

Approach to A Patient with Vertigo: A Guide For Physicians

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Abstract

The vestibular system helps to maintain balance and stabilise gaze. Balance is controlled primarily by the cerebellum, which receives input from the vestibular nuclei in the brain stem. This, in turn, receives input from the visual pathway, proprioception and inner ear. The vestibular apparatus (labyrinth) of the inner ear consists of three semicircular canals, and two otolith organs called the utricle and the saccule. Displacements and linear accelerations of the head, are detected by the two otolith organs. Receptors in the semicircular canals respond to the rotational movements of the head. When the head tilts to one side, receptors on the ipsilateral ear are stimulated and receptors in the contralateral ear are inhibited. These receptors send impulses to the brain stem and cerebellum via the vestibular nerve to control balance. Any disruption of this pathway can lead to vertigo¹.

Abnormalities in the vestibular/visual/proprioceptive system can cause symptoms of vertigo. Vertigo due to vestibular system abnormalities may result from damage or dysfunction in parts like the vestibular labyrinth or the vestibular nerve (peripheral causes), or occur due to disturbances in the central vestibular system, specifically in the brainstem and cerebellum².

Patients with abnormalities of vestibular system can present with symptoms of vertigo (spinning sensation), dysequilibrium (feeling of imbalance), light-headedness (sensation of giddiness/dizziness) and falls. Targeted history taking, clinical bedside examinations, functional testing of the vestibular system, and imaging are instrumental for correct diagnosis and therapy management. This article will act as a quick guide to help physicians evaluate dizzy patients.

Accurate diagnosis of a patient with vertigo, dizziness, or unsteadiness can be challenging, ranging from acute symptoms in emergency departments to chronic symptoms in outpatient settings. One of the challenges is the heterogeneity of symptoms on clinical presentation and the wide range of etiologies underlying these leading symptoms³. Vestibular symptoms can all be caused by a range of general medical, (e.g., hypoglycaemia, anaemia), neurological, (e.g., migraine, epilepsy, dysautonomia, cerebellar and extrapyramidal disorders), cardiovascular, (e.g., orthostatic hypotension, arrhythmias) and psychiatric conditions (e.g., panic attacks, generalised anxiety). These should be kept in mind while evaluating a patient with vestibular symptoms.

According to the International Classification of Vestibular Disorders (ICVD) created by Classification Committee of the

Bárány Society (CCBS), a patient presenting with vestibular symptoms can be grouped into three syndromes⁴. These are:-

- (i) acute vestibular syndrome (AVS) comprised of monophasic diseases of acute onset lasting days to weeks,
- (ii) episodic vestibular syndrome (EVS) encompassing illnesses manifesting recurrent attacks of vestibular symptoms each lasting seconds to days, and
- (iii) chronic vestibular syndrome (CVS) including conditions in which symptoms persist for a minimum of 3 months.

The acute vestibular syndrome (AVS) is usually defined as the rapid onset of dizziness or vertigo, nausea or vomiting, head motion intolerance, gait instability, and often nystagmus lasting for at least 24 hours⁵. AVS is the result of an acute unilateral, peripheral or central, vestibular lesion that causes a sudden asymmetry of the normal vestibular nuclei neuronal firing rate⁶. Although most cases of definite AVS are likely to be either vestibular neuritis or stroke, the differential diagnosis can include labyrinthitis, first attack of Meniere's disease or vestibular migraine. It is important, therefore, not to miss stroke as all other are relatively benign causes. Stroke is suspected if the patient presents with associated neurological symptoms such as weakness, dysarthria, sensory changes, ataxia or confusion. Risk factors for vascular disease, including smoking, diabetes, obesity, hypertension and hyperlipidaemia, need to be assessed to rule-out stroke and CVAs, which can lead to vertigo from ischaemia or infarction¹.

Additional symptoms associated with vertigo may originate from a peripheral lesion⁷, such as recent viral infection that

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can cause acute labyrinthitis and vestibular neuritis. Finally, it is essential to review a patient's medication prescriptions and social history for any substance or alcohol use. Medications that can impact vestibular function include anticonvulsants, antidepressants and antihypertensive agents.

Episodic vestibular syndrome (EVS) patient presents with recurrent episode of vertigo lasting seconds to days. The differential diagnosis include: BPPV, labyrinthine dehiscence, Meniere's disease and vestibular migraine. These can be due to some trigger or can be spontaneous. Trigger refers to a specific act or situation that provokes or aggravates the symptoms⁸. Bending over, lying down, rolling over in bed, may all trigger short vertigo attacks, typical for BPPV (Benign paroxysmal positional vertigo). Vestibular problems due to BPPV should cause vertigo in particular lying down position, and may also during the upright movement. If problems only occur during the erecting phase, it could be due to orthostatic hypotension. Valsalva manoeuvre, coughing, or loud sounds that cause vertigo can be attributed to perilymphatic fistula or a third mobile window lesion, e.g., the superior semicircular canal dehiscence. Some vertigo attacks occur completely spontaneously, such as those caused by Meniere's disease or vestibular migraine. Presence of associated aural symptoms such as tinnitus and hearing loss suggests Meniere's disease. Clinicians should also enquire about symptoms such as headache, photophobia, and visual auras, as these frequently accompany vestibular migraines.

