 4 or higher indicates a central cause of vertigo

ABCD2	STROKE FINDINGS: RISK SCORE ≥4	SCORE
Age	≥60 years	=1
Blood pressure	Systolic ≥140 or diastolic ≥/90	=1
Clinical features	Unilateral weakness Speech disturbance no weakness Any other symptom	=2 =1 =0
Duration of symptoms	<10 minutes 10 to 59 minutes ≥60 minutes	=0 =1 =2
Diabetes	Present	=1

Recurrent positional vertigo

Recurrent positional vertigo is frequently caused by BPPV, which is the most common aetiology of vertigo. Otoconia from the otolith organs pathologically displace the cupula within the semicircular canals causing vertigo triggered by head movements such as rolling in bed or reaching for items from the floor or high shelves.

Diagnosis requires both symptoms and nystagmus of the corresponding canal during positional testing using provocation manoeuvres (e.g. the Dix-Hallpike test). After initiation of the test, the nystagmus will usually start with a latency of approximately two to 20 seconds after the head is in the provoking position and last for less than a minute.

Other specific features supporting BPPV include a reversal of the nystagmus direction when returning to the upright position, and fatiguability of the nystagmus

with repeated testing. The direction of the nystagmus is specific to the semicircular canal and is elicited by its respective provocation manoeuvre (see Table 7).

Recurrent spontaneous vertigo syndrome

While the first-ever bout of spontaneous vertigo raises concerns for a vascular aetiology, recurrent spontaneous vertigo is most often caused by either vestibular migraine or Ménière's disease. When experienced for the first time, these conditions will present as AVS, and despite their distinct characteristics and associated clinical features they can still cause diagnostic difficulties.

Vestibular migraine

Vestibular migraine is the second most common cause of vertigo after BPPV. Vertigo is two to three times more common in migraineurs and women are affected two to three times more often than men. The pathophysiology of vestibular migraine remains uncertain but is likely to be of central nervous system origin and an association between migraine and vestibular dysfunction has been widely acknowledged.

Vestibular migraine is characterised by recurrent episodes of vertigo, disequilibrium or balance impairment lasting from minutes to greater than 72 hours, except for chronic migrainous vertigo which may persist for six months or more (Table 8).

TABLE 6: COMMON CAUSES OF CENTRAL AND PERIPHERAL VERTIGO

CENTRAL VERTIGO	PERIPHERAL VERTIGO
Posterior fossa infarct (79%)	Vestibular neuritis
Vertebrobasilar transient ischaemic attacks	Vestibular migraine
Arnold-Chiari malformation	Ménière's disease
Multiple sclerosis and neoplasm	Benign paroxysmal positional vertigo

TABLE 7: ELICITED NYSTAGMUS IN BENIGN PAROXYSMAL POSITIONAL VERTIGO BY SEMICIRCULAR CANAL INVOLVEMENT

PROVOCATION MANOEUVRE	AFFECTED CANAL	RIGHT	LEFT
Dix-Hallpike test	Posterior	Upbeating right torsion	Upbeating left torsion
Head-hanging test	Anterior	Downbeating right torsion	Downbeating left torsion
Supine-roll test	Horizontal	Horizontal	Horizontal

TABLE 8: DIAGNOSTIC CRITERIA OF VESTIBULAR MIGRAINE

A	At least five episodes with vestibular symptoms of moderate or severe intensity, lasting five minutes to 72 hours
B	Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders
C	One or more migraine features with at least 50% of the vestibular episodes. Headache with at least two of the following characteristics: ✓ One sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity ✓ Photophobia and phonophobia ✓ Visual aura
D	Not better accounted for by another vestibular diagnosis or International Classification of Headache Disorders

From Bárány Society/International Headache Society

"Recurrent positional vertigo is frequently caused by BPPV, which is the most common aetiology of vertigo"

Mnemonic features of DANISH

- **D**ysdiadochokinesia
- **A**taxia (gait and posture)
- **N**ystagmus
- **I**ntention tremor
- **S**lurred, staccato speech
- **H**ypotonia/heel-shin test

It may be associated with or without headache and may also be accompanied by myriad other symptoms including nausea, vomiting, photosensitivity, phonosensitivity, osmosensitivity, mild subjective hearing loss, tinnitus and aural fullness with or without visual aura.

Ménière's disease

The pathophysiology of Ménière's disease is generally attributed to the idiopathic excess of endolymphatic fluid within the acoustic and vestibular apparatus, either by excessive production or reduced elimination (see Table 9).

Cerebellar dizziness and vertigo

Dizziness, vertigo and imbalance due to cerebellar disease present a particular differential diagnostic challenge. With damage confined solely to the vestibulocerebellum, these patients typically do not present with the full spectrum of cerebellar symptoms and most of the mnemonic features of DANISH (see box above) are therefore absent. The key to this diagnosis is the examination of eye movements, as practically all patients have at least one oculomotor abnormality of cerebellar origin, such as saccadic ocular pursuit, gaze-evoked nystagmus, or downbeat and rebound nystagmus.

SUMMARY

Diagnosis of vertigo is complex. This article provides a general overview of the key issues to consider when assessing patients, and the initial diagnostic approach. It does not, however, provide a comprehensive account of individual tests and their interpretation to cover all possible peripheral and central vestibular conditions. Readers are therefore advised to exercise caution and further learning and development in this area is encouraged.

TABLE 9: DIAGNOSTIC CRITERIA OF MÉNIÈRE'S DISEASE

A	Two or more spontaneous episodes of vertigo, each lasting 20 minutes to 12 hours
B	Low- to medium-frequency sensorineural, fluctuating hearing loss
C	Fluctuating symptoms such as tinnitus or aural fullness in the affected ear
D	Not better accounted for by another vestibular diagnosis

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