GETTING A GRIP ON VERTIGO

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Vertigo is an illusion of movement due to an imbalance of tonic vestibular activity. It is usually rotatory, but sensations of body tilt or impulsion may also occur. Vertigo is commonly associated with nystagmus, oscillopsia, postural imbalance, and autonomic symptoms (e.g., sweating, pallor, nausea, vomiting). Unfortunately, the term is used inconsistently by clinicians.

Precipitating factors for vertigo may include head movements, coughing, sneezing and loud noises. Head movements attributed to imbalance within vestibular pathways and may produce vertigo even after compensation has occurred in response to a vestibular lesion. In addition, positional vertigo is frequently induced by ordinary head movements, such as looking up or to one side, lying down, sitting up or bending over. Coughing and sneezing may precipitate vertigo, particularly by changing middle ear pressure in patients with a post-traumatic perilymph fistula. Loud noises may also precipitate vertigo in patients with inner ear disease, such as Ménière disease; this is called the Tullio phenomenon. This article reviews the clinical presentation of vertiginous symptoms, offers advice on differential diagnosis, and recommends options for management.

Localization
Vertigo indicates dysfunction or imbalance within the central or peripheral vestibular pathways. Vertigo is usually rotatory, implying a disturbance of the semicircular canals or their central connections. Sensations of body tilt or impulsion indicate otolithic disturbances or dysfunction of central otolithic connections.

Neurologic symptoms associated with vertigo are particularly helpful in localizing the responsible lesions. Hearing loss and tinnitus generally imply peripheral dysfunction, usually involving the inner ear, but occasionally involving the internal auditory canal or the structures of the cerebellopontine angle. Because the motor fibers for facial expression pass in the seventh cranial nerve in close proximity to the vestibulo-cochlear sensory fibers in the eighth cranial nerve, peripheral-type facial paresis (involving both upper and lower facial muscles) may be associated with lesions of the internal auditory canal, cerebellopontine angle, or brainstem. Any of the following imply an intracranial basis for the dysfunction: diplopia, facial numbness, dysarthria, dysphagia, extremity weakness or numbness or incoordination.

Patients with vertigo often give confusing and contradictory accounts of the directionality of their symptoms, most likely because the vestibular and self-referred visual sensations of movement are oppositely directed. Therefore, it is helpful to determine the direction of the sensation of rotation of the body with the eyes closed, as this directional sensation is away from the side of a peripheral vestibular lesion.

Differential Diagnosis
Careful history, provocative testing and detailed examination will allow distinction of the major categories of dizziness in most cases, and will often allow a specific etiologic diagnosis as well. Particular attention should be given to the onset, duration and course of the vertigo, as well as any associated autonomic, auditory, or central nervous system signs and symptoms. This information alone is often sufficient to suggest a specific etiologic diagnosis.

Vertigo is usually rotatory, and patients frequently describe it as “spinning,” but they may also describe sensations of body tilt or impulsion. Vertigo is commonly associated with nystagmus, oscillopsia, postural imbalance, nausea, and vomiting. Autonomic symptoms (e.g., sweating, pallor, nausea, vomiting) are generally more severe with vertigo of peripheral origin than with vertigo of central origin. Common causes of vertigo in the elderly include benign paroxysmal positional vertigo, viral neurulabyrinthitis, trauma, toxins, and posterior circulation or labyrinthine ischemia. Common causes of vertigo in young adults include Meniere syndrome, viral neurulabyrinthitis, trauma, and toxins. Common causes of vertigo in children with normal ear drums (i.e., those who do not show middle ear effusion or otitis media) are migraine and benign paroxysmal vertigo.

