Evaluation of the patient with vertigo

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- "Dizziness" is a nonspecific term often used by patients to describe symptoms

- The most common disorders lumped under this term include vertigo, nonspecific "dizziness," disequilibrium, and presyncope.

- Approximately 40 percent of dizzy patients have peripheral vestibular dysfunction; 10 percent have a central brainstem vestibular lesion; 15 percent have a psychiatric disorder; and 25 percent have other problems, such as presyncope and disequilibrium . The diagnosis remains uncertain in approximately 10 percent. The distribution of causes varies with age. Older adults have a higher incidence of central causes of vertigo (approaching 20 percent), most often due to stroke.

- The patient's description is critical for classifying the etiology of dizziness

- In one series, the history was most sensitive for identifying vertigo (87 percent), presyncope (74 percent), psychiatric disorders (55 percent), and disequilibrium (33 percent)

- The physical examination generally confirmed but did not make the diagnosis. Positional changes in symptoms, orthostatic blood pressure and pulse changes, observation of gait, and detection of nystagmus were most helpful on physical examination

- Asking open-ended questions, listening to the patient's description of his or her symptoms, and checking and gathering additional information from specific questions should allow the clinician to form a hypothesis regarding the type of dizziness

- the clinician should also establish the time course, provoking and aggravating factors, concurrent symptoms, age, preexisting conditions, and the findings on physical examination. These factors are especially useful to narrow the differential diagnosis when the patient's subjective description is difficult to interpret

- Vertigo is a symptom, not a physical sign or a diagnosis
- Vertigo is a symptom of illusory movement.
- Almost everyone has experienced vertigo as the transient spinning dizziness immediately after turning around rapidly several times
- Vertigo can also be a sense of swaying or tilting
- Some perceive self-motion whereas others perceive motion of the environment.
- Patients often experience vertigo as an illusion of motion; some interpret this as self-motion, others as motion of the environment. The most common perception is a spinning sensation; patients may also use terms such as "whirling," "tilting," or "moving." However, not all patients describe their vertigo in such vivid terms. Vague dizziness, imbalance, or disorientation may eventually prove to be due to a vestibular problem

WAYS TO EVALUATE PATIENTS WITH DIZZINESS

A patient with dizziness can be approached in several ways, and what seems easiest or most useful for each clinician may vary by background, practice setting, specialty, and experience. ***No system is necessarily ideal for all specialties in all clinical settings***. All the approaches are similar and involve data gathering in the history and examination, but which data and which steps follow vary slightly in each approach.

Pattern recognition.

This approach focuses on ***using data from the history and examination to identify patterns (or overlapping patterns) that best fit with a specific cause or causes***. The disadvantage is that it takes time to gather details of the history and experience to become familiar with common clinical patterns of disorders that cause vertigo or dizziness. The advantage for the neurologist is that this approach is what neurologists do every day in evaluating patients. For example, neurologists routinely use these methods for evaluating patients with headache, muscle weakness, or unexplained spells

International Classification of Vestibular Disorders classification.

This is not so much an approach as it is an organizational scheme to proceed from symptoms and signs to syndromes to mechanisms in the cause of vertigo and dizziness. It consists of using information from the history and examination to place the patient's symptoms into one of ***three syndrome patterns: (1) acute vestibular syndrome; (2) episodic vestibular syndrome (episodic or triggered); or (3) chronic vestibular syndrome.*** Once categorized in one of these syndrome patterns, each syndrome being associated with a finite list of specific diagnoses or mechanisms .the differential diagnosis can be narrowed and pattern recognition is used to arrive at a specific diagnosis. A limit of this system is that some vestibular diagnoses involve symptoms that cross boundaries between episodic vertigo and chronic vertigo because disorders do not always fit neatly into the syndromic category. In addition, ***when a patient has more than one form of dizziness simultaneously, which occurs in nearly 20% of cases***, this must be recognized and the approach must be applied to each type of dizziness.

TABLE 1-3 **Common Vestibular Disorders Associated With Major Syndrome Categories[a]**

Syndrome	Description	Examples of disorders
Acute vestibular syndrome	A syndrome of acute-onset, continuous vertigo, dizziness, and unsteadiness lasting days to weeks often associated with nausea, vomiting, nystagmus, and vertigo or dizziness aggravated by head motion in any direction	Vestibular neuritis, stroke causing vertigo, acute drug toxicity, demyelinating disease vestibulopathy, Wernicke syndrome, selective serotonin reuptake inhibitor (SSRI) or serotonin norepinephrine reuptake inhibitor (SNRI) discontinuation
Episodic vestibular syndrome	A syndrome of recurrent spells of vertigo, dizziness, or unsteadiness lasting seconds to hours, occasionally days. The episodes may be associated with brief periods of nausea, nystagmus, loss of balance, headache, central nervous system symptoms, or hearing symptoms	Spontaneous: vestibular migraine, Ménière disease, transient ischemic attack (vertebrobasilar insufficiency), vestibular paroxysmia, cardiac causes (aortic stenosis, arrhythmia), episodic ataxias Triggered: benign paroxysmal positional vertigo, orthostatic intolerance or hypotension, motion sickness, central positional vertigo
Chronic vestibular syndrome	A syndrome of chronic vertigo, dizziness, or unsteadiness lasting months to years; symptom descriptions may include gait unsteadiness, ataxia, hearing loss, nausea, nystagmus, or oscillopsia; may result from a progressive neurodegenerative disorder, a static deficit in vestibular function, or evolving symptoms between episodic vestibular episodes	Persistent postural perceptual dizziness, bilateral vestibulopathy, late effects of stroke, cerebellar ataxias, posterior fossa neoplasms, chronic visually induced vertigo or dizziness, mal de débarquement

TiTrATE and ATTEST

TiTrATE is an acronym for ***Timing, Triggers, Associated symptoms and Targeted Examination***. ATTEST is an acronym for ***Associated symptoms, Timing, Triggers, Examination Signs and Testing***. These algorithms use memory aids to help clinicians recall which parts of the history are most essential. However, some pattern recognition is needed; pattern recognition is still very helpful for the most common urgent care causes of dizziness such as BPPV, vestibular neuritis, and stroke presenting with isolated dizziness or vertigo. This system was developed as a way of improving diagnosis, particularly for those in primary care and emergency settings who need to keep their history abbreviated.

