TABLE 169-3 Causes of Acute Ataxia in Children, Roughly in Order of Frequency

Cause	Example
Drug intoxication	Ethanol Isopropyl alcohol Phenytoin Carbamazepine Sedatives Lead, mercury
Idiopathic	Acute cerebellar ataxia of childhood
Infection and inflammation	Varicella Coxsackievirus A and B Mycoplasma Echovirus Postinfectious inflammation Postimmunization
Neoplasm	Neuroblastoma Other CNS tumors
Paraneoplastic	Opsoclonus-myoclonus syndrome
Trauma	Subdural or epidural posterior fossa hematoma
Congenital or hereditary	Pyruvate decarboxylase deficiency Friedreich's ataxia Hartnup disease
Hydrocephalus Cerebellar abscess Labyrinthitis/vestibular neuronitis Transverse myelitis Meningoencephalitis	

The latency from the prodromal illness to the onset of ataxia is from 2 days to 2 weeks. Other neurologic findings encountered included truncal ataxia, dysmetria, and, uncommonly, cranial nerve abnormalities. Patients with ataxia following varicella appear to have uniform excellent recovery compared with patients with acute cerebellar ataxia from other causes who may have some residual problems.[13] Little workup is needed if the ataxia occurs in the convalescent phase of varicella, and antiviral medications are not indicated. Otherwise, neuroimaging, lumbar puncture, and consultation are advisable. One study showed that although roughly half of the patients had cerebrospinal fluid inflammatory changes with pleocytosis or elevated immunoglobulin G index, MRI identified inflammatory changes in the cerebellum in only a minority of cases.[12] Another small report noted MRI abnormalities not only in the cerebellum, but also in other areas of the CNS. This "syndrome" may in fact consist of several subgroups, some of which involve transient demyelination.[14]

Posterior fossa mass lesions and other CNS masses may present with ataxia, although usually some abnormality of cranial nerves or strength will be discovered with careful examination. Attention is needed to exclude abnormalities on physical examination that might suggest problems not localized to the cerebellum. Abnormal ocular movements should increase the suspicion of a mass lesion. Acute ataxia associated with rapid chaotic eye movements (opsoclonus) and myoclonic extremity jerks of the head and extremities are the striking syndrome of opsoclonus-myoclonus. This may be a postviral syndrome but is often a paraneoplastic syndrome associated with a neuroblastoma located in the abdomen or chest.[10]

Unusual metabolic disorders such as pyruvate decarboxylase complex deficiency may present with ataxia. Family history may or may not suggest a metabolic disorder. Typically, the onset is gradual, but abrupt decompensations may occur. Other systemic or CNS abnormalities will be present.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 170

Vertigo

Brian Goldman

INTRODUCTION AND EPIDEMIOLOGY

Dizziness is a common complaint in patients >40 years old, leading to roughly 10 million visits to ambulatory care settings and 25% of ED visits.[1] Dizziness can lead to falls in elderly patients. The symptoms may persist and be incapacitating. Patients may use various terms for the complaint: dizziness may mean vertigo, syncope, presyncope, weakness, giddiness, anxiety, or a disturbance in mentation.

Vertigo is the perception of movement (rotational or otherwise) where no movement exists. **Syncope** is a transient loss of consciousness accompanied by loss of postural tone with spontaneous recovery. **Near-syncope** is light-headedness with concern for an impending loss of consciousness. **Psychiatric dizziness** is defined as a sensation of dizziness not related to vestibular dysfunction that occurs exclusively in combination with other symptoms as part of a recognized psychiatric symptom cluster.[2] **Disequilibrium** refers to a feeling of unsteadiness, imbalance, or a sensation of "floating" while walking.

Acute vestibular syndrome is a symptom complex consisting of vertigo, nausea and vomiting, intolerance to head motion, spontaneous nystagmus, unsteady gait, and postural instability caused by injury to peripheral or central vestibular structures. To be called acute vestibular syndrome, the associated vertigo must persist for at least 24 hours, thus excluding causes of transient vertigo such as benign paroxysmal positional vertigo (BPPV). Acute vestibular syndrome may be peripheral or central in origin. Clinical findings that distinguish central from peripheral causes include focal neurologic deficits such as hemiparesis, hemisensory loss, or gaze palsy. **The most common peripheral cause of acute vestibular syndrome is vestibular neuronitis. The most common central cause is ischemic stroke of the posterior fossa (brainstem or cerebellar), followed by demyelination. There is growing evidence that a significant number of patients with central acute vestibular syndrome are misdiagnosed in the ED.** A systematic review estimated that roughly 25% of patients presenting with acute vestibular syndrome have had a stroke.[3]

The time-honored paradigm of evaluating dizziness is based largely on the patient's subjective description. More recently, objective bedside physical examination has emerged as a more reliable way of arriving at the correct diagnosis.[4]

PATHOPHYSIOLOGY

The CNS coordinates and integrates sensory input from the visual, vestibular, and proprioceptive systems. Vertigo arises from a mismatch of information from two or more of the involved senses, caused by dysfunction in the sensory organ or its corresponding pathway.

