

VERTIGO – A CLINICAL APPROACH

Dizziness and other sensations of imbalance are one of the most frequent complaints among medical outpatients. The significance of these complaints however varies greatly. For the most part they are benign, but always there is a possibility that they may signal the presence of an important neurologic disorder. To correctly diagnose the underlying disease, the complaint of dizziness must be analyzed correctly. The nature of the disturbance of function should be determined first followed by its anatomic localization.

Dizziness is prevalent in all adult populations, causing considerable morbidity and utilization of health services. In the community, the prevalence of dizziness ranges from 1.8% in young adults to more than 30% in the elderly.¹ A recent population-based telephone survey in Germany showed that nearly 30% of the population had experienced moderate to severe dizziness.² Various disease entities may cause dizziness, and the reported frequency of specific diagnoses varies widely, depending on the setting, patient age, and investigator bias.¹

APPROACH TO THE PATIENT WITH DIZZINESS

“Dizziness” refers to various abnormal sensations relating

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to perception of the body’s relationship to space.³ The term “dizziness” is applied by the patient to a number of different sensory experiences – a feeling of rotation or spinning as well as nonrotatory swaying, lightheadedness, faintness, weakness, or unsteadiness.⁴ Blurring of vision, feelings of unreality, syncope, and even some seizure phenomena may be called “dizzy spells.” Thus, a careful history is necessary to determine exactly what a patient who states, “Doctor, I’m dizzy,” is experiencing.

In a classic paper, Drachman and Hart⁵ described four subtypes of dizziness: vertigo, presyncopal lightheadedness, disequilibrium, and other dizziness. Nearly 40 years later, this typology remains the basis of dizziness definition and classification, having long since displaced the narrower definition “vertigo” used earlier.¹

The first principle to observe in evaluating a dizzy patient is not to put any words in the patient’s mouth. This is, of course,

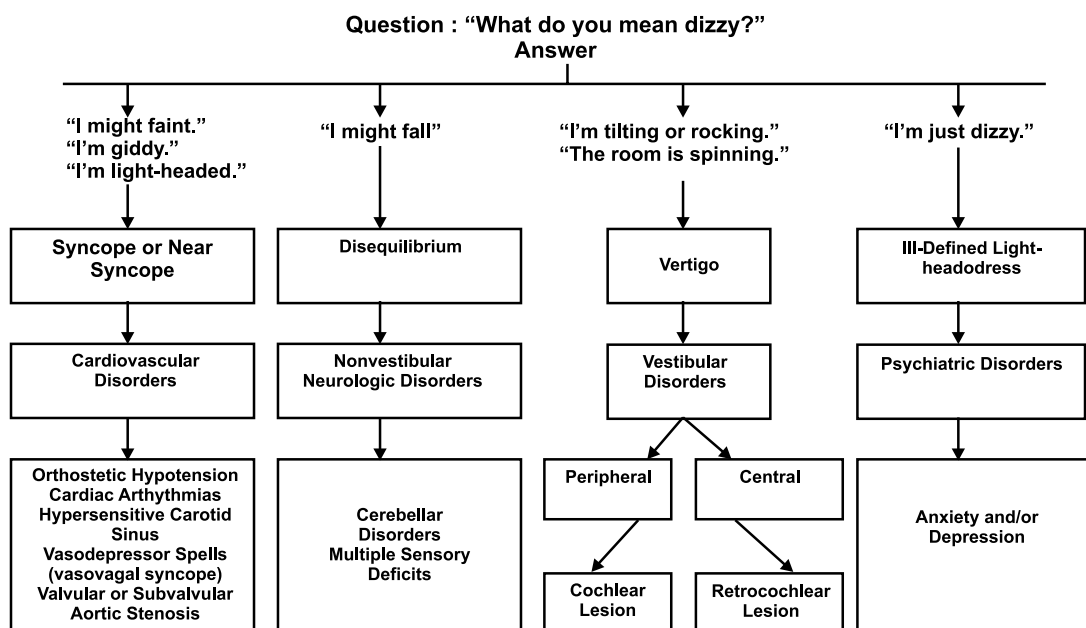


Fig. 1 : Approach to the patient with dizziness⁶

Table 1 : Approach to the differentiation of dizziness subtypes (modified from Sloane et al., 2001)¹

Dizziness subtype	Type of sensation	Temporal characteristics	Other specifications
Syncope or presyncope	A lightheaded, faint feeling, as though one were about to pass out	Typically occurs in episodes lasting seconds to hours	The following questions should be answered: 1. Do episodes occur only when the patient is upright, or do they occur in other positions? 2. Are episodes associated with palpitations, medication, meals, bathing, dyspnoea, or chest discomfort?
Disequilibrium	A sense of unsteadiness that is 1. primarily felt in the lower extremities, 2. most prominent when standing or walking, and 3. relieved by sitting or lying down	Usually present, although it may fluctuate in intensity	Identify whether symptom occurs in isolation or accompanies another dizziness subtype; describe exacerbating factors
Vertigo	A feeling that one or one's surroundings are moving (typically, spinning)	Episodic vertigo occurs in attacks that last seconds to days. Continuous vertigo is present all or most of the time for at least a week	Descriptions of episodic vertigo should include the characteristics, duration, and date of the first episode; length of episodes; and exacerbating factors
Ill-defined lightheadedness	A feeling not covered by the above definitions. May include swimming or floating sensations, vague lightheadedness, or feelings of dissociation. May be difficult for the patient to describe	Usually present all or most of the time for days or weeks, sometimes years	The following questions should be answered: 1. Is dizziness associated with anxiety or hyperventilation? 2. Is dizziness associated with changes in mood?

