CHAPTER 19

Dizziness and Vertigo

Andrew K. Chang and Jonathan S. Olshaker

PERSPECTIVE

Dizziness is an extremely common yet complex neurologic symptom that reflects a disturbance of normal balance perception and spatial orientation. An estimated 7.5 million patients with dizziness are seen each year in ambulatory care settings.[1] Dizziness is also one of the most common principal complaints in the emergency department (ED) and is responsible for 2.5% of all ED visits.[2] Among patients older than 60 years, 20% have experienced dizziness severe enough to affect their daily activity.[3]

"Dizziness" is an imprecise descriptor. Patients use the term to describe a variety of experiences, including sensations of motion, weakness, lightheadedness, unsteadiness, and depression. Even clinical experts do not uniformly agree to precise definitions, with some defining it broadly and others more narrowly. Dizziness is historically categorized into one of four categories based on symptom quality: vertigo (illusion of motion, often spinning), near syncope (feeling of impending faint), disequilibrium (loss of equilibrium when walking), and nonspecific dizziness.[4]

Dizziness can be caused by a myriad diseases. In older persons it is associated with a variety of cardiovascular, neurosensory, and psychiatric conditions and with the use of multiple medications.[5] The challenge for the emergency physician is to sift out the rare patient with a dangerous underlying disorder from the many others who have benign causes.

Pathophysiology

The maintenance of equilibrium and awareness of the body in relationship to its surroundings depend on the interaction of the visual, proprioceptive, and vestibular systems. The eyes, muscles, joints, and otic labyrinths continuously supply information to the brain about the position of the body. Visual impulses, mediated through the higher brain centers, provide information about body position in space. Impulses from proprioceptors of the joints and muscles supply data about the relative positions of the parts of the body. Impulses from the neck are of special importance in relating the position of the head to the rest of the body. The sense organs of the visual, vestibular, and proprioceptive systems are connected to the cerebellum by way of the vestibular nuclei in the brainstem. Any disease that interrupts the integration of these three systems may give rise to symptoms of vertigo.

The vestibular apparatus helps maintain head position and stabilize head movement. It is housed in the inner ear, or labyrinth, which lies embedded in the petrous portion of the temporal bone. Here it is vulnerable to trauma, blood-borne toxins, and infections in nearby structures, including the middle ear and meninges. The vestibular apparatus consists of three semicircular canals and two otolithic structures (the utricle and the saccule). The semicircular canals and the utricle are connected to each other and contain endolymph. The semicircular canals provide information about movement and angular momentum, whereas the utricle (via otoliths, which are calcium carbonate particles) provides information about the orientation of the body with respect to gravity.

The semicircular canals are paired (left and right ears) structures that normally respond to motion in a symmetrical manner. With inner ear disease, the resting discharge or the discharge stimulated by motion can be altered in one ear. This alteration produces asymmetrical responses and results in the perception of vertigo. For example, freely moving otoliths that are inappropriately located within the semicircular canals, as in benign paroxysmal positional vertigo (BPPV), can produce positional vertigo as the otoliths move under the influence of gravity.

Impulses leave the vestibular apparatus by the vestibular part of the acoustic nerve (cranial nerve VIII), enter the brainstem just below the pons and anterior to the cerebellum, and proceed to the four vestibular nuclei of the brainstem and to the cerebellum. From there, impulses travel along two pathways that contribute to the clinical manifestations of vertigo: the medial longitudinal fasciculus (MLF) and the vestibulospinal tract. In individuals with healthy vestibular systems, these connections allow the eyes to compensate for body movement in different directions and to maintain a visual axis that is stable with respect to the environment.

Nystagmus occurs when the synchronized vestibular information becomes unbalanced. Typically, it results from unilateral vestibular disease, which causes asymmetrical stimulation of the medial and lateral rectus muscles of the eye. This unopposed activity causes a slow movement of the eyes toward the side of the stimulus, regardless of the direction of deviation of the eyes. The cerebral cortex then corrects for these eye movements and rapidly brings the eyes back to the midline, only to have the process repeated.

By convention, the direction of nystagmus is denoted by the direction of the fast "cortical" component. Nystagmus caused by vestibular disease tends to be unidirectional and horizontorotary. If the nystagmus is vertical, a central lesion (either brainstem or cerebral) is usually the cause.

The vestibular nuclei send information to the lateral vestibulospinal tract, where they connect with motor neurons that supply the muscles of the extremities. This explains the phenomenon of false steps or other body movements made by people with a defective vestibular apparatus who are attempting to correct for an imagined change in position. Connections between the vestibular nuclei and the autonomic system account for the perspiration, nausea, and vomiting that commonly accompany an attack of

vertigo. Connections between the vestibular nuclei and the cerebellum account for the modulating influence of this organ on motor activity.

DIAGNOSTIC APPROACH

Differential Considerations

It is often helpful to have dizzy patients describe the sensation they are experiencing without using the word *dizzy*. When this is done, one can generally categorize the dizziness into one of four categories: vertigo, near syncope, disequilibrium, and nonspecific dizziness. Vertigo is an illusion of motion, classically described as the room spinning. Some further subdivide this into objective vertigo (external environment is spinning) and subjective vertigo (spinning of self). Near syncope is usually described as feeling faint or lightheaded. Disequilibrium is usually described as an unsteady gait. Nonspecific dizziness is generally thought to be related to anxiety. The validity of this symptom-oriented method of categorizing dizziness has been recently challenged.[6] For some patients, dizziness is simply a metaphor for malaise, representing a variety of causes, such as anemia, viral illness, or depression. The primary focus of this chapter is to provide a framework to differentiate vertigo from other types of dizziness and to identify potentially life-threatening causes of these symptoms.