Symptom description.

This now-outdated approach introduced in the early 1970s is based on categorizing the patient's description of dizziness as fitting into one of four categories: ***vertigo, presyncope, impaired equilibrium, or nonspecific dizziness***. Data over the past dozen years have made apparent that ***using a symptom characterization as the sole algorithmic branch point leads to more misdiagnosis when compared with using timing and trigger information***. This is especially true in the acute care setting such as in emergency departments. The symptom description approach was introduced at a time when it was believed that the most common single cause of dizziness was "hyperventilation," which was said to account for 22% of dizziness. This was tested by having the patient hyperventilate, and, if the sensation resembled his or her dizzy sensation, then "hyperventilation can often thus be diagnosed without further ado. This is no longer considered valid. Although some value remains in obtaining a description of the patient's sensation, its value in diagnosis tended to be overemphasized by using this approach. ***The description should be combined with other features of the history including onset, duration, triggers, factors that aggravate symptoms, and so on***

Causes of vertigo

Peripheral causes
Benign paroxysmal positional vertigo
Vestibular neuritis
Herpes zoster oticus (Ramsay Hunt syndrome)
Meniere disease
Labyrinthine concussion
Perilymphatic fistula
Semicircular canal dehiscence syndrome
Cogan syndrome
Recurrent vestibulopathy
Acoustic neuroma
Aminoglycoside toxicity
Otitis media
Central causes
Vestibular migraine
Brainstem ischemia
Cerebellar infarction and hemorrhage
Chiari malformation
Multiple sclerosis
Episodic ataxia type 2

Distinguishing vertigo from other types of dizziness

The spinning quality of vertiginous sensations is notoriously unreliable . Lack of spinning cannot be used to exclude vestibular disease, given the difficulty many patients have in putting their dizzy experience into words. On the other hand, some patients with presyncope from vasovagal or cardiac disease can interpret their sensation of dizziness as a spinning sensation

The time course, provoking factors, and aggravating factors of dizziness are more useful features in establishing the cause of dizziness. One study found that many physicians that evaluate patients with dizziness may rely too heavily on symptom quality for diagnosis and do not appreciate the clinical significance of these other features

Time course

- Vertigo is never continuous for more than a few weeks. Even when the vestibular lesion is permanent, the central nervous system adapts to the defect so that vertigo subsides over several weeks

- Constant dizziness lasting months is usually psychogenic, not vestibular. However, the physician must be clear on what a patient means by "constant." Some patients who say they have constant dizziness for months actually mean that they have a constant susceptibility to frequent episodic dizziness; this can be a vestibular problem

- A useful categorization divides patients with vertigo into those with acute prolonged severe vertigo (eg, vestibular neuronitis, stroke), recurrent spontaneous attacks (eg, Meniere disease, vestibular migraine), recurrent positionally triggered attacks (benign paroxysmal positional vertigo), and chronic persistent dizziness (eg, psychogenic, cerebellar ataxia)

Vertigo can occur as single or recurrent episodes and may last seconds, hours, or days. This time course of symptoms provides one of the best clues to the underlying pathophysiology of vertigo

●Recurrent vertigo lasting under one minute is usually benign paroxysmal positional vertigo (BPPV) .

●A single episode of vertigo lasting several minutes to hours may be due to migraine or to transient ischemia of the labyrinth or brainstem.

●The recurrent episodes of vertigo associated with Meniere disease or vestibular migraine also typically last hours but can be briefer .

●More prolonged, severe episodes of vertigo that occur with vestibular neuritis can last for days . This is also characteristic for vertigo originating from multiple sclerosis or infarction of the brainstem or cerebellum

Provoking factors

Certain types of vertigo occur spontaneously, while others are precipitated by maneuvers that change head position or middle ear pressure (eg, coughing, sneezing, or Valsalva maneuvers). Positional vertigo and postural presyncope are two common conditions that are frequently confused. Both are associated with dizziness upon standing, as when arising from bed. The key to the diagnosis is to determine whether dizziness can be provoked by maneuvers that change head position without lowering blood pressure or decreasing cerebral blood flow. Such maneuvers include lying down, rolling over in bed, and bending the neck back to look up. Dizziness in these settings suggests positional vertigo, not postural presyncope.

Aggravating factors

All vertigo is made worse by moving the head. This is a useful feature for distinguishing vertigo from other forms of dizziness. Many patients in the midst of a vertiginous attack are petrified to move. If head motion does not worsen the feeling, it is probably another type of dizziness

Certain kinds of movements may increase suspicion of damage to the otoliths, the organs that detect linear accelerations to the head. These include imbalance provoked by stop-and-go movements of elevators or cars in traffic, as well as standing on a boat .Attacks of BPPV are often provoked by specific head movements or postures (eg, rolling over in bed, extending the neck).