a good rule in taking any medical history, but it is particularly applicable in this instance. When the patient says, "I am dizzy," the physician should just ask, "What do you mean, dizzy?" and then wait for the response. This may take what seems to be a long time; nonetheless, one should never ask, "Does the room spin?" "Do your legs get weak?" "Do you feel as if you might stagger?" "Are you lightheaded?" because the answer to all these questions will nearly always be "yes". There are several responses that the patient may give (Figure 1).⁶ Based on these responses, a clinical approach to various subtypes of dizziness can be made (Table 1).

SYNCOPE OR PRESYNCOPE

Presyncope is a feeling of lightheadedness that is often described as a sensation of an impending faint – "I feel as if I might faint," or "I feel giddy or light-headed." It is episodic and usually results from diffuse temporary cerebral ischemia.¹ Some patients do faint (syncope); others have never actually fainted (near-syncope). Pathophysiologically, both syndromes suggest several cardiovascular disorders that produce a generalized decrease in cerebral blood flow; there is no qualitative difference between syncope and near-syncope with respect to the differential diagnosis.⁶

Circulatory syndromes that should be considered in the differential include *orthostatic hypotension*, which may have a number of causes, most of them iatrogenic (e.g., antihypertensive agents and/or vasodilators). *Cardiac arrhythmias* are a very frequent cause of syncope and near-syncope. If the history suggests arrhythmic episodes, holter monitoring may be required. *Hypersensitive carotid sinus* is relatively uncommon. *Vasovagal attacks* are otherwise known as the simple faint or the simple swoon. *Neurocardiogenic*

syncope is probably due to over activity of the baroreceptor reflex such that brief periods of hypotension result in disproportionate bradycardia and hypotension resulting in decreased cerebral blood flow and consequent loss of consciousness.⁶

DISEQUILIBRIUM

Disequilibrium is a sense of imbalance (postural instability) that is generally described as involving the legs and trunk without a sensation in the head – "I feel as if I might fall." This version of dizziness generally reflects one of two major categories of neurologic disease, apart from disorders of the vestibular system: *cerebellar ataxia* and *the multiple sensory deficits syndrome*.⁶ *Cerebellar ataxia* is due either to a primary disease of the cerebellum, e.g., cerebellar degeneration, or to a tumor in or near the cerebellum, e.g., in the cerebellopontine angle. Neurologic examination will ordinarily reveal such pathology.

The multiple sensory deficits syndrome reflects multiple abnormalities in the various sensory proprioceptive systems. When several of these systems fail in a given individual, the central nervous system receives conflicting proprioceptive input, with consequent dizziness. The typical patient is rather elderly, perhaps with some *visual disorder* due to cataracts, some *auditory disorder* due to presbycusis, and *peripheral neuropathy* due to diabetes and/or chronic use of alcohol. Such a patient typically complains of dizziness at night, for instance, when the lights are out or dim and he or she has to go to the bathroom.⁶

VERTIGO

Vertigo is an illusory or hallucinatory sense of movement of the

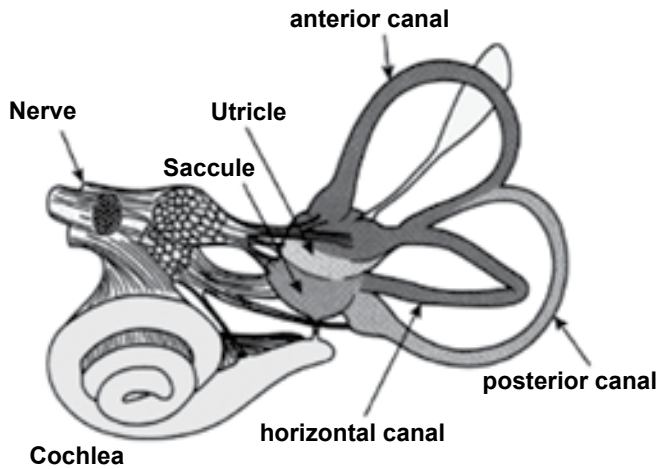


Fig. 2 :The vestibular system - semicircular canals and otolith organs

body or the environment, most often a feeling of spinning.⁷ In either case the patient says, "I feel as if I am tilting, rocking, or moving in some other way," or "I feel as if the room is spinning." It suggests a disturbance of the vestibular system; although psychological states, such as panic disorder, can also produce it.⁸ Vestibular system is responsible for keeping the central nervous system informed of the head's position in space, its relation to the pull of gravity, and its acceleration in various planes. The question is whether the vertigo is due to a disorder in the *peripheral nervous system* (the end organ or the peripheral nerve) or in the *central nervous system* (the brainstem or its projections to parts of the cerebral cortex, particularly the temporal lobe). Each lesion has its own differential diagnosis and treatment.⁶