If the patient has true vertigo, the clinician should determine whether the cause is a peripheral lesion, such as from the vestibular system, or a central process, such as cerebrovascular disease or a neoplasm. In most cases, peripheral disorders are benign, whereas central disorders have more serious consequences. Occasionally, as in the case of a cerebellar hemorrhage, immediate therapeutic intervention is indicated. Acute suppurative labyrinthitis is the only cause of peripheral vertigo that requires urgent intervention. Box 19-1 lists causes of vertigo and identifies the peripheral, central, and systemic diagnoses. Table 19-1 summarizes the different characteristics of peripheral and central vertigo.

Box 19-1 Causes of Vertigo

Peripheral Causes
Benign paroxysmal positional vertigo (BPPV)
Vestibular neuritis (or neuronitis)
Labyrinthitis (suppurative, serous, toxic, chronic)
Ménière's disease
Foreign body in ear canal
Acute otitis media
Perilymphatic fistula
Trauma (labyrinth concussion)
Motion sickness
Acoustic neuroma*

Central Causes
Infection (encephalitis, meningitis, brain abscess)
Vertebral basilar artery insufficiency
Subclavian steal syndrome
Cerebellar hemorrhage or infarction
Vertebral basilar migraine
Post-traumatic injury (temporal bone fracture)
Postconcussive syndrome
Temporal lobe epilepsy
Tumor
Multiple sclerosis

Systemic Causes
Diabetes mellitus
Hypothyroidism

*Cause of peripheral vertigo that proceeds centrally.

Pivotal Findings

History

The medical history is used to determine if true vertigo exists. Although usually described as the environment spinning, any sensation of disorientation in space or sensation of motion can qualify as vertigo. Some nausea, vomiting, pallor, and perspiration accompany almost all but the mildest forms of vertigo. A sensation of imbalance often accompanies vertigo, and this can be extremely difficult to distinguish from true instability until after the patient's symptoms have been reduced by treatment. True instability, disequilibrium, or ataxia indicates a higher likelihood of a central process.[7] Because the labyrinth has no effect on the level of consciousness, the patient should not have an associated change in mentation or syncope.

The *time of onset* and the *duration of vertigo* are important clues to the cause. Episodic vertigo that is severe, lasts up to several hours, and has symptom-free intervals between episodes suggests a peripheral labyrinth disorder. Vertigo produced primarily by a change in position also suggests a peripheral disorder, usually BPPV.

The presence of *auditory symptoms* suggests a peripheral cause of the vertigo, as in middle and inner ear problems. The ear with abnormal hearing is usually on the side of end-organ disturbance. Although vertigo can occur with acoustic neuroma, it is usually a progressive unilateral hearing loss, typically of several months' duration, that prompts the patient with acoustic neuroma to seek medical attention. Hearing loss, vertigo, and tinnitus form the characteristic triad of Ménière's disease.

Head injury can cause vertigo occasionally from intracerebral injury and more commonly from labyrinth concussion. Neck injury can cause vertigo from vertebral artery dissection, resulting in posterior circulation ischemia.[8]

Vestibular neuritis and labyrinthitis are very similar, though in labyrinthitis hearing is affected. Patients often develop continuous vertigo that can be worsened by movement and may take several months to fully resolve.

Are there associated neurologic symptoms? The patient or family members should be questioned about the time of onset of ataxia or gait disturbances. Ataxia of recent and relatively sudden onset suggests cerebellar hemorrhage or infarction in the distribution of the posterior inferior cerebellar artery or the superior cerebellar artery. Ataxia that is slowly progressive suggests chronic cerebellar

Table 19-1 Characteristics of Peripheral and Central Vertigo

CHARACTERISTIC	PERIPHERAL	CENTRAL
Onset	Sudden	Gradual or sudden
Intensity	Severe	Mild
Duration	Usually seconds or minutes; occasionally hours, days (intermittent)	Usually weeks, months (continuous) but can be seconds or minutes with vascular causes
Direction of nystagmus	One direction (usually horizontorotary)	Vertical, downbeating
Effect of head position	Worsened by position, often single critical position	Little change, associated with more than one position
Associated neurologic findings	None	Usually present
Associated auditory findings	May be present, including tinnitus	None

disorders. True ataxia may be difficult to discern from the unsteadiness that occurs when a patient with significant vertigo attempts to walk, though other findings such as nystagmus and dysmetria can often help narrow the differential diagnosis. The symptom of imbalance raises the likelihood of TIA and stroke. Isolated vertigo can be the only initial symptom of cerebellar and other posterior circulation bleeds, transient ischemic attacks (TIAs), and infarction.[9,10] One study showed that emergency physicians often did not make the correct diagnosis in patients with validated strokes or TIAs whose presenting symptom was only vertigo.[7] Identifying these individuals is a significant and important challenge for clinicians taking care of vertiginous patients. The vast majority of patients with isolated dizziness do not have TIA or stroke, but the risk of not identifying the few who do is significant for ultimate patient outcomes. Stroke has been seen in 3.2% of patients with dizziness syndrome, but only 0.7% of those with isolated dizziness had a stroke.[7] A recent study also showed that fewer than 1 in 500 patients discharged with a diagnosis of dizziness or vertigo experienced a major vascular event in the month after discharge.[11]