Episodic vertigo is generally of abrupt onset, but the duration of episodes varies considerably. Episodes of benign paroxysmal positional vertigo last seconds (up to a minute), whereas seizures generally last seconds or minutes, migraine aurae last minutes, transient ischemic attacks last minutes to hours (up to one day, by definition, but generally less than six hours), and attacks of Meniere syndrome last hours. Among causes of episodic vertigo, benign paroxysmal positional vertigo, migraine and Meniere syndrome are most commonly associated
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with autonomic symptoms. Similarly, transient ischemic attacks, migraine, and seizures are associated with central nervous system symptoms or signs. Meniere syndrome typically has episodes accompanied by auditory symptoms.

Monophasic vertigo may have an abrupt onset (e.g., with trauma, stroke, or demyelinating disease), a subacute onset (e.g., with vestibular neuronitis), or a subacute to chronic onset (e.g., with toxic vestibulopathy or posterior fossa masses). Autonomic symptoms and auditory symptoms are most common and generally most severe with peripheral vestibulopathies, such as with vestibular neuronitis or toxic vestibulopathies. Peripheral vestibulopathies are not associated with central nervous system symptoms and signs, whereas they are common or typical for the other causes of monophasic vertigo.

**DIAGNOSTIC WORKUP**

The history and physical examination are frequently sufficient to classify vague complaints of dizziness into one of the major categories such as vertigo, and perhaps even to suggest an etiologic diagnosis. Sometimes, however, patients’ descriptions of dizzy sensations are confusing, particularly when the events are episodic. Therefore, provocative testing is helpful in defining the subjective sensation and the often vague descriptions of dizziness and vertigo. Provocative testing can be used to produce physiologic sensations of vertigo that can then be compared and contrasted with the subjective sensations experienced by the patient.

Physiologic vertigo can be induced in the office either by rotational or caloric testing. Rotational testing in the office is performed by seating the patient in a rotary office chair with the head tilted 30 degrees forward, and then rotating the patient carefully 10 times over 20 seconds to 30 seconds. Tilting the head forward 30 degrees places both horizontal semicircular canals parallel to the floor; therefore, perpendicular to the axis of rotation in the chair. As a result, both horizontal ducts are affected with rotational testing, with output from one duct stimulated whereas output from the other duct is inhibited. The mismatch between the resulting vestibular imbalance and visual and somatosensory information produces physiologic vertigo that generally lasts less than one to two minutes.

During this time, one can observe peripheral vestibular nystagmus and past-pointing. Because of the risk of falls and injury, patients should not stand until the vertigo has resolved. Vertigo can also be produced with caloric testing, e.g., by injecting cold or warm water into the patient's external auditory canal; however, this is generally much more uncomfortable for the patient than rotational testing and is also more time consuming and messier for the examiner. Therefore, caloric testing is not recommended for provocative testing to determine the category of dizziness.

In all patients with dizziness or vestibular complaints, careful examination of the eyes, ears, cardiovascular system, nervous system and vestibular system is indicated. Vestibular imbalance is indicated by nystagmus, past-pointing, and postural and gait abnormalities.

Because different types of nystagmus have different clinical implications, it is important to carefully characterize nystagmus both by its appearance and by any precipitating and inhibiting factors. For example, nystagmus may be characterized by the symmetry of the oscillations, whether the oscillations are linear or rotatory, and whether the oscillations are unidirectional or direction changing. Jerk nystagmus is identified by a clear slow phase drift in one direction and a corrective quick phase in the opposite direction. Jerk nystagmus is traditionally described by the direction of the quick phases, e.g., “down beat” nystagmus. In contrast, pendular nystagmus is characterized by smooth sinusoidal oscillations of the eyes.

Precipitating factors for nystagmus may include specific eye and head positions. Pathologic nystagmus may be present in primary position (spontaneous nystagmus), with a change in eye position (gaze-evoked nystagmus), or with a change in head position (positional and positioning nystagmus). Spontaneous nystagmus is assessed by direct observation of the patient’s eyes while the patient is looking straight ahead either fixating on a target or with fixation removed. Gaze-evoked nystagmus is assessed similarly with the patient fixating on targets 30 degrees to the right, left, up, and down. Extreme eye positions should be avoided because they can result in “end-point” nystagmus in normal individuals. Positioning nystagmus is assessed with the Dix-Hallpike positioning test.