ILL-DEFINED LIGHTHEADEDNESS

There are patients who when asked, "What do you mean, dizzy?" respond, usually after a pause, "Dizzy." If the physician persists with "Do you mean you might faint?" or "Do you mean that you might fall?" or "Do you mean that the room spins?" the patient repeats, "No, I mean I'm dizzy." This disorder generally arises from various psychological disorders, most commonly anxiety and/or depression. It is extremely important to recognize instances when dizziness represents a metaphor for depression, because treatment for vertigo is likely to exacerbate depression, whereas treatment for depression might dramatically relieve the dizziness.⁶

ANATOMY AND PHYSIOLOGY OF THE VESTIBULAR SYSTEM ⁷

Before considering the evaluation of vertigo, a brief review of the neuroanatomy and neurophysiology of the vestibular system may be helpful.

The end organs of this system consist of the three semicircular canals and the otolithic apparatus (utricle and saccule), situated in the bony labyrinths of inner ears on each side (Figure 2). The canals transduce angular acceleration and the otoliths transduce

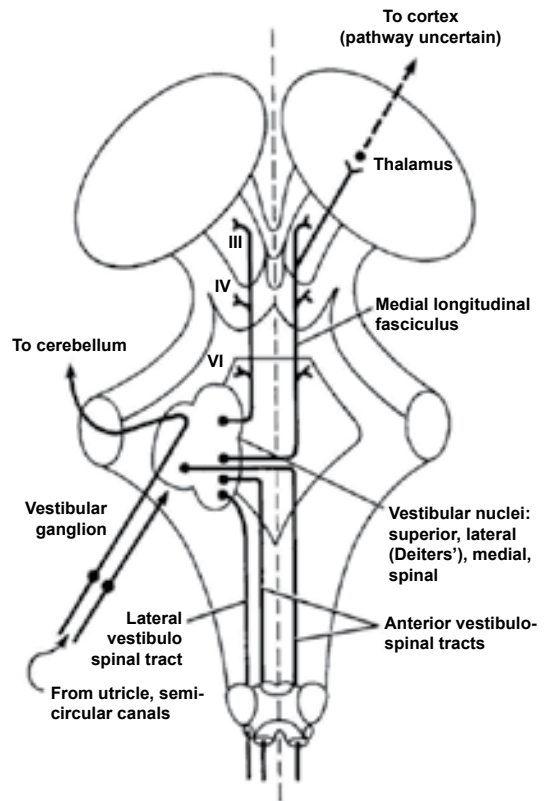


Fig. 3 : Connections of the vestibular system

linear acceleration and the static gravitational forces that provide a sense of head position in space. The principal projections from the vestibular nuclei are to the following structures (Figure 3):

1. Nuclei of the cranial nerves III, IV, and VI
2. Cerebellum
3. Spinal cord
4. Cerebral cortex

Physiologic considerations:

1. Projections from vestibular nuclei to the nuclei of the cranial nerves III, IV, and VI:

The vestibuloocular reflex (VOR): This reflex serves to maintain the visual stability during head movement and depends on the connections of vestibular nuclei to the nuclei of the cranial nerve III, IV, and VI. These connections are responsible for the nystagmus (to- and fro- oscillations of the eyes) that is an almost invariable accompaniment of vestibular dysfunction.

2. Projections from vestibular nuclei to the cerebellum:

The vestibular nerves and nuclei project to areas of the cerebellum (primarily flocculus and nodulus) that modulate the VOR.

3. Projections from vestibular nuclei to the spinal cord:

The vestibulospinal pathways assist in the maintenance of postural stability.

4. Projections from vestibular nuclei to the cerebral cortex:

Projections to the cerebral cortex, via the thalamus, provide conscious awareness of head position and movement in space.

Overall, three sensory systems subservise spatial orientation and posture:

1. The vestibular system
2. The visual system (retina to occipital cortex)
3. The somatosensory system (conveys peripheral information from skin, joint and muscle receptors)

These three stabilizing systems overlap sufficiently to compensate (partially or completely) for each other's deficiencies. Vertigo may represent either physiologic stimulation or pathologic dysfunction in any of the three sensory systems.