Past Medical History. Older age, male sex, hypertension, coronary heart disease, diabetes, and atrial fibrillation are examples of diseases that put patients at higher risk for TIA and stroke. It is important to identify what medications patients are taking, because many have direct vestibulotoxicity. The most commonly encountered are the aminoglycosides, anticonvulsants, alcohols, quinine, quinidine, and minocycline. Daily consumed substances such as caffeine and nicotine, which are not often thought of as medications, can have wide-ranging autonomic effects that may exacerbate vestibular symptoms.

Physical Examination

Vital Signs. Presence of hypotension suggests near syncope as the cause of dizziness. When subclavian steal syndrome, which also can cause vertebrobasilar insufficiency (VBI), is suspected, the pulse and blood pressure should be checked on both sides.

Head and Neck. Carotid or vertebral artery bruits suggest atherosclerosis and risk for TIA or stroke. The vertebral artery can be auscultated in the supraclavicular region.

Accumulation of fluid in the middle ear as a result of a middle ear infection may cause mild vertigo, as can occlusion of the eustachian tubes associated with an upper respiratory tract infection or descent barotrauma. A perforated or scarred eardrum may indicate a perilymphatic fistula, especially if the history includes previous trauma.

Examination of the eyes is key in assessing a patient with vertigo. Pupillary abnormalities may indicate third cranial nerve or descending sympathetic tract involvement. Papilledema suggests increased intracranial pressure. Extraocular movements should be assessed carefully because relatively subtle ocular movement abnormalities can be the only clue to a cerebellar hemorrhage. A sixth cranial nerve palsy ipsilateral to the hemorrhage may result from early brainstem compression by the expanding hematoma. Internuclear ophthalmoplegia, which indicates brainstem pathology, is recognized when the eyes are in a normal position on straight-ahead gaze, but on eye movement the adducting eye (cranial nerve III) is weak or shows no movement while the abducting eye (cranial nerve VI) moves normally, although often displaying a coarse nystagmus. This finding indicates an interruption of the MLF on the side of the third cranial nerve weakness and is virtually pathognomonic of multiple sclerosis.

Abnormal nystagmus is the cardinal sign of inner ear disease and the principal objective evidence of abnormal vestibular function. In nystagmus, the patient has difficulty maintaining the conjugate deviation of the eyes or has a postural control imbalance of eye movements.

The abnormal jerk nystagmus of inner ear disease consists of slow and quick components. The eyes slowly "drift" in the direction of the diseased, hypoactive ear, then quickly jerk back to the intended direction of gaze. Positional nystagmus, induced by changing the position of the head, strongly suggests an organic vestibular disorder, typically BPPV. Central nervous system causes of nystagmus are considered when the pattern of nystagmus is purely vertical, downbeating (fast phase beating toward the nose, or nonfatigable) or when the nystagmus is neither provoked nor relieved by repositioning maneuvers. The characteristics of nystagmus are one of the most valuable tools for distinguishing peripheral from central causes of vertigo (Table 19-2).

Table 19-2 Distinguishing Characteristics of Nystagmus with Central and Peripheral Vertigo

CHARACTERISTIC	CENTRAL	PERIPHERAL
Direction	Can be any direction, downbeating (fast phase beats toward nose)	Horizontal or horizontorotary, upbeating (fast phase beats toward forehead)
Position testing effects		
Latency	Short	Long
Duration	Sustained	Transient
Intensity	Mild	Mild to severe
Fatigability	Nonfatigable	Fatigable
Effect of visual fixation	Not suppressed, may be enhanced	Suppressed

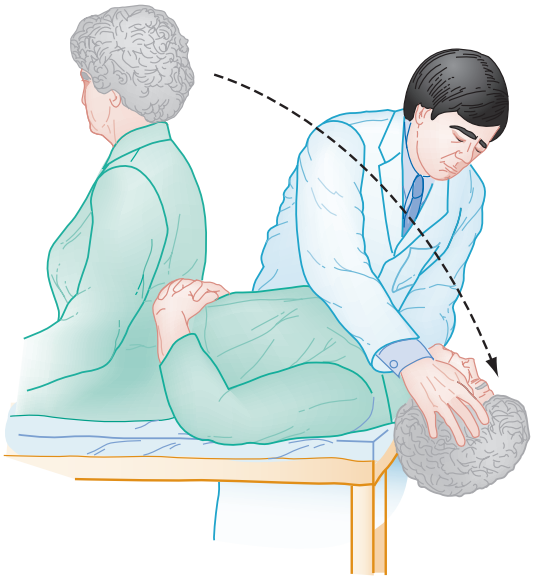

Figure 19-1. Testing for positional vertigo and nystagmus.