An important inhibiting factor for peripheral vestibular nystagmus is fixation. While fixating, these patients can use their visual pursuit system to counteract the nystagmus. In contrast, fixation does not suppress central vestibular nystagmus because patients with central vestibular disorders cannot utilize their pursuit system to suppress the nystagmus. Because central vestibular and visual pursuit pathways are highly integrated, central vestibular lesions damage both systems, thereby precluding inhibition by fixation.

In order to visualize peripheral vestibular nystagmus, special techniques may be needed to suppress fixation. Ophthalmoscopy is a readily available way of preventing fixation when the nonviewed eye is covered. The direction of linear nystagmus when viewed with an ophthalmoscope is reversed from that observed by direct inspection of the eye, as (1) the axis of rotation of the eye is perpendicular to the line of sight; and (2) the retina lies behind the center of rotation to the eye whereas the cornea lies in front. Torsional nystagmus can also be detected with an ophthalmoscope by observing the vessels
EXPLORING THE PATHOPHYSIOLOGY OF VERTIGO

Physiologic imbalances in neural discharges within the vestibular system are produced with head movements, rotation and caloric stimulation. Pathological imbalances in the vestibular system can be produced by impairments either in the vestibular inputs or in the central connections of the vestibular system. Vertigo results from a mismatch between the converging inputs and the expected sensory patterns. For example, acute unilateral labyrinthine dysfunction produces vertigo because the sensation of self-motion associated with the vestibular tone imbalance is inconsistent with expectations based on visual and somatosensory information. The vertigo ultimately resolves, usually because of a rebalancing centrally rather than a return of function peripherally; i.e., central compensation corrects the mismatch between inputs and expectations.

The clinical manifestations of vestibular tone imbalance are produced through various vestibulo-ocular and vestibulo-spinal reflexes, as well as through the connections of the central vestibular system to cortical and brainstem centers. A disturbance of cortical spatial orientation produces the sensation of vertigo. Nystagmus is due to a direction-specific imbalance in the vestibulo-ocular reflexes. If eye movements do not match head movements, then images move across the retina, producing blurred vision and an illusory visual sensation of environmental motion called oscillopsia. Postural imbalance is caused by abnormal activation of monosynaptic and polysynaptic vestibulospinal pathways. Finally, nausea and vomiting are due to activation of the medullary-vomiting center.

around the macula. The direction of torsional nystagmus is not reversed when viewed with an ophthalmoscope because the axis of rotation is parallel to the line of sight.

Peripheral vestibular nystagmus is a mixed linear-rotatory jerk nystagmus that beats in one direction away from a hypo-functioning labyrinth. With semicircular duct stimulation or dysfunction, eye movements occur in the plane of an affected semicircular duct. In all forms of peripheral vestibular nystagmus, nystagmus amplitude and frequency increases with gaze in the direction of the quick phases due to summation of tonic driving forces and elastic restoring forces both moving the eyes in the direction of the nystagmus slow phases.

The presence of any of the following suggests a central cause for the nystagmus: (1) the nystagmus has a pendular appearance; (2) the nystagmus is purely rotatory or purely linear; (3) the nystagmus changes direction with gaze in different directions; (4) the nystagmus is not suppressed with fixation; (5) vertigo is mild or absent; (6) nausea is absent; and (7) there are central neurologic signs or symptoms. Despite these helpful rules, small cerebellar strokes may mimic labyrinthine lesions clinically. Therefore, particularly in the elderly, great care should be taken in excluding a central cause for acute vertigo, and central lesions should certainly be suspected if the clinical features are atypical.12-15