PATHOPHYSIOLOGY OF VERTIGO AND NYSTAGMUS⁹

ORIGIN OF VERTIGO

The maintenance of the sense of balance and spatial orientation depends on input from the vestibular labyrinth, visual system, and proprioceptive nerves arising from tendons, muscles, and joints.¹⁰ The vestibular nuclei, which are in the medulla and lower pons, receive input from the vestibular labyrinth via the vestibular branch of cranial nerve VIII and from the cerebellum.¹¹ The vestibular nuclei, in turn, send efferent fibres to the cerebellum, the medial longitudinal fasciculus, and the vestibulospinal tract (Figure 3). Visceral manifestations of vertigo (such as nausea and vomiting) are caused by altered input to the dorsal nucleus of the vagus nerve from the vestibular nuclei. Conscious awareness of vertigo resides in the superior temporal gyrus of the cerebral cortex¹⁰ and involves a mismatch between input to the cerebral cortex from the visual, proprioceptive, and vestibular systems.¹² Lesions in various locations, including the inner ear, brainstem, and cerebellum, may all be manifested as vertigo.

ORIGIN OF NYSTAGMUS

Nystagmus is the objective accompaniment of vertigo and is defined best as a "rhythmical oscillation of the eyes, with a fast movement in one direction and a slow movement in the other."¹³ The fast component may be horizontal, vertical, rotatory, or any combination of these.¹⁴ There are two clinically relevant kinds of nystagmus in evaluating vertigo:

1. Spontaneous nystagmus is elicited by having the patient look straight ahead, up, down, to the right, and to the left. This type of nystagmus is not influenced by head position.¹⁵ It is normal to have a few beats of nystagmus with extreme lateral gaze.¹⁴

2. Positional nystagmus is elicited by a head-hanging manoeuvre (Figure 4).¹⁴

Altered input passing from the vestibular nuclei to the nuclei of the extraocular muscles through the medial longitudinal fasciculus produces nystagmus. This input may be modified by information arising from the cerebral cortex and the cerebellum.¹⁴ For example, the fast component of spontaneous nystagmus depends on interaction between the vestibular system and the cerebral cortex.

EVALUATION OF VERTIGO

The first step includes a complete history and physical examination.

Following questions should be asked detailing the characteristics of vertigo experienced by the patient:¹⁶

Is the symptom constant or episodic?

With episodic vertigo, what is the duration and frequency of attacks?

How did it begin (e.g., gradual or sudden)?

Are there accompanying symptoms?

Are there aggravating or alleviating factors?

Are there identifiable triggers?

A brief general medical examination is important. Identifying orthostatic blood pressure changes (supine, sitting, and standing) can be diagnostic in the correct clinical setting such as in those with orthostatic symptoms. Orthostatic hypotension is probably the most common general medical cause of dizziness among patients referred to neurologists. Identifying an irregular heart rhythm also may lead to correct diagnosis. Other measures to be considered in individual patients include a visual acuity measurement (as adequate vision is important for balance) and a musculoskeletal inspection (significant arthritis can impair gait).¹⁶ This should be followed by a general neurologic examination with particular attention to the VIII cranial nerve (both the vestibular and cochlear components).

COCHLEAR VIII NERVE FUNCTION

The bedside hearing examination is not very sensitive as a screening tool for hearing loss but can provide important information in patients with auditory symptoms. When a patient has auditory complaints, or when an audiovestibular disorder is strongly suspected, a standard audiogram should be performed as it more accurately assesses the wide spectrum of the auditory system.

The **whisper test** has been shown to be the most sensitive test in picking up hearing loss at the bedside.¹⁷ For this test, the examiner stands behind the patient to prevent lip reading and occludes and masks the non-test ear using a finger to rub and close the external acoustic meatus. The examiner then whispers a set of three to six random numbers and letters. The patient is considered to have

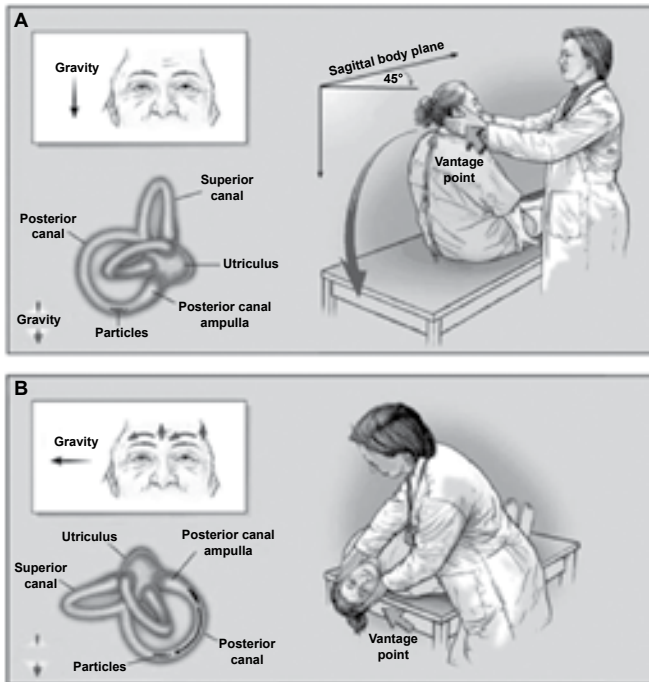


Fig. 4 :The Dix-Hallpike Test of a patient with benign paroxysmal positional vertigo affecting the right ear
 In Panel A, the examiner stands at the patient's right side and rotates the patient's head 45 degrees to the right to align the right posterior semicircular canal with the sagittal plane of the body. In Panel B, the examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The arrows over the eyes in the inset depict the direction of nystagmus in patients with typical benign paroxysmal positional vertigo. The presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown.

passed the screening test if he/she repeats at least 50% of the letters/numbers correctly.