Positional Testing. Positional testing can confirm the diagnosis of some causes of peripheral vertigo. The Hallpike test,[12] also known as the Dix-Hallpike test or the Nylen-Barany test, confirms the diagnosis of posterior canal BPPV. The head is turned 45 degrees to one side and then the patient is moved from an upright seated position to a supine position with the head overhanging the edge of the gurney (Fig. 19-1). The eyes are observed for nystagmus, and the patient is queried for the occurrence of vertigo. The patient is then brought back up to the seated position and the test is repeated with the head turned 45 degrees to the other side. In general, if the patient has posterior canal BPPV, only one side should be positive, and this indicates the involved side. Once the diagnosis has been confirmed, the patient can be treated with the Epley maneuver (see later). If the Hallpike test result is negative or seems to be positive bilaterally, one can use the roll test to test for the horizontal canal variant of BPPV. Unlike in the Hallpike

test, the head does not need to overhang the edge of the gurney, and the patient will have reproduction of symptoms and horizontal nystagmus with the head turned in either direction. In the roll test the patient lies flat on the gurney (unlike the Hallpike test, the head does not need to overhang the edge of the gurney) and the examiner turns the patient's head up to 90 degrees to one side. If the otoliths are located in the horizontal canal, the patient will develop vertigo and horizontal nystagmus. The examiner then turns the head to the other side. Again, if otoliths are located in the horizontal canal, the patient will develop vertigo and horizontal nystagmus (note that the direction of the nystagmus will change). Thus, unlike the patient with posterior canal BPPV, the patient with horizontal canal BPPV will have reproduction of symptoms and horizontal nystagmus with the head turned in either direction. The side that is involved is the one with the more violent symptoms and more dramatic nystagmus. The horizontal canal variant of BPPV can be treated with the "barbecue roll" (see later).

The head thrust, or head impulse test, is used to diagnose vestibular neuritis and labyrinthitis.[13] The physician stands face to face with the patient and places both hands on the sides of the patient's head. The patient stares at the examiner's nose while the examiner rapidly turns the patient's head approximately 10 degrees to one side. Normally the patient's eyes should keep focusing on the examiner's nose. If there is a problem with the vestibular nerve, the eyes will temporarily move along with the head. A corrective saccade will then occur, in which the eyes jerk back toward the midline. If a saccade is seen, this denotes a positive head-thrust test result and indicates vestibular nerve dysfunction and confirms the diagnosis of vestibular neuritis.

Neurologic Examination. The presence of cranial nerve deficits suggests a space-occupying lesion in the brainstem or cerebellopontine angle. The corneal reflex is a sensory cranial nerve V and motor cranial nerve VII circuit. Its diminution or absence can be one of the early signs of an acoustic neuroma, which is a slow-growing tumor of the vestibular-cochlear nerve (the eighth cranial nerve). Acoustic neuromas rarely manifest with vertigo and instead typically manifest with sensorineural hearing loss. The Rinne and Weber tests are used in patients with hearing loss. In the Rinne test, which tests for conductive hearing loss, a vibrating tuning fork is held against the mastoid bone. Once the patient can no longer hear the sound from the tuning fork, the tuning fork is moved toward the ear. If there is no conductive hearing loss, the patient will be able to hear the tuning fork. With acoustic neuroma, the result of the Rinne test is normal (the patient can still hear the tuning fork), though both air and bone conduction are reduced as compared with someone who does not have an acoustic neuroma. In the Weber test, which tests for sensorineural hearing loss, a vibrating tuning fork is held against the middle of the forehead. If there is no sensorineural hearing loss, the sound is heard equally in both ears. In patients with acoustic neuroma, the sound will localize to the normal ear. Seventh cranial nerve involvement (e.g., Bell's palsy) causes facial palsy that affects the entire side of the face. In supranuclear facial paralysis, the forehead is spared because these muscles receive bilateral cortical innervation.

The patient is evaluated specifically for evidence of cerebellar dysfunction. This examination is performed when the patient is both sitting and standing because truncal ataxia may become obvious only when the patient has to sit, stand, or walk unaided. Dysmetria is the inability to arrest a muscular movement at the desired point and should be assessed with finger-to-finger or finger-to-nose pointing. Dysdiadochokinesia (an inability to perform coordinated muscular movement smoothly) is assessed with rapid alternating movements. Gait is evaluated when the patient gives a history suggesting ataxia, although examination may be impossible during an attack of vertigo. Any marked abnormality (e.g., consistent falling or a grossly abnormal gait) should suggest a central lesion, especially in a patient whose vertiginous symptoms have subsided. The main features of a cerebellar gait are a wide base (separation of legs), unsteadiness, irregularity of steps, tremor of the trunk, and lurching from side to side. The unsteadiness is most prominent on arising quickly from a sitting position, turning quickly, or stopping suddenly while walking. Patients with gait ataxia cannot perform heel-to-toe walking.

Ancillary Testing

Most routine laboratory testing is not helpful in the evaluation of a vertiginous patient except for a finger-stick blood glucose test.[14] Blood counts and blood chemistries are sometimes helpful when it is difficult to distinguish whether "dizziness" is vertigo or near syncope. An electrocardiogram should be obtained if there is a possibility of myocardial ischemia or dysrhythmia.