The Dix-Hallpike positioning test can precipitate vertigo in patients with episodic symptoms, especially when the symptoms appear to be related to either head position or head movements. The Dix-Hallpike positioning test allows detection of positionally-induced nystagmus, particularly that associated with benign paroxysmal positional vertigo.11

The patient is instructed to stare off into space and avoid looking at any specific object during the procedure. The patient's head is turned to one side, and then the patient is rapidly moved backward from a sitting to a head hanging position. Turning the head to one side during the maneuver places the ipsilateral posterior semicircular duct in a parasagittal plane; when the patient is subsequently moved backward, the movement is in the plane of that duct. The examiner maintains the patient in the head-hanging position for approximately one minute and observes the patient's eyes for nystagmus. Anticipation or the experience of vertigo may make patients anxious. Calm but firm reassurance from the examiner is often necessary to complete the maneuver.

Like peripheral spontaneous nystagmus, peripheral positioning nystagmus is a mixed linear-rotatory jerk nystagmus. Peripheral positional vestibular nystagmus generally beats upward and toward the under-most ear and has a latency of from one to 45 seconds and a duration of less than 60 seconds. It lessens or disappears with repetition of the offending head positioning. The presence of any of the following suggests a
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Central cause for the nystagmus: (1) the nystagmus begins immediately on assuming the offending head position; (2) the nystagmus has a pendular appearance; (3) the nystagmus is purely rotatory or purely linear; (4) the nystagmus changes direction with gaze in different directions; (5) the nystagmus is not suppressed with fixation; (6) the nystagmus continues indefinitely with maintenance of the offending head position; (7) the nystagmus persists with repetition of the offending head positioning; (8) vertigo is mild or absent; (9) nausea is absent; or (10) there are central neurologic signs or symptoms. As with spontaneous vestibular nystagmus, great care should be taken in excluding a central cause for acute vertigo, particularly in the elderly.

Corrective saccades after single rapid head turns can be helpful in identifying the side of vestibular dysfunction, particularly when spontaneous nystagmus is absent. In this test, sometimes referred to as the head-thrust test, the patient's head is turned rapidly to one side by the examiner, while the patient attempts to maintain fixation on an object six feet or more away. The examiner observes the patient for corrective saccades. The gaze of a patient with unilateral labyrinthine dysfunction shifts only when the head moves quickly toward the dysfunctional side; an oppositely directed compensatory saccade corrects the gaze error. Thus, leftward saccades following rapid rightward head movements indicate right vestibular dysfunction, whereas rightward saccades following rapid leftward head movements indicate left vestibular dysfunction. As mentioned earlier, caloric testing may also be helpful in determining the side of a peripheral vestibular lesion but is more time consuming and uncomfortable for patients.

Clinical disturbances of the vestibulospinal pathways are assessed with several tests, including past-pointing, stance, the Romberg test and tandem gait with eyes closed.

When assessing a patient for past-pointing, the patient is asked to sit facing the examiner with index finger extended and pointing at, but not touching, the examiner's extended finger. The patient is then asked to raise the arm to a vertical position with the index finger pointing at the ceiling, and subsequently return the arm to the initial position. This is repeated several times with the eyes closed. Consistent deviation of the arm to one side is past-pointing. If extralabyrinthine inputs are not minimized by keeping the eyes closed and the arm extended, visual or proprioceptive signals will permit accurate localization of the target even if vestibular function is impaired; for this reason, the standard finger-nose-finger test is not helpful in identifying past-pointing. In acute vestibular lesions, patients past-point toward the affected side; however, the test can be misleading because central nervous system compensation rapidly corrects the past-pointing and can produce a drift to the opposite side.

With acute unilateral vestibular lesions, patients have impaired postural control and may sway or fall toward the lesion. Although this is helpful diagnostically, the examiner must take great care when assessing stance and gait in patients with vestibular complaints because patients may suddenly fall and injure themselves. The examiner must provide adequate support for the patient to prevent falls and injuries. In patients who are unable to maintain their stance without support when their eyes are open, it is unnecessary and potentially dangerous to proceed with the Romberg test or an unsupported assessment of gait.