Auditory testing. Examination of the cochlear system involves three steps whether or not the patient complains of hearing loss.⁶

1. **Pure tone hearing loss**

The first is to test for *pure tone hearing loss*. This can be done quite reliably in the office by comparing the sensitivity of the patient's ears with your own, using a ticking watch or the sound of your fingers rubbing together.⁶

2. **Sensorineural vs. conductive hearing loss**

If there is a pure tone hearing loss, the next step is to determine whether it is a sensorineural hearing loss, i.e., a neurologic problem, or a conductive hearing loss, i.e., a dis-

order in the middle ear interfering with the functions of the ossicles. These determinations are made by using two tests, the Weber and Rinne.

The Weber test is performed by placing a vibrating tuning fork (512 Hz) at the midline of the skull (vertex), forehead, bridge of the nose, or upper incisors and asking the patient whether the sound is heard in the middle of the head or in both ears equally, towards the left or towards the right. In asymmetrical or unilateral hearing impairment, the tone lateralizes to one side. Lateralization indicates an element of conductive impairment in the ear in which the sound localizes, a sensorineural impairment in the contralateral ear, or both.¹⁶

The Rinne test is of greater use in an office setting. Bone and air conduction are compared by placing the tuning fork first over the mastoid bone until the sound fades and then 1 inch in front of the ear. Normal persons can hear the fork about twice as long by air as by bone conduction. If the ossicles are not functioning because of otosclerosis, cholesteatoma, etc., the air and bone conduction become equal or bone conduction becomes better of the two. If, however, there is sensorineural hearing loss, air conduction remains better than bone conduction.⁶

3. **Cochlear vs. retrocochlear hearing loss**

The third step in the hearing examination, needed only if there is a sensorineural loss, is perhaps the most important of the differential procedures but paradoxically the one least well known to many physicians. The question being asked is whether the sensorineural deficit is due to end organ disease (cochlear) or to peripheral or central neural disease (retrocochlear).

Speech discrimination testing can be done as an office procedure to differentiate a cochlear from retrocochlear sensorineural hearing loss. The examiner whispers random numbers or words in the affected ear. At the same time, a sound is made in the other ear so that the patient cannot hear the words through that ear. Putting a finger in the patient's other ear and moving it around will serve the purpose. This is done on both sides five or 10 times, the patient is asked to repeat the words each time, and the two ears are compared. In people with cochlear-type sensorineural hearing loss, such as occurs in Meniere's disease, speech discrimination is not perfect, but it is *relatively* preserved. On the other hand, in patients with retrocochlear hearing loss, such as accompanies an acoustic schwannoma, there is a disproportionate loss of speech discrimination. Thus, a patient with a cochlear hearing loss should be able to understand 70% or more of the words heard, whereas a patient with a retrocochlear hearing loss might understand only two out of 10 words. *If there is any question of a retrocochlear hearing loss, one should order an audiogram.*⁶

VESTIBULAR VIII NERVE FUNCTION

A unilateral or bilateral vestibulopathy can be identified by the

Table 2 : Characteristics of peripheral versus central positional vertigo^{7,18,19}

Features	Peripheral	Central
Latency ^a	3 – 40 s	None: immediate vertigo and nystagmus
Duration	< 1 min.	Symptoms may persist longer
Fatigability ^b	Yes, lasts 10-30 sec, rarely as long as 1 min.	No
Habituation ^c	Yes	No
Reproducibility ^d	Variable	Good
Intensity of vertigo	Severe vertigo, marked nystagmus, nausea	Usually mild vertigo, less intense nystagmus, rare nausea

^aTime between attaining head position and onset of vertigo or nystagmus.

^bDisappearance of symptoms with maintenance of offending position.

^cLessening of symptoms with repeated trials.

^dLikelihood of symptom production during any examination session.

Table 3 : Features of peripheral and central vertigo^{7,19}

Sign/symptom	Peripheral (Labyrinth)	Central (Brainstem/Cerebellum)
Direction of associated nystagmus	Unidirectional; fast phase opposite lesion ^a	Bidirectional or unidirectional
Purely horizontal nystagmus without torsional component	Uncommon	Common
Vertical or purely torsional nystagmus	Never present	May be present
Visual fixation	Inhibits nystagmus and vertigo	No inhibition
Severity of vertigo	Marked	Often mild
Direction of spin	Towards fast phase (away from lesion)	Variable
Direction of fall	Towards slow phase	Variable
Duration of symptoms	Finite (minutes, days, weeks) but recurrent	May be chronic
Tinnitus and/or deafness	Often present	Usually absent
Associated CNS abnormalities	None	Extremely common (e.g., diplopia, dysarthria, hiccups, cranial neuropathies, ataxia, hemisensory loss or even paralysis)
Common causes	BPPV, infection (labyrinthitis), Meniere's disease, neuronitis, ischemia, trauma, toxins	Vascular, demyelinating, neoplasm

^a In Meniere's disease, direction of the fast phase is variable

head thrust test.