Radiologic Imaging. A cross-sectional study of the National Hospital Ambulatory Medical Care Survey (NHAMCS) suggests significant overuse of computed tomography (CT) in low-risk patients.[15] Risk factor assessment and symptom patterns can be extremely helpful in deciding which patients warrant imaging and admission. Older age, male sex, hypertension, coronary artery disease, diabetes, and atrial fibrillation put patients at higher risk for more serious causes of dizziness and vertigo.

If cerebellar hemorrhage, cerebellar infarction, or other central lesions are suggested, emergent CT or magnetic resonance imaging (MRI) of the brain is indicated. MRI, when available, has become the diagnostic modality of choice when cerebellar processes other than acute hemorrhage are possible. MRI is particularly useful for the diagnosis of acoustic neuromas and for sclerotic and demyelinating lesions of the white matter, as seen in multiple sclerosis. Acute vertigo by itself does not warrant urgent CT or MRI in all patients, particularly patients in whom a clear picture of peripheral vertigo emerges. But as mentioned earlier, many studies strongly support the use of imaging in patients of advanced age or at risk for cerebrovascular disease.[7,9,16] CT, although often useful for identifying hemorrhage, is insensitive for acute stroke presentations, especially for infarction within the posterior fossa. MRI with magnetic resonance angiography (MRA) is considered a much more sensitive modality and should be performed quickly in patients with changing neurologic signs and symptoms, suggesting impending posterior circulation occlusion.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for other peripheral, central, and systemic causes of vertigo is large (see Box 19-1). More detailed information is given on selected causes in Table 19-3, including the most common peripheral causes of true vertigo: BPPV, vestibular neuritis, labyrinthitis, and Ménière's disease.

Diagnostic Algorithm

Most cases of vertigo are of peripheral origin and are not usually life-threatening. The diagnostic approach focuses on identifying entities that either immediately or in the near future can lead to death or significant morbidity (Fig. 19-2).

MANAGEMENT

Management is based on an accurate diagnosis that distinguishes the serious central causes of vertigo from the less serious, albeit more debilitating, peripheral causes (Fig. 19-3). Any suggestion of cerebellar hemorrhage warrants immediate imaging with CT or MRI and neurosurgery consultations. VBI should be considered in any patient of advanced age or at high risk of cerebrovascular disease with isolated, new-onset vertigo without an obvious cause.[17,18] Because of the possibility of progression of new-onset VBI in the first 24 to 72 hours, hospital or observation unit

Table 19-3 Differential Diagnosis of Patients with True Vertigo

CAUSE	HISTORY	ASSOCIATED SYMPTOMS	PHYSICAL
Peripheral			
1. Benign paroxysmal positional vertigo	Short-lived, positional, fatigable episodes.	Nausea, vomiting.	Single position can precipitate vertigo. Positive result on Hallpike test (posterior semicircular canal) or Roll test (horizontal canal).
2. Labyrinthitis			
A. Serous	Mild to severe positional symptoms. Usually coexisting or antecedent infection of ear, nose, throat, or meninges.	Mild to severe hearing loss can occur.	Usually nontoxic patient with minimal fever elevation.
B. Acute suppurative	Coexisting acute exudative infection of the inner ear. Severe symptoms.	Usually severe hearing loss, nausea, vomiting.	Febrile patient showing signs of toxicity. Acute otitis media.
C. Toxic	Gradually progressive symptoms: Patients on medication causing toxicity.	Hearing loss that may become rapid and severe, nausea and vomiting.	Hearing loss. Ataxia common feature in chronic phase.
3. Ménière's disease	Recurrent episodes of severe rotational vertigo usually lasting hours. Onset usually abrupt. Attacks may occur in clusters. Long symptom-free remissions.	Nausea, vomiting, tinnitus, hearing loss.	Positional nystagmus not present.
4. Vestibular neuritis	Sudden onset of severe vertigo, increasing in intensity for hours, then gradually subsiding over several days but can last weeks to months. Can be worsened with positional change. Sometimes history of infection or toxic exposure that precedes initial attack. Highest incidence is found in third and fifth decades.	Nausea, vomiting. Auditory symptoms do not occur.	Spontaneous nystagmus toward the involved ear may be present.
5. Acoustic neuroma	Gradual onset and increase in symptoms. Neurologic signs in later stages. Most occur in women aged 30 to 60.	Hearing loss, tinnitus. True ataxia and neurologic signs as tumor enlarges.	Unilateral decreased hearing. True truncal ataxia and other neurologic signs when tumor enlarges. May have diminution or absence of corneal reflex. Eighth cranial nerve deficit may be present.
Central			
1. Vascular disorders			
A. Vertebrobasilar insufficiency	Should be considered in any patient of advanced age with isolated new-onset vertigo without an obvious cause. More likely with history of atherosclerosis. Can occur with neck trauma. Initial episode usually lasts seconds to minutes.	Often headache. Usually neurologic symptoms including dysarthria, ataxia, weakness, numbness, double vision. Tinnitus and deafness uncommon.	Neurologic deficits usually present, but initially neurologic examination can be normal.
B. Cerebellar hemorrhage	Sudden onset of severe symptoms.	Headache, vomiting, ataxia.	Signs of toxicity. Dysmetria, true ataxia. Ipsilateral sixth cranial nerve palsy may be present.
C. Occlusion of posterior inferior cerebellar artery (Wallenberg's syndrome)	Vertigo associated with significant neurologic complaints.	Nausea, vomiting, loss of pain and temperature sensation, ataxia, hoarseness.	Loss of pain and temperature sensation on the side of the face ipsilateral to the lesion and on the opposite side of the body, paralysis of the palate, pharynx, and larynx. Horner's syndrome (ipsilateral ptosis, miosis, and decreased facial sweating).
2. Head trauma	Symptoms begin with or shortly after head trauma. Positional symptoms most common type after trauma. Self-limited symptoms that can persist weeks to months.	Usually mild nausea.	Occasionally, basilar skull fracture.