In patients with vestibular lesions, the tendency to fall toward the lesion is accentuated when patients are prevented from using vision to compensate for the vestibular imbalance; this is the basis of the Romberg test. In the Romberg test, the patient is first asked to stand with eyes open and feet together. If the patient is unable to maintain balance in this position, the stance is widened until this is possible. The patient is then asked to close his or her eyes. Patients with proprioceptive or vestibular dysfunction may be unable to maintain this position. Patients with unilateral dysfunction usually sway or fall toward the side of the lesion, particularly if the dysfunction is acute. Because of central nervous system compensation, the test is less sensitive to chronic unilateral vestibular dysfunction. As with past-pointing, overcompensation may result in falls toward the “good” side.

With eyes open, tandem walking may be impaired with acute vestibular lesions. It is, however, mainly a test of cerebellar function, because vision compensates for chronic vestibular and proprioceptive dysfunction. A better test of vestibular function is tandem walking with eyes closed. When cerebellar and proprioceptive function is normal, imbalance during this
test indicates vestibular dysfunction. However, the direction of falling does not reliably indicate the side of the lesion. The test is also difficult for normal elderly persons.

In some cases, additional diagnostic tests will be required. These should be ordered selectively, depending on the type of dizziness and suspected underlying etiologies. Diagnostic studies that may be helpful in selected patients with vertigo (not attributable to benign paroxysmal positional vertigo) include audiometry, electronystagmography, bithermal caloric testing, brainstem auditory evoked potentials, and cranial imaging. In particular, a high percentage of patients with normal examinations and nonspecific vertigo suffer from peripheral vestibular dysfunction that can be documented with electronystagmography and rotatory chair testing. Cranial imaging is essential in patients with a first attack of acute persistent vertigo, especially in association with central signs or age over 50 years, in order to exclude cerebellar infarction.

**MANAGEMENT**

Drugs should be reviewed in all patients with dizziness, whether clearly vertiginous or not. Drugs associated with dizziness include alcohol and other central nervous system depressant medications (e.g., benzodiazepines, barbiturates, phenothiazines), aminoglycoside antibiotics, anticonvulsants, antidepressant medications, antihypertensive medications, chemotherapeutic agents, loop diuretics (e.g., furosemide), and salicylates. The elderly are particularly susceptible to drug ototoxicity because (1) they are more likely to receive ototoxic drugs; (2) they have less reserve (due to age-associated vestibular end organ changes, pre-existing sensorineural hearing loss, and previous treatment with ototoxic drugs); (3) they are more likely to have impaired renal function.

Alcohol can produce a positional vertigo syndrome (i.e., positional alcohol nystagmus) because the alcohol can diffuse into the labyrinth and change the specific gravity of the rotation-transducing organs (i.e., the cupulae of the semicircular ducts). Aminoglycosides can produce irreversible damage to labyrinthine hair cells, with resulting sensory disequilibrium or (if somewhat asymmetric) vertigo, along with oscillopsia, hearing loss, and tinnitus. Similar clinical pictures can occur with antimalarial agents, cisplatinum, ethacrynic acid, furosemide, minocycline, and salicylates. The dysfunction is usually reversible with furosemide, minocycline, and salicylates.

The treatment of dizziness varies by type and cause. In general, it is symptomatic and directed at the underlying cause. Vertigo, especially acute persistent vertigo, may be alleviated with vestibular sedatives. Some types of vertigo may also be amenable to specific curative therapies, most notably with canalith repositioning maneuvers for benign paroxysmal positional vertigo. Vertigo associated with migraine may respond to abortive and prophylactic antimigraine treatments. Other forms of episodic vertigo (such as Meniere syndrome) may benefit from pharmacological or surgical therapies.