HEAD THRUST TEST

In this test, the examiner stands directly in front of the patient. With the patient's head held in the examiner's hand, he/she is

Table 4 : Common causes of vertigo^{9,19}

Peripheral causes of vertigo

1. Benign paroxysmal positional vertigo
2. Vestibular neuronitis
3. Recurrent vestibulopathy
4. Classic Meniere's disease
5. Head trauma (labyrinthine concussion)
6. Otosclerosis
7. Herpes zoster oticus
8. Cholesteatoma
9. Perilymph fistula
10. Aminoglycoside ototoxicity

Central causes of vertigo

1. Vascular: vertebrobasilar transient ischemic attacks, cerebellar or brain stem stroke
2. Cerebellopontine angle tumors: acoustic neuroma, meningioma, cholesteatoma, metastatic tumor
3. Demyelinating disease: multiple sclerosis, postinfectious demyelination
4. Vertebrobasilar migraine
5. Cranial neuropathy: focal involvement of VIII nerve or in association with systemic disorders
6. Intrinsic brainstem lesions: tumor, arteriovenous malformations
7. Seizure disorders (rare)
8. Heredofamilial disorders (such as spinocerebellar degeneration)

Systemic causes of vertigo and dizziness¹⁹

1. Drugs and toxins (including anticonvulsants, hypnotics, antihypertensives, alcohol, analgesics, tranquilizers, quinine, ethacrynic acid, aminoglycoside antibiotics (especially streptomycin, gentamicin), salicylates, benzene, arsenic, arsine)
2. Hypotension, presyncope (including primary cardiac causes and postural hypotension from a wide variety of causes)
3. Infectious diseases (including syphilis, viral and other bacterial meningitides, and systemic infection)
4. Endocrine diseases (including diabetes and hypothyroidism)
5. Vasculitis (including collagen vascular disease, giant cell arteritis, and drug-induced vasculitis)
6. Other systemic conditions (including haematological disorders [polycythemia, anemia, and dysproteinemia], sarcoidosis, granulomatous disease, and systemic toxins)

instructed to focus on the examiner's nose. The head is then quickly moved about 5 to 10 degrees to one side. In patients with normal vestibular function, the VOR results in movement of the eyes in the direction opposite the head movement. So, the patient's eyes remain fixed on the examiner's nose after sudden movement. This test is repeated in the opposite direction. If a corrective saccade is seen bringing the patient's eyes back to the examiner's nose after the head thrust, impairment of the VOR in the direction of head movement is identified.¹⁶

TESTING FOR NYSTAGMUS

Ask the patient to look about 45° to the right and to the left (asking the patient to look beyond 45° is not useful, since when asked to look too far in either direction, about 10% of the normal population show some degree of gaze-evoked end-point nystagmus). If nystagmus develops note the direction of the fast

phase, the direction of the slow phase, and in what position of the eyes they occur.⁶

Next the patient should be put through a series of positions called the *Nylen-Barany* manoeuvre (*aka Dix-Hallpike*) (Figure 4). All vertigo is positional to some extent, but if vertigo is positional only, there are specific pathogenetic and prognostic implications.⁶ Positional vertigo is precipitated by a recumbent head position, either to the right or to the left. The vertigo and accompanying nystagmus have features that differ from the less common central positional vertigo (Table 2) due to lesions in and around the IV ventricle.⁷

CRITERIA FOR LOCALIZING THE LESION

Table 3 shows the clinical features which may help in deciding whether the lesion causing vertigo is anatomically located peripherally in either the end organ or the peripheral nerve, or centrally in the brainstem or cerebellum. These criteria specify only the anatomic localization without implying anything about the severity or seriousness of the underlying disease.

SYNTHESIZING THE DATA

Thus, by testing the auditory system and the vestibular system, one can divide all cases of vertigo into three categories:

1. *Peripheral* (by vestibular criteria) *cochlear disease* (by auditory criteria and signs),
2. *Peripheral* (by vestibular criteria) *retrocochlear disease* (by auditory criteria), and
3. *Central disease*.

Table 4 lists the various common causes of vertigo.

DIAGNOSTIC FORMULATIONS

The symptoms and findings on examination lead to a tentative diagnosis i.e. the etiology.

PERIPHERAL VESTIBULOPATHY

A patient who complains of episodic spinning vertigo, with or without auditory symptoms, with normal neurological examination, and who has evidence of reduced vestibular function in one ear should initially be placed in this diagnostic category. Such patients often respond to vestibular suppressant medications early in the course and thereafter to vestibular exercises. Peripheral vestibulopathy of idiopathic, infectious, or posttraumatic origin is one of the most common causes of a single attack of vertigo (Table 5).