Table 19-3 Differential Diagnosis of Patients with True Vertigo—cont'd

CAUSE	HISTORY	ASSOCIATED SYMPTOMS	PHYSICAL
3. Vertebrobasilar migraine	Vertigo almost always followed by headache. Patient has usually had similar episodes in past. Most patients have a family history of migraine. Syndrome usually begins in adolescence.	Dysarthria, ataxia, visual disturbances, or paresthesias usually precede headache.	No residual neurologic or otologic signs are present after attack.
4. Multiple sclerosis	Vertigo presenting symptom in 7-10% and appears in the course of the disease in a third. Onset may be severe and suggest labyrinth disease. Disease onset usually between ages 20 and 40. Often history of other attacks with varying neurologic signs or symptoms.	Nausea and vomiting, which may be severe.	May have horizontal, rotary, or vertical nystagmus. Nystagmus may persist after the vertiginous symptoms have subsided. Bilateral internuclear ophthalmoplegia and ataxic eye movements suggest multiple sclerosis.
5. Temporal lobe epilepsy	Can be initial or prominent symptom in some patients with the disorder.	Memory impairment, hallucinations, trancelike states, seizures.	May have aphasia or convulsions.
6. Hypoglycemia	Should be considered in diabetics and any other patient with unexplained symptoms.	Sweating, anxiety.	Tachycardia, mental status change may be present.

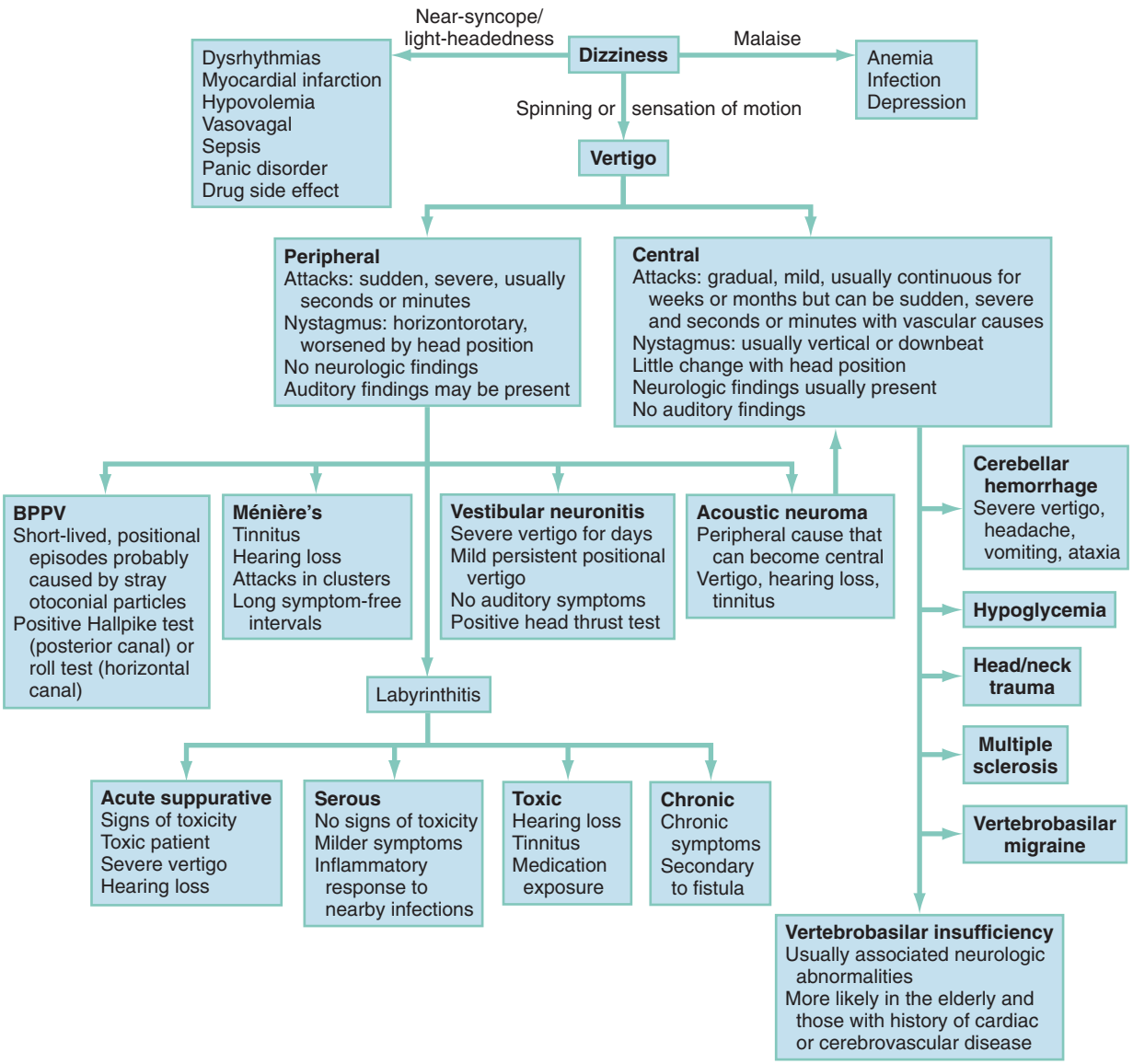

Figure 19-2. Diagnostic algorithm for dizziness and vertigo. *BPPV,* benign paroxysmal positional vertigo.