Vertigo aggravated by coughing, sneezing, exertion, or loud noises (Tullio phenomenon) should raise suspicion of either a perilymphatic fistula, in which there is an abnormal connection between the middle ear and the perilymphatic space of the inner ear, or a superior semicircular canal dehiscence, in which there is a defect in the roof of the superior semicircular canal. Both conditions allow pressure to be transmitted from the cerebrospinal fluid (CSF) space to the inner ear

Head trauma is an important historical feature and can produce vertigo by a variety of mechanisms Barotrauma, middle ear surgery, and straining with weight-lifting and bowel movements have been reported to produce a perilymphatic fistula.
Recent hyperextension injury to the neck, usually with persistent neck pain, suggests the possibility of vertebral artery dissection with brainstem or labyrinthine ischemia.
Recent viral symptoms may suggest acute vestibular neuritis, which is believed to be produced by viral or postviral inflammation of the eighth cranial nerve. However, a history of recent viral illness is both nonspecific and insensitive; less than one-half of patients with vestibular neuritis will report this

Associated signs and symptoms

Vertigo, whether of central or peripheral origin, is generally accompanied by nystagmus and postural instability. Other signs and symptoms may be useful in distinguishing between central and peripheral causes of vertigo.

- Acute vertigo due to a vertebrobasilar stroke is almost always accompanied by other evidence of brainstem ischemia such as diplopia, dysarthria, dysphagia, weakness, or numbness . However, infarction of the cerebellum may present as vertigo with no other symptoms. Focal neck pain may suggest vertebral artery dissection.
- Vertigo in patients with multiple sclerosis may also be preceded by or associated with other neurologic dysfunction, depending on the locus of demyelination.
- Deafness and tinnitus suggest a peripheral lesion of the inner ear. A sensation of aural fullness typically accompanies attacks of Meniere disease
- Headache, photophobia, and phonophobia suggest migrainous vertigo. Many patients with migrainous vertigo will also experience visual aura in at least some of their attacks.
- Shortness of breath, palpitations, and sweating may suggest a panic attack but can occur with vertigo too. Vertigo is often so terrifying that such symptoms are not uncommon with vestibular disease

IMPACT ON QUALITY OF LIFE.

Establishing the impact on the patient's life is important in gauging how aggressive to be in the workup and treatment. Along with this assessment should be a discussion on what the patient expects or would like from the visit. Some patients may have what seems to be fairly minor dizziness, but they are very worried about it due to their own concerns with having something serious. In some of those cases, once they feel it has been adequately established to be benign, they do not want any medication but would consider other approaches. Some patients, particularly those with persistent postural perceptual dizziness (also referred to as PPPD), may have a normal examination but view their lives as severely negatively impacted by thesymptoms. Knowing this will help produce a better treatment plan and a more agreeable experience and outcome for the patient

Prior medical history

●A prior history of migraine suggests that this may be the etiology of vertigo.

●The presence of stroke risk factors such as hypertension, diabetes mellitus, smoking, and a history of vascular disease support a diagnosis of vertebrobasilar ischemia. Patients with an episode of vertigo and one or more risk factors for stroke have a substantial risk of subsequent stroke: an 8 percent two-year risk with one or two risk factors and 14 percent two-year risk with three or more risk factors .

●Past head trauma is a common antecedent to BPPV and persistent postural perceptual dizziness.

●Certain medications are associated with vestibular (eg, cisplatin, aminoglycosides) or cerebellar (eg, phenytoin) toxicity.

PREVIOUS TREATMENTS TRIED.

In some cases, if no treatment has yet been tried or it is the patient's first medical evaluation, this is a brief conversation. In other cases, many opinions and tests have been offered but no actual treatments have been attempted. If a patient has been treated, the previous treatment can be noted; if it failed in the past, perhaps it should not be repeated. Some patients may have symptoms amenable to physical therapy but say it did not work in the past. It can be helpful to ask the patient to recount what was done by the therapist and how many sessions were attended. Some patients fail to respond because the therapist mistakenly tried to treat for BPPV but the patient actually has vestibular neuritis or another condition. Other patients may not have complied with a reasonable trial because they were not convinced it would help. In other cases, a medication may have been used but at such a low dosage or for such a short therapeutic trial it should be reconsidered.

PRESYNCOPE

Presyncope is the prodromal symptom of fainting or a near faint. Presyncope occurs more commonly than syncope. It usually lasts for seconds to minutes and is often recognized by the patient as "nearly blacking out" or "nearly fainting." When the symptoms are less intense, their description may be less clear. Patients may also report lightheadedness, a feeling of warmth, diaphoresis, nausea, and visual blurring occasionally proceeding to blindness. An observation of pallor by onlookers usually indicates presyncope. Presyncope usually occurs when the patient is standing or seated upright and not when supine (if the latter, one should suspect a cardiac arrhythmia rather than hypotension).

A history of cardiac disease, including cardiac dysrhythmias (tachycardias or bradyarrhythmias), coronary heart disease, and congestive heart failure, is relevant . The patient should be asked specifically about palpitations, chest discomfort, or dyspnea (although this may suggest anxiety as an alternative cause as well).

The etiology and evaluation of presyncope are the same as for syncope. Orthostatic hypotension, cardiac arrhythmias, and vasovagal attacks are some of the more common causes

DISEQUILIBRIUM

Disequilibrium is a sense of imbalance that occurs primarily when walking. Chronic dizziness or disequilibrium can cause significant impairment of physical and social functioning, particularly in older adults .

Disequilibrium may result from peripheral neuropathy, a musculoskeletal disorder interfering with gait, a vestibular disorder, a cerebellar disorder, and/or cervical spondylosis . Patients with Parkinson disease frequently suffer from disequilibrium and are subject to postural hypotension as well as imbalance . Cervical spondylosis may be associated with dizziness that is apparently related to a disturbance in postural control , although this is not a universally accepted cause of dizziness . Visual impairment, whether from underlying eye disease or poor lighting, typically exacerbates the sense of imbalance. This is also true of cerebellar disorders. Cerebellar disorders can affect mainly gait, but often have associated dysarthria and eye signs, such as gaze-evoked nystagmus, poor smooth pursuit, and downbeat nystagmus. If the cerebellar hemisphere is also involved, there will be incoordination of limbs.