Visual inputs provide spatial orientation. Proprioceptors help relate body movements and indicate the position of the head relative to that of the body. The vestibular system (via the otoliths) establishes the body's orientation with respect to gravity. The cupulae's sensors track rotary motion. The three semicircular canals sense orientation to movement and head tilts and are filled with a fluid called *endolymph*. The endolymphatic sac produces glycoproteins that create an osmotic

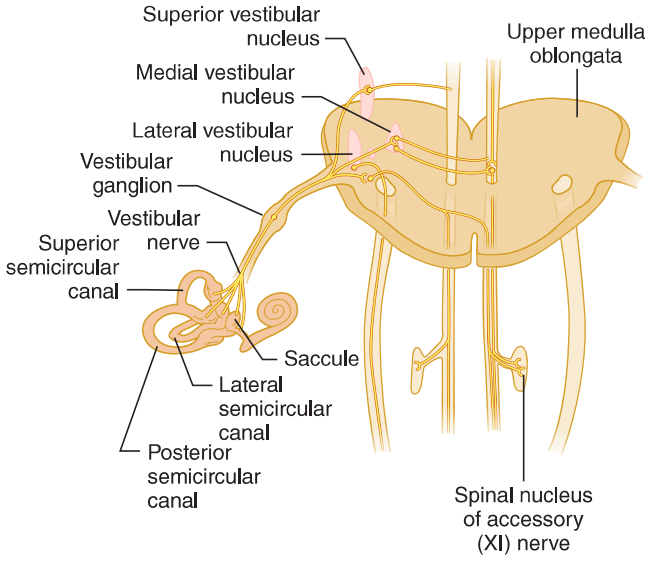

FIGURE 170-1. Vestibular innervation.

sink necessary to maintain flow. The movement of fluid in the semicircular canals causes specialized hair cells inside the canals to move, causing afferent vestibular impulses to fire. Sensory input from the vestibular apparatus travels to the nucleus of the eighth cranial nerve (**Figure 170-1**).

The medial longitudinal fasciculus, the red nuclei, the cerebellum, the parietal lobes, and the superior temporal gyrus of the cerebral cortex integrate the various sensory inputs. Connections between these structures and the oculomotor nuclei that drive the vestibulo-ocular reflex complete the system. The vestibulo-ocular reflex prevents visual blurring from head movements and body sway.

Balanced input from the vestibular apparatus on both sides is the norm. Unilateral lesions of the vestibular apparatus as well as excessive unilateral firing due to abnormal motion of the endolymph produce imbalanced activity and vertigo. Rapid head movements accentuate the imbalance. Symmetric bilateral damage does not usually produce vertigo but may lead to truncal or gait instability.

The most striking clinical sign associated with vertigo is **nystagmus**, a rhythmic movement of the eyes that has both a fast and a slow component, with direction named by its fast component. The slow component is due to the vestibulo-ocular reflex and is generated by excitation of the semicircular canal, producing eye movement away from that canal. The fast component of nystagmus is caused by the cortex, which exerts a quick corrective movement in the opposite direction. Vestibular disorders produce nystagmus that is provoked when the affected side is in the dependent position, and the characteristic pattern is vertical and rotational or horizontal. **Vertical nystagmus by itself usually indicates a brainstem abnormality.** However, an atypical pattern of nystagmus in the absence of other signs of CNS disease does not necessarily indicate central pathology.[5]

The prevalence of dizziness increases with age and is due to decreases in visual acuity, proprioception, and vestibular input, plus an increase in free-floating otoconia within the semicircular canals that cause BPPV. Older patients are also more likely to take medications that cause dizziness.

PHYSIOLOGIC VERTIGO

Physiologic vertigo results from a mismatch of visual, proprioceptive, and vestibular input. This may be the pathogenesis of motion sickness as well as the transient visual vertigo associated with watching a film that captures the visual sensation of motion, by attending complex visual environments, such as shopping malls, and by viewing complex floor or wallpaper patterns.