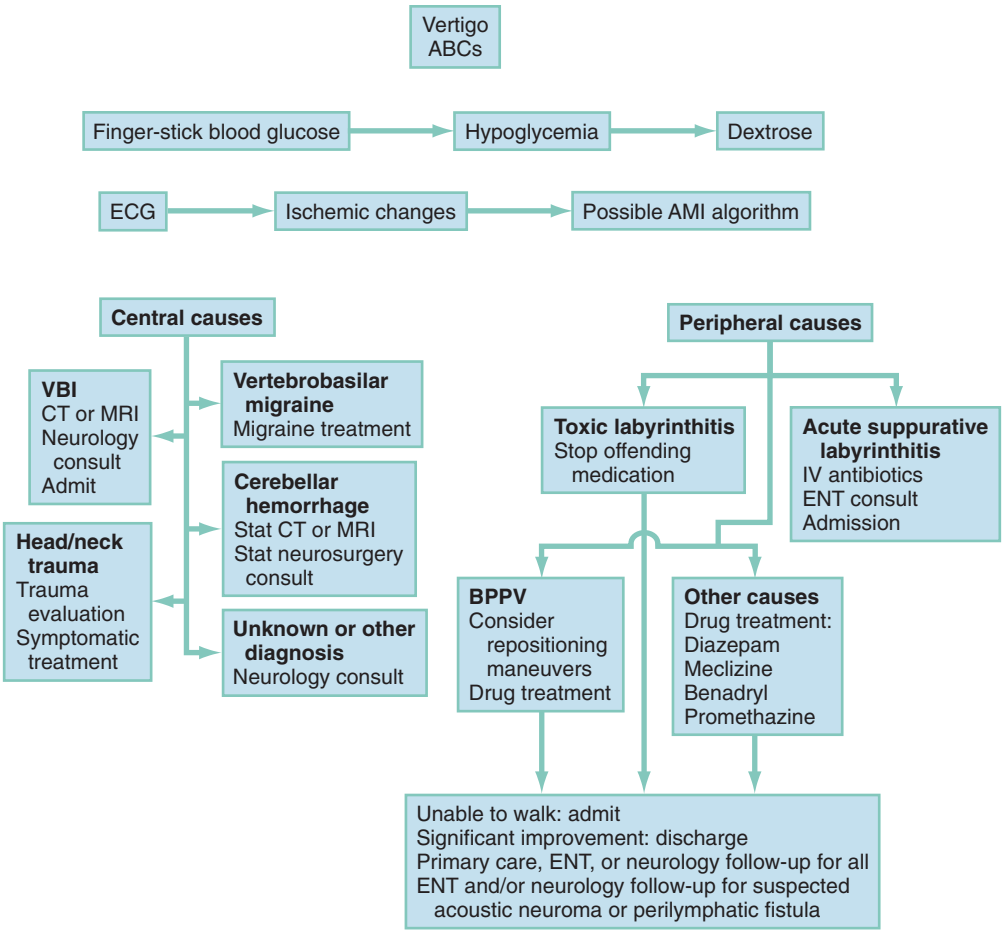

Figure 19-3. Management algorithm for vertigo. *AMI,* acute myocardial infarction; *BPPV,* benign paroxysmal positional vertigo; *CT,* computed tomography; *ECG,* electrocardiogram; *ENT,* ear, nose, and throat; *MRI,* magnetic resonance imaging; *VBI,* vertebrobasilar insufficiency.

admission and consideration of early MRA are reasonable, even in a stable patient. Changing or rapidly progressive symptoms suggest impending posterior circulation occlusion. If CT or MRI excludes hemorrhage as the source of the patient's symptoms, an immediate neurologic consultation, emergency angiography, and possible anticoagulation are indicated.

Acute bacterial labyrinthitis (see Table 19-3) requires hospital admission, intravenous antibiotics, and, occasionally, surgical drainage and débridement. In cases of toxic labyrinthitis, the offending medication is discontinued immediately.

Some cases of Ménière's disease have been treated successfully with vasodilation and diuretic therapy. Diets low in sodium and caffeine and cessation of smoking also have been helpful. Chemical ablation of vestibular function with gentamicin and streptomycin is an option in severe Ménière's disease.

Vestibular neuritis is often thought of as similar to Bell's palsy. A large multicenter randomized clinical trial[19] showed that corticosteroids were helpful in the treatment of vestibular neuritis but antivirals, such as acyclovir, were not. In this study a 22-day taper of methylprednisolone was used beginning with a dose of 100 mg each morning.