The physician should inquire about symptoms of neurologic and gait disorders, especially those suggestive of parkinsonism, cerebellar incoordination, or peripheral neuropathy. In the series cited above, few patients volunteered that their dizziness was associated with walking, standing, turning, or falling; most with disequilibrium required observation of gait and a neurologic examination to identify the diagnosis

NONSPECIFIC DIZZINESS

Nonspecific dizziness is often difficult for the patient to describe. He or she may simply insist, "I am dizzy." Patients may choose from suggested descriptions to say they are "giddy" or "lightheaded"; however, they may also endorse a fainting or spinning sensation.

Psychiatric disorders may be the primary cause of nonspecific dizziness in some cases . One-quarter of such individuals had major depression, one-quarter had generalized anxiety or panic disorder, and the remainder had somatization disorder, alcohol dependence, and/or personality disorder in one series . Other series report higher rates of panic disorder

Nonspecific dizziness is sometimes related to hyperventilation. This usually occurs in settings that are at least mildly stressful. Dizziness that accompanies hyperventilation, anxiety, or depression often builds up gradually, waxes and wanes over a period of 20 minutes or longer, and gradually resolves. There may be no sensation of "air hunger" since these patients are hyperventilating only to a slight degree.

- There are no physical signs that are diagnostic of nonspecific dizziness. Most patients are healthy, young individuals without detectable disease involving the neurologic, cardiovascular, or otolaryngologic systems. Purposeful hyperventilation is one means to confirm that diagnosis. The patient is coached to hyperventilate until he or she becomes dizzy, then to identify whether or not the dizziness mimics spontaneously occurring symptoms. If so, the patient will be convinced, as well as the physician, that hyperventilation is the etiology. However, the examiner must observe the eyes of the patient to see if there is nystagmus; some pathologic vestibular lesions are exacerbated or unmasked by hyperventilation. If nystagmus is seen, the diagnosis is a vestibular lesion, not hyperventilation.

- Reproducing symptoms by hyperventilation is often reassuring to the patient and in itself therapeutic. It is possible for individuals to learn to breathe less deeply and through the nose, thereby limiting hyperventilation. If patients understand that a number of minutes must elapse before the symptoms resolve, they can spontaneously abort their own attacks. Treatment of anxiety or depression with pharmacotherapy should be based upon the symptoms of these disorders, not necessarily upon the presence of nonspecific dizziness

DIZZINESS IN OLDER PATIENTS

- Dizziness in the older adult deserves specific mention because of its high prevalence, up to 38 percent in some series, and its attendant risk of falls, functional disability, institutionalization, and even death

- Assessment of dizziness in older patients is challenging because it is frequently attributable to multiple problems, including vertigo, cerebrovascular disease, neck disorders, physical deconditioning, and medications . Visual impairment from cataracts and other conditions is common in older adults and likely exacerbates the disability that is associated with dizziness . One study found that 44 percent of patients aged 65 to 95 years had more than one condition causing dizziness .Some call this entity multiple-sensory defect dizziness.

In a population-based study of 1087 community-living individuals 72 years of age or older, 261 (24 percent) reported having an episode of dizziness during the two months prior to study onset and that the dizziness (whether persistent or intermittent) had been present for at least one month . The investigators found seven characteristics that were independently associated with dizziness on multivariate analysis:

- Anxiety trait
- Depressive symptoms
- Impaired balance (path deviation and time to turn circle greater than four seconds)
- Past myocardial infarction
- Postural hypotension (mean decrease in blood pressure ≥20 percent)
- Five or more medications
- Impaired hearing

	Vertigo	Presyncope	Disequilibrium	Nonspecific Dizziness
Symptom description	Illusion of motion, imbalance	Going to pass out, faint	Imbalance, unsteady, symptoms not in the head	Light-headed, foggy, floating
Onset	Usually sudden	Usually sudden	Sudden to slow	Poorly defined
Duration	Seconds to hours	Seconds to minutes	Acute to chronic	Subacute to chronic
Triggers	Head motion, position change	Orthostatic maneuvers, urination, cough, dehydration	Standing or walking, not when sitting or lying	Stress, situational, nonspecific
History	None or episodes	None or episodes	Chronic	Chronic
Associated symptoms	Nausea, ear symptoms (hearing loss, tinnitus), brainstem symptoms (diplopia, slurring, numbness, weakness, incoordination, ataxia)	Graying vision, warmth, diaphoresis, nausea, palpitations, chest pain	Slurring, incoordination	Many

Examination

Nystagmus

in a patient with acute vertigo, nystagmus is usually visible with the patient looking straight ahead. If the lesion is peripheral, the fast phase is away from the affected side. Usually, nystagmus increases in frequency and amplitude with gaze toward the side of the fast phase, eg, leftward gaze increases left-beating nystagmus, if present (Alexander law).

- Type of nystagmus. A mixed horizontal-torsional jerk nystagmus results if a peripheral lesion affects all three semicircular canals or the vestibular nerve on one side. The horizontal fast phases beat toward the normal ear, as do the upper poles of the eyes for the torsional fast phases. The jerk nystagmus from peripheral disease occasionally appears purely horizontal, but it is never purely torsional or vertical. (Also, pendular nystagmus is never due to peripheral vestibular disease.) The jerk nystagmus with central lesions may have any trajectory.