CLINICAL FEATURES

The conditions that cause acute undifferentiated vertigo are summarized in **Table 170-1**. Vertigo is usually categorized as "peripheral" or "central," and making the distinction between the two is the most important part of the evaluation. **Peripheral vertigo is caused by disorders affecting the vestibular apparatus and the eighth cranial nerve, whereas central vertigo is caused by disorders affecting central structures, such as the brainstem and the cerebellum.** Peripheral vertigo tends to cause distressing symptoms, but is seldom life threatening; the reverse tends to be true for central vertigo. Disorders causing central vertigo often require urgent diagnostic imaging or consultation with a neurologist or neurosurgeon.

Some of the features that distinguish peripheral causes from central causes (mainly cerebrovascular disease) are found in **Table 170-2**. **Note that many of the so-called distinguishing features of peripheral acute vestibular syndrome may also be found in some patients with cerebrovascular disease.**[6]

HISTORY

An approach to vertigo is shown in **Figure 170-2**. Try to obtain an unprompted description of the patient's "dizziness" and avoid leading questions that bias the patient's responses. However, the initial description may not be a reliable predictor of underlying pathology.[7]

Patients with acute vestibular syndrome have continuous vertigo or dizziness for more than 24 hours. Thus, the list excludes causes of brief transient episodes such as BPPV, Ménière's syndrome, and transient ischemic attack. If the patient has experienced true vertigo, determine whether the vertigo is of peripheral or central origin, and determine the temporal pattern and precipitating causes (**Table 170-3**). Peripheral vertigo is more likely than central vertigo to be intense and to be associated with nausea, vomiting, diaphoresis, tinnitus, hearing loss, and photophobia.

Headache is generally not a feature of peripheral causes of acute vestibular syndrome but is associated with central causes. Dizziness and

TABLE 170-1 Causes of Acute Undifferentiated Vertigo

Vestibular/otologic	Benign paroxysmal positional vertigo Traumatic: following head injury Infection: labyrinthitis, vestibular neuronitis, Ramsay Hunt syndrome
Systemic conditions with vestibular/otologic effects	Ménière's syndrome Neoplastic Vascular Otosclerosis Paget's disease Toxic or drug-induced: aminoglycosides
Neurologic	Vertebrobasilar insufficiency or vertebral artery dissection Lateral Wallenberg's syndrome Anterior inferior cerebellar artery syndrome Neoplastic: cerebellopontine angle tumors Cerebellar disorders: hemorrhage, degeneration Basal ganglion diseases Multiple sclerosis Infections: neurosyphilis, tuberculosis Epilepsy Migraine headaches Cerebrovascular disease
General	Hematologic: anemia, polycythemia, hyperviscosity syndrome Toxic: alcohol Chronic renal failure Metabolic: thyroid disease, hypoglycemia

TABLE 170-2 Differentiating Peripheral from Central Causes of Acute Undifferentiated Vertigo

	Peripheral	Central
Onset	Sudden or insidious	Sudden
Severity of vertigo	Intense spinning	Ill defined, less intense
Prodromal dizziness	Occurs in up to 25%; often single episode	Occurs in up to 25%; recurrent episodes suggest transient ischemic attacks
Intolerant of head movements/Dix-Hallpike maneuver	Yes	Sometimes
Associated nausea/diaphoresis	Frequent	Variable
Auditory symptoms	Points to peripheral causes	May be present
Proportionality of symptoms	Usually proportional	Often disproportionate
Headache or neck pain	Unusual	More likely
Nystagmus	Rotatory-vertical, horizontal	Vertical
CNS symptoms/signs	Absent	Usually present
Head impulse test	Abnormal	Usually normal
HINTS examination (combined horizontal head impulse test, nystagmus, and test of skew)	Normal on all three bedside tests	Abnormal on at least one of three bedside tests

headache suggest migraine but also occur with vertebral artery dissection or aneurysm.[8]

Head trauma and medications can precipitate episodes of dizziness or interfere with central adaptation. Recent head or neck trauma is a risk factor for **dissection of the vertebral artery**. In patients with acute vertigo, a recent history of trauma should spark concern for dissection even if pain is absent.