CENTRAL VESTIBULAR DISORDERS

Patients with central vestibular disorders usually have vague descriptions of their symptomatology; they rarely describe true spinning vertigo or symptoms evoked by position change, and they have abnormalities, such as nystagmus or hyperreflexia, on examination. They are candidates for additional

Table 5 : Differential diagnosis of attacks of dizziness/vertigo¹⁹

Single attack	Recurrent attacks	Chronic disequilibrium
• Acute peripheral vestibulopathy	• Peripheral vestibulopathy	• Uncompensated peripheral vestibulopathy
• Trauma	• Benign paroxysmal positional vertigo	• Cerebellopontine tumor
• Perilymph fistula	• Meniere's disease	• Multiple sclerosis
• Air travel	• Vertebrobasilar ischemia	• Brainstem infarct
• Ramsay Hunt syndrome	• Migraine	• Drugs
• Syncope and presyncope	• Complex partial seizure	• Ototoxicity
	• Familial periodic ataxia	• Chronic otomastoiditis
		• Autonomic neuropathy
		• Multiple sensory deficits
		• Arteritis (vasculitis)

Table 6 : Medications that often cause dizziness¹

Class of Medication	Probable Mechanism	Example
α1-Adrenergic antagonists	Orthostatic hypotension	Prazosin
Alcohol	Hypotension, osmotic effects	Wine, cough syrups
Aminoglycosides	Ototoxicity	Gentamicin
Anticonvulsants	Orthostatic hypotension	Carbamazepine
Antidepressants	Orthostatic hypotension	Desipramine
Anti-Parkinsonian medication	Orthostatic hypotension	Levodopa
Antipsychotics	Orthostatic hypotension	Olanzapine
β-Blockers	Hypotension or bradycardia	Atenolol
Calcium-channel blockers	Hypotension, vasodilation	Verapamil
Class Ia antiarrhythmics	Torsades de pointes	Procainamide
Digitalis glycosides	Hypotension	Digoxin
Diuretics	Volume contraction; vasodilation	Hydrochlorothiazide
Narcotics	Central nervous system depression	Morphine, propoxyphene
Oral sulfonylureas	Hypoglycemia	Tolazamide
Vasodilators	Hypotension, vasodilation	Hydralazine

neurological diagnostic investigations. If a history of alteration of consciousness is present, a rare presentation of temporal lobe seizure may be considered and an electroencephalogram and MRI brain performed. The presence of progressive hearing loss or central auditory findings during audiometric testing leads to suspicion of cerebellopontine angle tumour and the appropriate neuroradiologic investigations. While encountering a patient with chronic vestibular symptoms (even including episodic imbalance) and failure to respond to medical therapy, however, one should carry out a neuroradiologic investigation, even in the presence of completely normal neurologic examination. They may have unsuspected multiple cerebral and brainstem infarctions or may be experiencing the late-life onset of multiple sclerosis. There is considerable overlap in the conditions causing single, recurrent or chronic attacks of dizziness (Table 5). Various drugs causing dizziness are enumerated in Table 6.

Table 7 : Distinguishing among common peripheral and central vertigo syndromes ¹⁶

Cause	History of vertigo	Duration of vertigo	Associated symptoms	Physical examination
Peripheral				
Vestibular neuritis	Single, prolonged episode	Days to weeks	Nausea, imbalance	“Peripheral” nystagmus, positive head thrust test, imbalance
BPPV	Positionally triggered episodes < 1 minute		Nausea	Characteristic positionally triggered burst of nystagmus
Meniere’s disease	May be triggered by salty foods	Hours	Unilateral ear fullness, tinnitus, hearing loss, nausea	Unilateral low- frequency hearing loss
Vestibular paroxysmia	Abrupt onset; spontaneous or positionally triggered	Seconds	Tinnitus, hearing loss	Usually normal
Perilymph fistula	Triggered by sound or pressure changes	Seconds	Hearing loss, hyperacusis	Nystagmus triggered by loud sounds or pressure changes
Central				
Stroke/TIA	Abrupt onset; spontaneous	Stroke, > 24 hours; TIA, usually minutes	Brainstem, cerebellar	Spontaneous “central” nystagmus; gaze-evoked nystagmus; usually focal neurologic signs
Multiple sclerosis	Subacute onset	Minutes - weeks	Unilateral visual loss, diplopia, incoordination, ataxia	“Central” types or rarely “peripheral” types of spontaneous or positional nystagmus; usually other neurologic signs
Neurodegenerative disorders	May be spontaneous or positionally triggered	Minutes - hours	Ataxia	“Central” types of spontaneous or positional nystagmus; gaze-evoked nystagmus; cerebellar, extrapyramidal and frontal signs
Migraine	Onset usually associated with typical migraine triggers	Seconds - days	Headache, visual aura, photo-/phonophobia	Normal interictal exam. Ictal examination may show “peripheral” or “central” types of spontaneous or positional nystagmus
Familial ataxia syndromes	Acute - subacute onset; usually triggered by stress, exercise, or excitement	Hours	Ataxia	“Central” types of spontaneous or positional nystagmus; ictal, or even interictal, gaze-evoked nystagmus; ataxia; gait disorders

The salient distinguishing features among the common peripheral and central vertigo syndromes are illustrated in Table 7.