- Visual fixation tends to suppress nystagmus that is due to a peripheral lesion, but it does not usually suppress nystagmus from a central lesion . This can be tested with Frenzel lenses, which are large magnifiers that blur vision and prevent visual fixation. A peripheral lesion is likely if nystagmus increases when Frenzel lenses are in place

- Another way to test the effect of fixation is by covering and uncovering one eye during fundoscopy of the other. One group of investigators has proposed that examination with a penlight (substituting for the ophthalmoscope) may also allow examination for nystagmus with and without fixation . A peripheral disorder is likely if nystagmus increases on covering the fixating eye. It should be kept in mind that, in the ophthalmoscopic examination, the direction of nystagmus appears reversed because the optic nerve head is behind the center of eye rotation

- Testing nystagmus in different gaze positions can provide other localizing clues. In peripheral lesions, the predominant direction of nystagmus remains the same in all directions of gaze. Nystagmus that reverses direction (ie, right-beating in right gaze then left-beating in left gaze) suggests gaze-evoked nystagmus, which is due to an abnormality of central circuits . However, the absence of this feature does not rule out a central cause of vertigo. Nystagmus that reverses direction with convergence also suggests a central lesion

TABLE 5.3 Peripheral Versus Central Nystagmus

	Peripheral	Central
Appearance	Combined torsional, horizontal, and vertical Nystagmus beats away from the affected side.	Often pure vertical, horizontal, or torsional; any trajectory
Fixation	Inhibits	No effect
Gaze	Obeys Alexander law (Nystagmus increases when looking toward the side of the fast phase.)	May change direction; does not obey Alexander law

Balance and gait

The ability to stand or walk unsupported and the direction of falling may provide useful clues to the origin of vertigo, although it may be difficult to persuade a patient with severe vertigo to attempt to walk.

Unilateral peripheral disorders generally cause patients to lean or fall toward the side of the lesion. Patients may be uncomfortable and reluctant to move because of their vertigo, but they can still walk. Romberg testing will cause the patients to fall or tilt to one side.

Patients with an acute cerebellar stroke are often unable to walk without falling. The direction of tilting or falling with Romberg testing may vary.

The sensitivity of balance testing may be increased by other maneuvers such as eye-closing, standing on foam, and performing head movement

Other neurologic signs

- A careful neurologic examination should be performed since the presence of additional neurologic abnormalities strongly suggests the presence of a central lesion. A search should be made for cranial nerve abnormalities, motor or sensory changes, dysmetria, or abnormal reflexes.

- In particular, the abnormal facial sensation, ptosis, anisocoria, and diplopia of a lateral medullary infarction may be overlooked by both patients and clinicians when the vertigo is profound.

- However, the absence of other neurologic signs does not exclude a central process. In particular, a midline or inferior cerebellar infarction may produce no neurologic signs other than nystagmus and gait instability on examination

Office hearing tests

- Bedside tests of hearing and an examination of the tympanic membrane can be useful in distinguishing the etiology of vertigo. The otoscopic examination provides evidence of acute or chronic otitis media.

- The examiner can easily test hearing by several methods. One is to softly whisper into each ear and then ask the patient to repeat what was whispered. Another is to hold the examiner's hands next to the patient's ears but out of the patient's eyesight. The examiner then rubs the fingers together on one side, with a sham rubbing movement on the opposite side, and asks the patient to report when the finger scratching sound is heard and from which ear it is heard. Another method is to vibrate a 512 Hz tuning fork, placing it close to one ear and then the other in rapid succession so that the patient can compare the loudness.

Interpreting Weber and Rinne tests: Conductive versus sensorineural hearing loss

Interpreting Weber and Rinne tests: Conductive versus sensorineural hearing loss

	Weber lateralizes	Rinne test
Conductive loss		
Good ear		AC > BC
Bad ear	To bad ear	BC > AC
Sensorineural loss		
Good ear	To good ear	AC > BC
Bad ear		AC > BC*

AC > BC: Air conduction better than bone conduction (normal Rinne).

BC > AC: Bone conduction better than air conduction (abnormal Rinne).

* For patients with severe sensorineural hearing loss, the patient may report BC > AC because the sound is being sensed by the "good" (contralateral) ear.

Unilateral sensorineural hearing loss suggests a peripheral lesion; audiometry is required for confirmation. If no obvious cause of unilateral sensorineural hearing loss (eg, Meniere disease) has been identified by history, a magnetic resonance imaging (MRI) or computed tomography (CT) scan of the posterior fossa and internal auditory canal is necessary.While associated hearing loss strongly points to a peripheral origin of vertigo, the absence of hearing loss has less localizing value

Dix-Hallpike maneuver — Positional maneuvers are designed to produce vertigo and elicit nystagmus in patients with a history of positional dizziness. These maneuvers are most useful in patients who do not have symptoms or nystagmus at rest and whose vertigo is episodic. The Dix-Hallpike maneuver tests for canalithiasis of the posterior semicircular canal, which is the most common cause of BPPV. Other maneuvers are used to provoke the nystagmus of less common variants of BPPV (anterior canal, horizontal canal).

The latency, transience, and fatigability, coupled with the typical mixed upward vertical and torsional direction, are important features in diagnosing BPPV due to posterior canalithiasis . Deviation from these features may occur with rarer types of peripheral positional vertigo, such as those due to anterior or horizontal canalithiasis, but should also raise suspicion of a central lesion.

Head impulse test — The head impulse test (or head thrust test) is performed by instructing the patient to keep his or her eyes on a distant target while wearing his or her usual prescription eyeglasses. The head is then turned quickly and unpredictably by the examiner, approximately 15°; the starting position should be approximately 10° from straight ahead.