Evaluate the following groups for central vertigo: older patients, those with hypertension or cardiovascular disease, those with other risk factors for stroke, or those taking warfarin. Patients with acute vestibular syndrome and more than one vascular risk factor appear to be at increased risk of stroke. Although age is a known risk factor for stroke, patients age 50 and older with symptoms of acute vertigo and no neurologic signs are more likely to have vestibular neuronitis than stroke.[9] However, age per se does not rule out stroke as the cause of central acute vestibular syndrome. In a study of patients with cerebellar infarctions who were misdiagnosed on initial ED presentation, half of the patients misdiagnosed were younger then 50 years of age who presented with headache and vertigo. All of the patients misdiagnosed had either an incomplete or a poorly documented neurologic examination. Almost all of the patients had a CT scan of the brain that was initially interpreted as normal. The overall mortality was 40%; among survivors, 50% had disabling neurologic sequelae. This study underscores the need for a careful neurologic assessment in all such patients.[10]

PHYSICAL EXAMINATION

Perform complete ear, neurologic, and vestibular examinations in patients presenting with vertigo or dizziness. Examine the external auditory canal and tympanic membrane for evidence of otitis media, cholesteatoma, and other pathology. Test for hearing, and also perform Webber and Rinne testing. Hearing loss usually points to peripheral causes such as vestibular neuronitis and Ménière's syndrome. However, auditory symptoms and signs may also be due to ischemia of the inner ear associated with cerebrovascular disease.

When assessing patients with acute vestibular syndrome, keep the possibility of cerebrovascular disease in mind. The presence of focal neurologic deficits indicates a central etiology. However, focal deficits are not uniformly present in central vertigo. A systematic review found focal neurologic deficits in 80% of patients with acute vestibular syndrome caused by stroke.[3] In the **HINTS** study, neurologic (e.g., facial palsy, hemiparesis, limb ataxia) or oculomotor (e.g., internuclear ophthalmoplegia, gaze palsy, vertical nystagmus) abnormalities were reported in 51% of 76 patients with a central cause and none of 25 patients with a peripheral cause.[8] The presence of gait unsteadiness and severe truncal ataxia (inability to sit unaided with arms crossed) suggests vertigo due to stroke. A positive Romberg test is rarely found in patients with peripheral vertigo. The absence of focal neurologic deficits in some patients with central acute vestibular syndrome has led to growing interest in other methods of bedside testing.

HINTS Testing HINTS testing is an important advance in the rapid assessment of ED patients with acute vestibular syndrome. **HINTS** is an acronym that stands for horizontal head impulse test, nystagmus, and test of skew—three bedside tests that when taken together reliably help distinguish central (usually stroke) from peripheral acute vestibular syndrome.

The horizontal head impulse test is also known as the **Halmagyi head thrust** and, by itself, is the most helpful bedside test for distinguishing central from peripheral vertigo. The Halmagyi head thrust assesses the vestibulo-ocular reflex. Ask the patient to fixate on a visual target, while the examiner rotates the patient's head rapidly first from the center position to 40 degrees left and back again to the center position. Repeat the test on the right side. Carefully watch the patient's eyes for the response to the maneuver. The intact vestibulo-ocular reflex compensates for the rotational head thrust by rapidly and smoothly moving the eyes in the direction **opposite** to the head thrust. This is the normal physiologic response to a passive head thrust; an intact vestibulo-ocular reflex maintains fixation of the eyes with respect to the patient's environment. Thus, the patient will be able to maintain his or her gaze on the visual target throughout the head thrust.

If the vestibulo-ocular reflex is impaired, then the patient will not be able to maintain his or her gaze on the visual target, and the patient will exhibit a rapid simultaneous movement of both eyes (known as a **saccade**) in order to reacquire fixation upon the visual target. This is an abnormal response to the horizontal head impulse test.[11]

Thus, in a patient with no symptoms or signs of acute vestibular syndrome, a normal horizontal head impulse test (i.e., no corrective saccade) means that the vestibulo-ocular reflex is normal. **In a patient with acute vestibular syndrome, an abnormal response horizontal head impulse test (i.e., a corrective saccade) usually indicates a peripheral vestibular lesion, while no corrective saccade is highly suspicious for a stroke.**[12] In the HINTS study, the head impulse test had both a sensitivity and specificity comparable to that of MRI.[8]

The second part of HINTS testing that helps predict central acute vestibular syndrome is direction-changing horizontal nystagmus on lateral gaze. An abnormal response, which consists of right-beating nystagmus on right gaze and left-beating nystagmus on left gaze with or without nystagmus when the patient looks straight ahead, is believed to represent failure of gaze-maintaining structures located in the brainstem and the cerebellum. Direction-changing nystagmus has low sensitivity but high specificity for correctly identifying central causes of acute vestibular syndrome. The third part of HINTS testing is known as skew deviation or skew and refers to vertical ocular misalignment during the alternate cover test. Skew deviation only occurs in the presence of right-left imbalance in sensory inputs from the vestibular system to the oculomotor syndrome. As with direction-changing nystagmus, skew deviation is not sensitive but is highly specific for central acute vestibular syndrome.