In patients with acute persistent vertigo from peripheral vestibular lesions, recovery occurs more rapidly and more completely when vestibular exercises are begun as soon as possible after the onset of symptoms. A number of variations of vestibular exercises have been advocated, some more complex than others. Typically, eye and head movements are begun as

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<th>TABLE 1. DIFFERENTIAL DIAGNOSIS OF VERTIGO</th>
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<td><strong>Vertigo May be Confused With</strong></td>
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<td>• Dysequilibrium (e.g., due to impaired peripheral sensation)</td>
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<td>• Presyncope (e.g., impending faint, hypoglycemia)</td>
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<td>• Psychophysiologic (psychogenic) dizziness</td>
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<td>• Seizures</td>
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**Frequent Causes of Vertigo**

*In the elderly:*
- Benign paroxysmal positional vertigo
- Labyrinthine ischemia
- Toxic vestibulopathy
- Traumatic vestibulopathy
- Vertebrobasilar ischemia
- Viral labyrinthitis

*In younger adults:*
- Meniere syndrome
- Viral labyrinthitis
- Traumatic vestibulopathy
- Toxic vestibulopathy

*In children:*
- Benign paroxysmal vertigo
- Migraine
- Otitis media
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soon as possible after the acute vertigo and autonomic symptoms subside; then, more complicated exercises involving head movements, bending, standing, and moving about are gradually introduced as the patient improves. Patients can begin while still in bed by repetitively moving the eyes up and down and then side-to-side at first slowly, then quickly. Then patients repetitively move their head forward and backward and side-to-side at first slowly, then quickly.

Progression is next to performing exercises while sitting: eye and head movements as described above, then rotating head and shoulders at first slowly, then quickly, and then bending forward and picking up objects from the floor. Next, patients practice changing from sitting to standing, at first with eyes open and then with eyes closed. While standing, eye and head movements are practiced. Patients then practice moving about: turning around; walking across a room at first with eyes open and then closed; standing on one foot, at first with eyes open and then closed; climbing up and down steps with eyes open; and playing games involving stooping, stretching, and aiming (e.g., shuffleboard or bowling).

The exercises should be performed for at least five minutes several times per day. Specific head positions and movements that precipitate vertigo should be sought and repetitively performed to facilitate vestibular compensation. Antivertiginous medications can be used during the exercises to help control both the vertigo and the autonomic symptoms. Although there are theoretical reasons to imagine that vestibular sedatives may limit the efficacy of vestibular exercises, there is no solid evidence that such medications affect either the rate or degree of vestibular compensation. Instrumental rehabilitation training on a moving platform can also be effective for treating vestibular balance disorders and can result in improved control of balance and greater independence in activities of daily living, although such treatment often is not readily available.

In a given patient, it is often difficult to predict what drug or combination of drugs will be most effective in alleviating the symptoms of acute persistent vertigo. The drug or drug combination is empirically chosen based on the known effects of each drug and on the course and severity of the patient’s symptoms. Severe forms of acute persistent vertigo are especially distressing, particularly when accompanied by nausea and vomiting; antivertiginous medications with both sedative and antiemetic effects are helpful in these situations (e.g., promethazine, droperidol, dimenhydrinate). Chronic recurrent vertigo is less distressing and interferes less with daily activities; agents with less sedating properties will help patients carry on with their normal routine (e.g., meclizine, trimethobenzamide). Agents that commonly cause confusion because of central nervous system depressant or anticholinergic properties should be utilized carefully or avoided in the elderly (e.g., diazepam, phenobarbital, dimenhydrinate, meclizine). Also, parenterally-administered drugs that may produce hypotension or respiratory depression (e.g., diazepam, droperidol, phenobarbital, prochlorperazine, or trimethobenzamide administered either intramuscularly or intravenously as appropriate), should generally be used only in a hospital setting.

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15. Smouha EE, Roussos C. Atypical forms of paroxysmal positional nystagmus. Ear Nose Throat J 1995;74:649-

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