The normal response is that the eyes remain on the target . The abnormal response is that the eyes are dragged off of the target by the head turn (in one direction), followed by a saccade back to the target after the head turn; this response indicates a deficient VOR on the side of the head turn, implying a peripheral vestibular lesion (inner ear or vestibular nerve) on that side

- For distinguishing vestibular dysfunction from nonvestibular dizziness, the head impulse test is reported to have a higher specificity (82 to 100 percent) than sensitivity (34 to 39 percent) . However, the use of caloric testing as the gold standard in these studies may not be appropriate and may underestimate sensitivity . In one report, flexing the head forward 30° during the test increased sensitivity to as high as 71 to 84 percent .
- An abnormal head impulse test is reported to be a useful test to distinguish between central and peripheral vertigo, particularly in the setting of acute prolonged vertigo, when the examiner is trying to differentiate between vestibular neuritis and cerebellar infarction . The head impulse test is normal in most patients with isolated cerebellar lesions (31 of 34 in one series) . It is important to note that a peripheral injury may result from infarction to the inner ear or eighth cranial nerve, though

Other vestibular signs

- **Skew deviation** – A skew deviation is a vertical misalignment of the two eyes resulting from a supranuclear (relative to the ocular motor nuclei) pathology, usually located in the brainstem . This may also be caused by a vestibular lesion because of imbalance in otolithic-ocular reflexes.

- **HINTS examination** – An examination that includes the head impulse test (ie, head thrust test), evaluation for direction-changing (ie, gaze-evoked) nystagmus, and a test of skew has been called the HINTS examination .The presence of any one of three clinical signs (a normal head impulse test, direction-changing nystagmus, or a skew deviation) suggests central rather than peripheral vertigo in patients with an acute sustained vestibular syndrome

- **Ocular tilt reaction** – The OTR is a triad of skew deviation, torsional tilt of the eyes with the upper poles tilted toward the eye that is lower, and head tilt toward the eye that is lower. This typically results from a central lesion affecting otolithic pathways.

- **Tilt of the subjective visual vertical** – Tilt of the SVV is a sensitive sign of acute static vestibular imbalance attributed to dysfunction of the utricular component of the otoliths. SVV tilt can be measured at the bedside using a simple device made from a bucket with a straight line drawn on the bottom

- **Head-shaking visual acuity** – Head-shaking visual acuity is tested by having the patient look at an eye chart in the distance wearing his or her customary distance vision eyeglasses. Customary eyeglasses are important since the VOR is calibrated for a given rotational magnification that varies with different strengths of glasses. The patient reads the eye chart while his or her head is shaken continuously over a small range at approximately 2 Hz. Then, the patient reads the chart again while his or her head is still. Head-shaking acuity that is more than four lines worse than the head-still acuity indicates a poor VOR

Head-shaking nystagmus

Head-shaking nystagmus is elicited by the patient shaking the head from side to side for 15 to 40 seconds with eyes closed or Frenzel lenses in place. The shaking causes vigorous stimulation of the horizontal semicircular canals of both sides; head velocity data are stored in the cerebellum, decaying slowly after the head stops moving. When the shaking stops, the patient then opens his or her eyes and attempts to look straight ahead. If both sides were equally activated, the decay of velocity information will be balanced, and the eyes will be still.

If there is unilateral labyrinthine damage, the asymmetric neural output will generate a nystagmus that beats away from the damaged side. This test will be normal in patients with bilateral symmetric disease, unlike rapid head thrusts or caloric responses. The test is abnormal in patients with unilateral central vestibular lesions

Caloric testing

Lack of a response to warm or cold water on one side suggests disease on that side, usually in peripheral lesions. Central vestibular disorders may also cause caloric hyposensitivity, especially if they involve the vestibular root entry in the medulla. This test can cause significant distress, including nausea and vomiting in awake patients; it is rarely performed in the office evaluation of vertigo but may be useful in the vestibular laboratory . Caloric testing is also used in the evaluation of coma.

●**Others** – Hyperventilation can elicit nystagmus in several central and peripheral vestibular pathologies, and may be a particularly sensitive test in patients with cerebellopontine tumors. In one study, ipsilesional hyperventilation-induced nystagmus was found in 84 percent of patients with cerebellopontine tumors compared with 34 percent in other vestibular diseases

Clinical features of peripheral versus central vertigo

Clinical features of peripheral versus central vertigo

	Peripheral	Central
Nystagmus		
Features (direction and type)	Unidirectional, fast component toward the normal ear; never reverses direction Horizontal with a torsional component; never purely torsional or vertical	Sometimes reverses direction when patient looks in the direction of slow component Can be any direction; note that purely vertical or purely torsional nystagmus is a central sign
Effect of visual fixation	Suppressed	Not suppressed
Postural instability	Unidirectional instability, walking preserved	Severe instability, patient often falls when walking
Deafness or tinnitus	May be present	Usually absent
Other neurologic signs and symptoms	Absent	Often present (eg, diplopia, ataxia, dysarthria, dysphagia, focal or lateralized weakness)

TABLE 5.3 Peripheral Versus Central Nystagmus

	Peripheral	Central
Appearance	Combined torsional, horizontal, and vertical Nystagmus beats away from the affected side.	Often pure vertical, horizontal, or torsional; any trajectory
Fixation	Inhibits	No effect
Gaze	Obeys Alexander law (Nystagmus increases when looking toward the side of the fast phase.)	May change direction; does not obey Alexander law

Features that Suggest **Central Vestibular Dysfunction**

(eg, brainstem or cerebellar stroke)

VS

Features that Suggest **Peripheral Vestibular Dysfunction**

(eg, vestibular neuritis, labyrinthitis)

The presence of any **ONE** feature suggests **central** origin:

Neurologic Examination

Assess for findings such as:
Diplopia, weakness, numbness, anisocoria, ataxia, dysarthria

Focal Neurologic Findings **PRESENT**

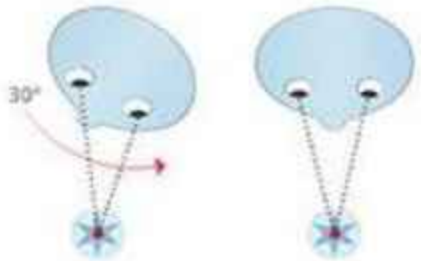
(Absence does not rule out central origin.)