All three bedside tests (horizontal head impulse test, nystagmus, and skew) have been combined into the HINTS battery of tests that define a clinical prediction rule for stroke. Just one finding specific for central acute vestibular syndrome on any of the three tests that make up the HINTS battery is considered 100% sensitive and 96% specific for stroke.[8] A cross-sectional study of patients with acute vestibular syndrome at high risk for stroke found that HINTS testing substantially outperformed ABCD2 risk scoring for stroke diagnosis in the ED and also outperformed MRI obtained within the first 2 days of symptom onset.[13] A current review concluded that with appropriate training in the

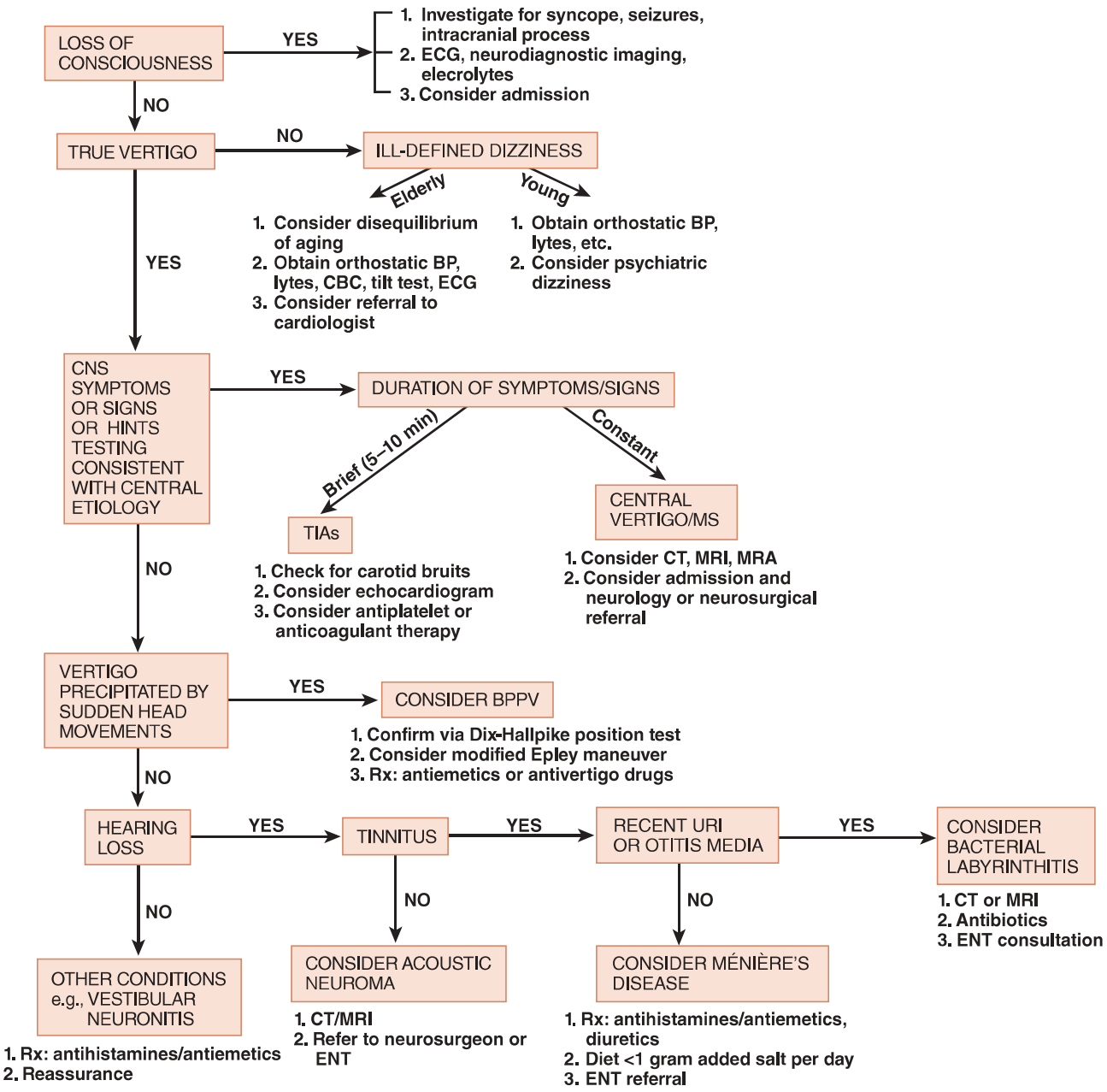

FIGURE 170-2. Guideline approach to vertigo. BP = blood pressure; BPPV = benign paroxysmal positional vertigo; CBC = complete blood count; CNS = central nervous system; ENT = ear, nose, and throat; MRA = magnetic resonance angiography; MS = multiple sclerosis; Rx = treatment; TIAs = transient ischemic attacks; URI = upper respiratory infection.