Distinguishing various timing and triggers of dizziness in patients helps to narrow the differential diagnosis and focus on serious treatable causes. Many medical causes commonly present with episodic dizziness or vertigo, (e.g., cardiac arrhythmia, orthostatic hypotension, transient hypoglycaemia).

Chronic vestibular Disorders (CVS) include Vestibular migraine and Bilateral vestibulopathy which could be due to ototoxic medications, idiopathic, infection, age-related or autoimmune conditions. These patients present with dizziness, postural instability and oscillopsia.

When combined with a comprehensive history, a focused physical examination can help further differentiate between various causes of vertigo. Examination should involve the following¹:-

- **Eye examination:**

- **Nystagmus testing:** Nystagmus is quick, jerky, involuntary movements of the eye. Nystagmus has a slow phase (vestibular driven) and fast phase (corrective saccade). Direction of nystagmus is

denoted by the direction of fast component. In peripheral lesions, the predominant direction of nystagmus remains the same regardless of the direction of gaze, while central lesions may present with nystagmus that reverses direction⁹. Horizontal gaze direction changing nystagmus, (i.e., right beating nystagmus in right gaze and left beating nystagmus in left gaze) is a hallmark of a central lesion. In addition, the presence of vertical or purely torsional nystagmus is suggestive of central lesions as well¹⁰.

- **Head Impulse Test (HIT):** A functional vestibular system allows one to maintain gaze during rotation through vestibulo-ocular reflexes (VOR). The head impulse test is a physical examination technique to test VOR. In this exam, patients are asked to fix their gaze on a target such as examiner's nose. The head is turned quickly to the right or left by about 15 degrees. A typical response occurs when the eyes remain on the target. An abnormal response is when the eyes are dragged off the target in the direction of head turning, followed by a corrective saccade back to the target. This response implies a peripheral lesion resulting in a deficient vestibulo-ocular reflex on the side of the head turn (Fig. 1).
- **Skew Test:** This test involves the examiner covering one eye and observing for a vertical or horizontal shift in the uncovered eye. Central lesions sometimes produce a slight skew deviation (Fig. 2).
 - When the head impulse test is combined with an examination of nystagmus and a test for skew, this is referred to as the Head Impulse-Nystagmus-Test for Skew (HINTS)⁶. A normal head impulse test on both sides with direction-changing nystagmus and/or skew deviation is suggestive of a central lesion. An abnormal head impulse test with unidirectional nystagmus and absent skew deviation strongly suggests a peripheral lesion.

Note: The HINTS test may be more sensitive for diagnosing acute stroke than magnetic resonance imaging (MRI) within the first 48 hours following symptom onset⁶.

- ❖ **Neurological examination:** A focused neurological examination including gait, balance and coordination needs to be performed. The gait, balance assessment (Romberg's test, Untenberger's test and foam test), and examination for cerebellar signs (such as finger nose test, test for



Fig. 1: Head Impulse Test. 1A): Patient is asked to fix gaze on a target such as examiner's nose. 1B), 1C): The head is turned quickly to the right or left by about 15 degrees. A typical response occurs when the eyes remain on the target.

dysdiadokinesia) can exclude central causes.

- ❖ **Ear examination:** An otoscopic examination should be performed to visualise the tympanic membrane for any retraction pockets as seen in cholesteatoma or vesicles on the pinna that can be seen in a Herpes zoster infection. Vertigo triggered by applying intermittent pressure on the tragus (Fistula test - Fig. 3) or with the Valsalva manoeuvre is seen in a perilymphatic fistula. A hearing assessment should be performed in such cases to look for sensorineural hearing loss.
- **Cardiovascular examination:** Pulse, blood pressure, heart rate and rhythm should be checked. Orthostatic blood pressure measurement must be done in suspected cases.

Neuroimaging is indicated¹ if:-

- Clinical examination is not consistent with a peripheral lesion
- prominent risk factors for CVA are present
- associated with neurological signs and symptoms such as diplopia, dysarthria.



Fig. 2: Test for Skew. 2a), 2b): This test involves the examiner covering one eye and observing for a vertical or horizontal shift in the uncovered eye

The preferred methods of imaging are brain MRI and magnetic resonance (MR) angiography, as computed tomography (CT) scan is less sensitive than MRI for detecting and evaluating central lesions due to their limited resolution of posterior fossa structures. However, if a brain MRI is unavailable or contraindicated, a CT scan with thin cuts may be used, primarily through the brainstem and cerebellum¹¹. In these cases, referral to a neurologist is recommended¹².

Treatment

The management of vertigo depends on its aetiology, and addressing the underlying cause often alleviates the symptoms. Pharmacological interventions may help suppress vestibular symptoms during acute episodes, which can persist for several hours to days. It is important to make healthcare professionals aware that vestibular suppressants (such as prochlorperazine) should be used for a limited duration, as excessive use may unnecessarily hinder the brain and central nervous system's natural compensatory mechanisms for peripheral vertigo. Steroids can be beneficial in some instances such as vestibular neuritis. Therapeutic repositioning manoeuvres such as Epley's manoeuvre is the recommended treatment for BPPV. Additional non-pharmacologic treatments for patients with permanent unilateral or bilateral vestibular dysfunction include physical therapy with vestibular rehabilitation.

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