Focal Neurologic Findings **ABSENT**

HINTS Examination

(three-part examination)

NORMAL

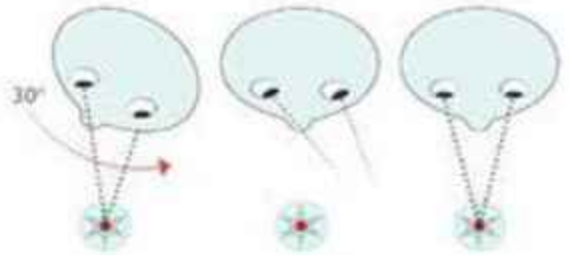


Vestibulo-ocular reflex is preserved.
Eyes are able to remain fixed on target despite head movement.

Head Impulse Test

The patient is asked to fixate on a target *(eg, examiner's nose)*. The examiner gently turns the patient's head approximately 30 degrees to one side. The examiner rapidly turns the head back to midline. The eye movements are assessed. The maneuver is repeated with a head turn in the opposite direction.

ABNORMAL



Vestibulo-ocular reflex is not preserved.
Head movement causes eyes to move off target. This is followed by a quick saccade *(rapid jerky eye movement)* back to target. The abnormal finding will occur with a head turn in only one direction.

DIRECTION-CHANGING NYSTAGMUS

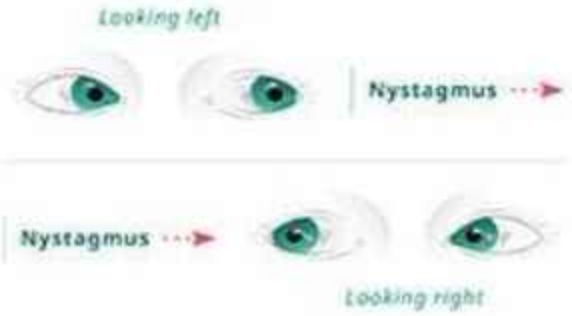
Direction of nystagmus **differs** when the patient looks left and right.

Nystagmus

The patient is asked to look to the left and then to the right. The direction of the fast phase of the beating of nystagmus is assessed.

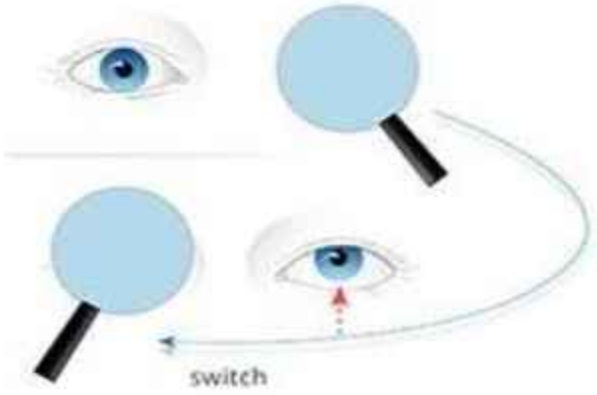
Looking left *Looking right*

UNIDIRECTIONAL NYSTAGMUS



Direction of nystagmus **remains the same** when the patient looks left and right.

SKEW DEVIATION

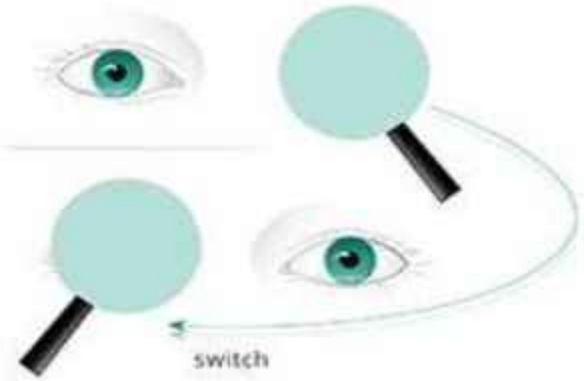


Eye exhibits vertical or diagonal movement when cover is moved.

Test of Skew

The patient is asked to look at the examiner's nose. The examiner covers the left eye then quickly moves the cover to the right eye. The left eye is observed for vertical *(or diagonal)* movement. The cover is moved back to the left eye. The right eye is assessed for vertical *(or diagonal)* movement.

ABSENT SKEW



Neither eye exhibits vertical or diagonal movement when cover is moved.

KEY CONCEPTS

Brainstem or cerebellar STROKE is the most common central cause of acute-onset sustained vertigo in adults, making prompt identification of patients with vertigo of central origin essential. The presence of any ONE focal neurologic finding or HINTS examination finding that suggests central vestibular dysfunction *(normal head impulse test, direction-changing nystagmus, or skew deviation)* is an indication for URGENT NEUROIMAGING.

A peripheral cause of acute-onset sustained vertigo is most likely in patients who have ALL of the following:

- No focal neurologic findings
- Abnormal head impulse test
- Unidirectional nystagmus
- Absent skew

Associated hearing loss and/or tinnitus also supports peripheral origin. Vestibular neuritis and labyrinthitis are the primary peripheral causes of acute-onset sustained vertigo.

Rarely, the HINTS examination findings can be misleading. In patients with inner ear infarction resulting from a vertebrobasilar ischemic stroke, the HINTS examination findings are identical to those in peripheral vestibular dysfunction. Therefore, patients with high baseline risk for stroke *(eg, older adults with cardiovascular risk factors)* who present with acute-onset sustained vertigo are also candidates for urgent neuroimaging.