technique, it is reasonable to use HINTS testing as a tool to distinguish central from peripheral vertigo.[14]

Dix-Hallpike Maneuver **The diagnosis of BPPV involving the posterior canal is aided by the Dix-Hallpike position test.** Do not perform this test on patients with carotid bruits, a history of prior cerebrovascular disease, or risk factors or concern for vertebrobasilar insufficiency, because the Dix-Hallpike maneuver carries a theoretical risk of precipitating a stroke. In addition, use caution in patients with spinal injury or cervical spondylosis. The test may provoke vertigo. Pretreatment with 50 milligrams of dimenhydrinate IM or IV may make the test more tolerable but will not obliterate nystagmus. Have patients keep their eyes open at all times and stare at the examiner's nose or forehead. Start with the patient seated upright on the examining table. To test the right posterior semicircular canal, rotate the head 30 to 45 degrees to the right. Keeping the head in this position, rapidly bring the patient supine until the head is 20 degrees below the level of the examining table. Rotatory nystagmus following a latency of no more than 30 seconds is considered a positive test; the nystagmus exhibits rapid eye torsions toward the affected ear and lasts for 10 to 40 seconds. Return the patient to the upright sitting position, and repeat the test on the left side. **The side exhibiting the positive test is the side of the lesion.**[15]

TABLE 170-3 Temporal Patterns of Vertigo

Pattern	Conditions
Seconds	Benign paroxysmal positional vertigo, postural hypotension
Minutes	Transient ischemic attacks
Hours	Ménière's syndrome
More than 24 hours	Acute vestibular syndrome: peripheral or central

TABLE 170-4 Imaging and Ancillary Testing for Vertigo and Dizziness

Condition	Suggested Tests
Bacterial labyrinthitis	CBC, blood cultures, CT scan, or MRI for possible abscess; lumbar puncture if meningitis suspected
Vertigo associated with closed head injury	CT scan or MRI
Near-syncope	ECG, Holter monitor, CBC, glucose, electrolytes, renal function, table tilt testing
Cardiac dysrhythmias	ECG, Holter monitor
Suspected valvular heart disease	ECG, echocardiography
Nonspecific dizziness; disequilibrium of aging	CBC, electrolytes, glucose, renal function tests
Thyrotoxicosis	Thyroid-stimulating hormone, triiodothyronine, thyroxine
Cerebellar hemorrhage, infarction, or tumor	CT or MRI
Vertebral artery dissection	Cerebral angiogram to include neck vessels or MRA
Vertebrobasilar insufficiency	ECG, cardiac monitoring, echocardiogram, carotid Doppler, MRI, MRA

Abbreviation: MRA = magnetic resonance angiography.

DIAGNOSIS

IMAGING AND ANCILLARY TESTS

Patients with signs or symptoms concerning for central vertigo require emergent imaging and laboratory investigations (**Table 170-4**). CT alone is not adequate because it does not visualize the brainstem well.[16] MRI is more sensitive than CT.[17] For patients who may have vertebrobasilar insufficiency, MRI with magnetic resonance angiography of the head, neck vessels, and circle of Willis; duplex US of the carotid arteries; and neurologic consultation are indicated. Detailed cochleovestibular testing can be done by a specialist.

TREATMENT

SYMPTOMATIC TREATMENT FOR PERIPHERAL VERTIGO

Short-term treatment with antiemetic and vestibular suppressant pharmacotherapy is a mainstay for patients with peripheral vertigo (**Table 170-5**).[18] Withdraw such treatments as soon as possible to facilitate central vestibular compensation.[19]

DRUG THERAPY

Pharmacotherapy is used to treat specific conditions, reduce symptoms, or enhance vestibular compensation. Drugs with anticholinergic effects can be quite effective for vertigo. The agent of choice is transdermal scopolamine. H_1 antihistamines are commonly prescribed drugs for their anticholinergic effects. H_2 antihistamines are not effective. Calcium channel blockers (Table 170-5) possess antihistaminic and antidopaminergic activity and are indicated for the symptomatic relief of vertigo in patients not responding to scopolamine or antihistamines and are also indicated for vestibular migraine. Neuroleptics such as promethazine and metoclopramide reduce nausea and vomiting by blocking brainstem dopaminergic receptors. They too are indicated as second-line treatment. **Do not use prochlorperazine and chlorpromazine in vertigo caused by orthostatic hypotension.** Ondansetron, a serotonin 5-HT_3 receptor antagonist, has been used to treat intractable vertigo in brainstem disorders as well as vertigo due to multiple sclerosis.