Canalith repositioning procedures, such as the Epley maneuver, are extremely effective in treating BPPV.[20,21] The Epley maneuver involves sequential rotations of the head, holding each position for approximately 30 seconds, as demonstrated in Figure 19-4. The "barbecue roll"[22] is a simple maneuver that can be used to treat the horizontal canal variant of BPPV (diagnosed by the roll test). The patient lies flat on the gurney with the head turned 90 degrees to the involved side. The head is then rotated in 45-degree intervals away from the involved side (each turn is held approximately 30 seconds or until nystagmus and vertigo resolve). Eventually the patient needs to turn over into the prone position. The maneuver is completed once the head has returned to the original starting position.

Two relatively recent practice guidelines were published that included information on the use of medications to treat BPPV. One found no evidence to support a recommendation of any medication in the routine treatment of BPPV.[23] The other concluded that clinicians should not routinely treat BPPV with vestibular suppressant medications. However, both guidelines were from specialty societies whose patients often have chronic and likely milder forms of BPPV than patients who develop acute BPPV and come to the ED. For ED patients who either fail canalith repositioning maneuvers or are actively vomiting and/or too symptomatic to undergo repositioning maneuvers, it is reasonable to administer vestibular suppressants. The most commonly used are the antiemetic medications, which not only suppress nausea and vomiting but also decrease the sensation of vertigo. Although promethazine (Phenergan) is likely the most effective parenteral vestibular suppressant, the U.S. Food and Drug Administration (FDA) recently gave intravenous use of promethazine a black box warning, and it is now recommended to be administered only in intramuscular or oral forms. Intravenous ondansetron is an effective alternative parenteral vestibular suppressant.[24] Intravenous metoclopramide is not thought to be helpful with acute vertigo, and there is controversy as to whether intravenous prochlorperazine is effective. Patients with intractable vertigo and vomiting that are unresponsive to antiemetics can be given an

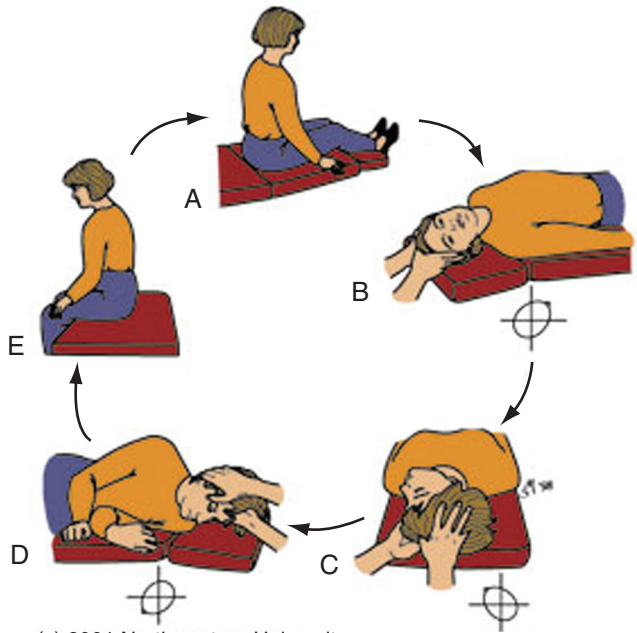

Figure 19-4. The Epley maneuver for benign paroxysmal peripheral vertigo, also known as the *particle repositioning* or *canalith repositioning procedure.* (Image used with permission of Timothy C. Hain, Professor of Neurology, Feinberg School of Medicine, Northwestern University, www.dizziness-and-balance.com/disorders/bppv/bppv.html.)

intravenous benzodiazepine, such as intravenous diazepam. However, it is generally recommended not to discharge patients with oral benzodiazepines because their use can interfere with the process of vestibular habituation, in which the vestibular system learns to adapt to the mismatch of information it is receiving.

Meclizine (Antivert) is the primary oral medication for ambulatory treatment of acute BPPV. It is usually prescribed as 25 mg every 6 to 8 hours as needed. This medication can exacerbate symptoms in patients with nonvertiginous types of dizziness, such as near syncope or disequilibrium, so the physician should be reasonably confident of the diagnosis of BPPV before prescribing meclizine. Transdermal scopolamine has shown disappointing results for treatment of peripheral vertigo but may be considered a third-line option.

Most patients with vertigo have self-limited disease processes that have a specific organic cause. With patient education combined with reassurance and judicious use of medications, the treatment of a dizzy patient can be rewarding for the patient and the physician.

DISPOSITION

Documented or suspected cerebellar hemorrhage or infarction, VBI, and acute bacterial labyrinthitis require diagnostic evaluation, treatment, and, usually, hospitalization. In patients older than age 55, particularly patients with vascular disease, admission for observation and imaging of cerebral vasculature should be considered if the diagnosis of BPPV is uncertain. Most younger patients with peripheral causes of vertigo can be discharged from the ED after symptoms have been controlled. Some patients may have such severe symptoms (e.g., intractable vomiting, inability to walk) despite medications that they require hospital admission for observation status for intravenous hydration, vestibular suppressants, and antiemetics. Reassessment of neurologic examination findings and response to therapy are important to ensure that the vertigo is not of central origin. Discharged patients should receive primary care, neurology, or otolaryngology follow-up, particularly if symptoms are not significantly improved within 72 hours or are worsening despite symptomatic treatment.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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