Another practical approach to diagnosis often starts with the time course of symptoms, in part because patients are more reliable at describing the duration rather than the quality of their symptoms.

●Acute onset, sustained vertigo

Common diagnoses in this setting include vestibular neuritis, demyelinating disease, and a stroke in the brainstem or cerebellum. Clinicians use the presence of cerebrovascular risk factors and associated neurologic deficits to determine the relative probability of a cerebrovascular etiology . The HINTS examination, in particular the head impulse test, can also be particularly useful in this setting to distinguish a peripheral cause of vertigo (eg, vestibular neuritis) from a central, cerebrovascular cause . In a young patient without cerebrovascular risk factors in whom a normal head impulse test suggests a localization within the central nervous system, multiple sclerosis might be more likely. However, the utility of the head impulse test or the HINTS examination has not been specifically studied in this setting. An MRI focusing on the brainstem should be performed when a central nervous system etiology is suspected.

●**Episodic vertigo** – Likely diagnoses in patients with episodic vertigo depend on the duration of events as well as the presence of associated features:

•Very brief episodes of isolated vertigo that are precipitated by predictable movements or positions of the head are often caused by benign paroxysmal peripheral vertigo (BPPV). The Dix-Hallpike maneuver can help confirm this diagnosis.

•The diagnosis of episodes with a longer duration (minutes to hours) may be further distinguished by the presence or absence of associated clinical features. As examples, associated headache suggests vestibular migraine; unilateral hearing loss, tinnitus, and ear fullness suggest Meniere disease; and other brainstem neurologic deficits suggest vertebrobasilar transient ischemia. The diagnoses of these conditions are discussed separately.

TABLE 5.5 Causes of Peripheral Vertigo

Cause	Characteristics
BPPV	Brief, recurrent, positional nystagmus only in provoking position
Vestibular neuritis	Acute, single episode, viral prodrome
Ramsay Hunt syndrome (herpes zoster oticus)	Acute, single episode, vesicles in/near the ear, facial nerve palsy, deafness
Ménière syndrome	Recurrent, last minutes to hours, unilateral ear symptoms of fullness, hearing loss, tinnitus
Trauma	History of trauma
Perilymphatic fistula	Episodic, associated with Valsalva, loud sounds (Tullio phenomenon), history of trauma
Superior canal dehiscence	Episodic, associated with Valsalva, loud sounds
Cogan syndrome	Ménière-like syndrome with interstitial keratitis
Acoustic neuroma	Rare vertigo, more imbalance, unilateral hearing loss or tinnitus
Medications	Aminoglycoside exposure
Otitis	Evidence of otitis
Labyrinth ischemia	Presence of vascular risk factors, sudden vertigo, and hearing loss
Recurrent vestibulopathy	Recurrent attacks but without the ear symptoms to suggest Ménière syndrome

Laboratory Testing

Blood tests for vertigo are rarely helpful as a matter of routine but may be indicated in some cases. ***Patients taking antiepileptic drugs that may account for dizziness or abnormal eye movements may need drug levels assessed. For patients with impaired balance, vitamin B12, methylmalonic acid, hemoglobin A1c, and thyroid function studies may be warranted. For patients with possible orthostatic dizziness, a complete blood cell count and comprehensive metabolic panel may be ordered.*** In patients with bilateral fluctuating hearing with or without vertigo and in whom luetic otitis or autoimmune inner ear disease is suspected***, an antinuclear antibody screen, erythrocyte sedimentation rate, and ests for syphilis*** can be obtained. Patients presenting with ataxia should have a test for ***anti–glutamic acid decarboxylase 65 (GAD65) antibodies and possibly vitamin E level.*** Although anti-GAD65 antibodies are associated with stiff person syndrome, in some people high levels of anti-GAD65 antibodies may manifest primarily with cerebellar ataxia with imbalance and "dizziness," oftentimes with downbeating nystagmus that is amenable to treatment with immunotherapy (eg, mycophenolate, cyclosporine, rituximab, cyclophosphamide). For anyone suspected of having dizziness in association with Wernicke syndrome, assessment and possible empiric treatment for this condition should be considered

Cardiac and Hemodynamic Testing

If cardiogenic near-syncope is suspected, an ***ECG, echocardiogram, Holter monitoring, and cardiology referral*** can be obtained. ***Tilt-table testing*** can be helpful in patients with unexplained syncope but also in those with suspected recurrent, unexplained near-syncopal dizziness

Vestibular Testing

Vestibular testing is performed to determine the functionality and integrity of **the peripheral vestibular apparatus and pathways**, so ***it should generally be done to confirm or refute a hypothesis***. Of course, like many other tests in medicine, it can be sometimes helpful in situations in which the cause is very uncertain to at least ascertain that the vestibular structures and reflexes remain intact..

Imaging Studies

Imaging may be indicated ***when the cause of dizziness is uncertain or the examination reveals findings of CNS dysfunction***. ***For patients with dizziness and vertigo, a noncontrast head CT has a very low yield of identifying a cause when patients with headache, trauma to the head and neck, altered mental status, focal neurologic deficits, or recent head or neck surgery are excluded.*** In one prospective analysis of patients presenting to an emergency department with dizziness, none of the 200 studies found a causative lesion, and head CT in this setting was deemed not cost effective. ***Temporal bone CT*** is indicated to identify lesions such as cholesteatoma or lesions within the labyrinth, including canal dehiscence. ***Brain MRI without contrast is a reasonable first step, and MRI with and without contrast is warranted if a vestibular schwannoma or other structural lesion of the cerebellopontine angle is a consideration.***

Head and neck CT angiography (CTA) or head and neck magnetic resonance angiography (MRA) may be appropriate when dizziness or vertigo may have a vascular cause