Small doses of benzodiazepines such as diazepam and clonazepam may be used sparingly to relieve severe anxiety accompanying vertigo. However, these agents bind to γ-aminobutyric acid receptors in the CNS and thus may impair vestibular compensation. Benzodiazepines are given for central ocular motor disorders that cause nystagmus.

Methylprednisolone is indicated for vestibular neuronitis. However, the antiviral drug valacyclovir has not been proven to be efficacious in clinical trials. Anticonvulsant drugs are indicated for prophylaxis of vestibular migraine; gabapentin is indicated for dizziness associated with multiple sclerosis.

Antihistamines can cause sedation and anticholinergic adverse effects. Antidopaminergic neuroleptic agents can induce or exacerbate orthostatic hypotension. These drugs also cause somnolence and acute dystonia and can exacerbate anticholinergic adverse effects. Avoid using medications with overlapping anticholinergic and antidopaminergic effects in combination. Do not treat patients with nonvertiginous dizziness and disequilibrium of aging with antivertigo medications.

DISORDERS CAUSING PERIPHERAL VERTIGO

Peripheral vertigo produces an intense sensation of spinning or hurtling toward the ground or surrounding walls of abrupt onset. It is worsened by rapid movement and by changes in head position and is frequently associated with nausea, vomiting, diaphoresis, bradycardia, and hypotension.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

BPPV is a mechanical disorder of the inner ear causing transient vertigo (with autonomic symptoms) and associated nystagmus that is precipitated by certain head movements. The lifetime prevalence of BPPV is 2.4% with a 1-year incidence of 0.6%, with women and patients over the age of 50 more likely to be affected. The mean duration of an episode is 2 weeks; 86% of patients affected seek medical attention.[20]

BPPV is believed to be caused by inappropriate activation of a semicircular canal, typically the posterior semicircular canal and typically unilateral, by the presence of free-floating particles or otoconia. The otoconia become displaced from the utricular macula by aging, head trauma, or labyrinthine disease. The particles tend to clump in the long arm of the posterior semicircular canal of the endolymph system. Once the clump reaches sufficient mass, changes in head position cause gravitation of the particles, which creates a plunger effect on the endolymph, causing the cupula to be displaced. This results in inadvertent neural firing, causing both vertigo and nystagmus.

The onset of BPPV is sudden and is precipitated by rolling over in bed, lying supine, leaning forward, looking up at the sky or ceiling, or turning the head. Because the symptoms fatigue, they tend to be worse in the morning. Patients may eliminate the offending activities. There is no associated hearing loss or tinnitus and no physical findings on examination of the external auditory canal.

Several findings support a diagnosis of BPPV (**Table 170-6**). There is a latency period of 1 to 5 seconds between assuming the offending head position and onset of vertigo and nystagmus. Both the vertigo and nystagmus crescendo to a peak of intensity and then subside within 5 to 40 seconds. Unlike vestibular neuronitis, the head thrust test in BPPV is normal and there is no spontaneous nystagmus. The Romberg test is negative, and the gait is normal. Posterior canal **BPPV can be diagnosed using the Dix-Hallpike test** (see "Physical Examination" section).[15] Symptoms disappear with repeated testing. The supine roll test (Pagnini-McClure test[21]) for horizontal canal BPPV is done as follows: place the patient supine, turn the head to the right and observe for nystagmus, and then turn the head back to neutral position. Repeat by turning the head to the left. The side with the most prominent nystagmus is the side of the affected canal.

BPPV involving the anterior semicircular canal is rare, and the Dix-Hallpike maneuver may elicit downbeating nystagmus with the affected ear up. However, any downbeating nystagmus should raise strong suspicion for a cerebellar or brainstem lesion (**Table 170-7**).[21] Patients with isolated BPPV often undergo many tests with little or no diagnostic yield.[22]