PERIPHERAL COCHLEAR LESIONS

Labyrinthitis is thought to be a result of viral infection of the endolymph and perilymph affecting both the vestibular and cochlear components of the system. The usual history is viral illness followed by acute onset of severe spinning vertigo and sensorineural deafness with tinnitus. Examination shows a classic peripheral picture by vestibular criteria and a classic cochlear picture by auditory criteria. Paracentesis of the perilymph may show growth of common ubiquitous viruses, such as coxsackievirus or echovirus. Despite its severe onset, labyrinthitis is a benign illness, which resolves completely in three to six weeks. Patients regain normal hearing and vestibular function.

Vestibular neuronitis, or acute vestibulopathy, is thought to be pathogenetically identical to labyrinthitis but without any hearing symptomatology. If the patient has vertigo unaccompanied by a hearing abnormality, it is strictly speaking impossible to be sure whether the disease is cochlear or retrocochlear. However, its natural history is also benign, and it clears up completely in three to six weeks, which makes a retrocochlear illness very unlikely.

Meniere’s disease is caused by a cryptogenic hydrops of the endolymph such that there is intermittent swelling of the semicircular ducts, with damage to the hair cells. An attack of

Meniere’s syndrome is classically characterized by a dull ache in the region of the mastoid process or around the ear associated with severe tinnitus, a cochlear kind of sensorineural hearing loss, and severe spinning vertigo. It however, does not clear up completely in three to six weeks, and patients are left with residual hearing loss. Several months or years later a similar attack may occur, leaving the patient with even more severe hearing loss. About 15% of these patients will have bilateral disease in subsequent years.

Benign positional vertigo, or Barany’s vertigo, usually occurs in older patients and is characterized by the sudden onset of a peripheral vestibular syndrome *with no auditory aspect*. It is present only in certain positions, which are specific to the individual. Typically, the patient reports that a few moments after attaining a certain position, a severe vertigo occurs in which the world spins in one direction while the patient has a sensation of falling in the other direction. If he or she does not move, the vertigo stops, which implies that it is transient in type. All the symptoms can be reproduced using the Nylen-Barany maneuver (Figure 4). Benign positional vertigo has a natural course, which improves gradually over a six-month period and ends with complete recovery.

PERIPHERAL RETROCOCHLEAR SYNDROMES

VESTIBULAR SCHWANNOMA

A second category of disease is a peripheral type of vertigo but

with retrocochlear hearing loss, i.e., patients are found to have poor speech discrimination. Such patients should always have an audiogram; if the audiogram confirms retrocochlear hearing loss, a CT scan with special views of the internal auditory meatus is indicated. It is important to recognize the presence of a tumor while it is still contained within the internal auditory meatus and thus, surgically resectable. Vestibular schwannomas (acoustic neuromas) are histologically benign tumors, but they can become quite dangerous. *Any patient with a history of progressive hearing loss should at some time during the evaluation have a careful audiogram, and if any retrocochlear characteristics are found, a CT scan with careful views of the internal auditory meatus should be ordered.*

CENTRAL LESIONS

This group includes patients with vertical or bidirectional nystagmus in the same position of the head.⁶

DEMYELINATING ILLNESS

Demyelinating illnesses, such as multiple sclerosis, can and often do produce vertigo, presumably because there are lesions somewhere in the vestibular system in the brainstem.

VASCULAR DISEASE AFFECTING THE BRAINSTEM

In approaching vascular disease affecting the brainstem, it should be remembered that the most common manifestation of vertebrobasilar insufficiency is vertigo, but vertigo is almost never the *only* manifestation. Such patients can also be expected to complain of double vision, weakness of the limbs, sensory loss, dysarthria, and dysphagia.

DISORDERS OF THE TEMPORAL LOBE

Temporal lobe seizures arising from trauma, tumors, or prior strokes can, as one of their manifestations, produce vertigo.

DRUGS

All drugs that act by intoxicating the reticular activating system in the core of the brainstem - including all anticonvulsants and all sedatives, may produce nystagmus in two different directions in the same position of the head. When the patient looks to the right, the nystagmus beats to the right; when the patient looks to the left, it beats to the left. Overdosage can produce vertigo.

TREATMENT

The treatment of dizziness or vertigo depends specifically on the cause. A detailed account of treatment modalities is out of the scope of this review. However, it should be born in mind that simply going on prescribing the vestibular suppressant drugs when the patient's dizziness is due to orthostatic hypotension or due to some drug toxicity may not solve the purpose, rather may be detrimental. Thus, it is crucial to analyze the symptom of "dizziness" thoroughly to make the actual etiology clear before

deciding the treatment modality.

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