TABLE 170-5 Pharmacotherapy of Vertigo and Dizziness

Category	Drug	Dosage	Indications	Advantages	Disadvantages
Anticholinergics	Scopolamine	0.5 milligram transdermal patch (behind ear) three to four times a day	Vertigo, nausea	Useful if patient is vomiting	Sometimes difficult to obtain
Antihistamines	Dimenhydrinate	50–100 milligrams IM, IV, or PO every 4 h	Vertigo, nausea	Inexpensive	Drowsiness/anticholinergic effect
	Diphenhydramine	25–50 milligrams IM, IV, or PO every 4 h	Vertigo, nausea	Inexpensive	Drowsiness/anticholinergic effect
	Meclizine	25 milligrams PO two to four times a day	Vertigo, nausea		Drowsiness/anticholinergic effect
Antiemetics	Hydroxyzine	25–50 milligrams PO four times a day	Vertigo, nausea	Inexpensive	Drowsiness/anticholinergic effect
	Metoclopramide	10–20 milligrams IV, PO three times a day	Vertigo, nausea	Effective, versatile	Occasional extrapyramidal effect
	Ondansetron	4 milligrams IV two to three times a day; 8 milligrams PO twice a day			
	Promethazine	25 milligrams IM, PO, or PR three to four times a day	Vertigo, nausea	Useful if vomiting	Occasional extrapyramidal effect
Benzodiazepines	Diazepam	2–5 milligrams PO two to four times a day	Central vertigo, anxiety related to peripheral vertigo	Inexpensive	Dependency, may impair vestibular compensation
	Clonazepam	0.5 milligram PO two times a day	Central vertigo, anxiety related to peripheral vertigo	Inexpensive	Dependency, may impair vestibular compensation
Calcium antagonists	Cinnarizine	25 milligrams PO two to three times a day	Peripheral vertigo, vestibular migraine	Nonsedating	Lesser clinical experience
	Nimodipine	30 milligrams PO two times a day	Peripheral vertigo, vestibular migraine	Nonsedating	Lesser clinical experience
	Flunarizine	20 milligrams PO two times a day	Ménière's syndrome	Well tolerated	Not available in the United States
Vasodilators	Betahistine	48 milligrams PO three times a day for up to 6–12 mo	Ménière's syndrome	Well tolerated	Little evidence of efficacy for other causes of peripheral vertigo
Corticosteroids	Methylprednisolone	100 milligrams/d tapered by 20 milligrams/d every fourth day	Vestibular neuronitis	Well tolerated	Efficacy largely unproven; adverse effects associated with corticosteroids
Antivirals	Valacyclovir	1000 milligrams three times a day for 7 d	Vestibular neuronitis	Well tolerated	Efficacy largely unproven
Anticonvulsants	Carbamazepine	200–600 milligrams/d	Vestibular paroxysmia	Inexpensive	Monitor CBC and liver function tests
	Topiramate	50–100 milligrams/d	Vestibular migraine prophylaxis	Well tolerated	Not well evaluated; does not abort acute vertigo
	Valproic acid	300–900 milligrams/d	Vestibular migraine prophylaxis	Well tolerated	Not well evaluated; does not abort acute vertigo
	Gabapentin	300 milligrams four times per day	MS associated dizziness	Reduces acquired pendular nystagmus of multiple sclerosis	Known adverse effect profile
β-Blockers	Metoprolol	100 milligrams/d	Vestibular migraine prophylaxis	Long experience	Known adverse effect profile

Perform the particle-repositioning maneuver (or Epley maneuver) for patients with posterior canal BPPV in the ED.[23] **This maneuver uses gravity to induce the particles to move along the semicircular canals until they end up inside the utricle where unlikely to cause vertigo.** It is indicated in patients with suspected BPPV plus a positive Dix-Hallpike test (**Figure 170-3**). Do not perform these maneuvers in patients with neck or cervical spine abnormalities, or if carotid or vertebral dissection is suspected.

TABLE 170-6 Supportive Findings in Benign Paroxysmal Positional Vertigo

Latency period of <30 s between the provocative head position and onset of nystagmus.
The intensity of nystagmus increases to a peak before slowly resolving.
Duration of vertigo and nystagmus ranges from 5–40 s.
If nystagmus is produced in one direction by placing the head down, then the nystagmus reverses direction when the head is returned to the sitting position.
Repeated head positioning causes both the vertigo and accompanying nystagmus to fatigue and subside.
Abnormal horizontal head impulse test indicating abnormal vestibule-ocular reflex function.
HINTS testing not indicative of stroke.

Abbreviation: HINTS = horizontal head impulse test, nystagmus, and test of skew.

TABLE 170-7 Benign Paroxysmal Positional Vertigo (BPPV)

Canal Affected by BPPV	Frequency (%)	Diagnostic Maneuver	Nystagmus (direction named by fast component)
Posterior	85	Dix-Hallpike, affected ear down	Upbeat, affected ear down
Horizontal	10–17	Supine roll test (Pagnini-McClure test)	Horizontal, changes direction when head is turned to right or left while supine
Anterior	1	Dix-Hallpike, affected ear up	Downbeating, great concern for brainstem or cerebellar lesions

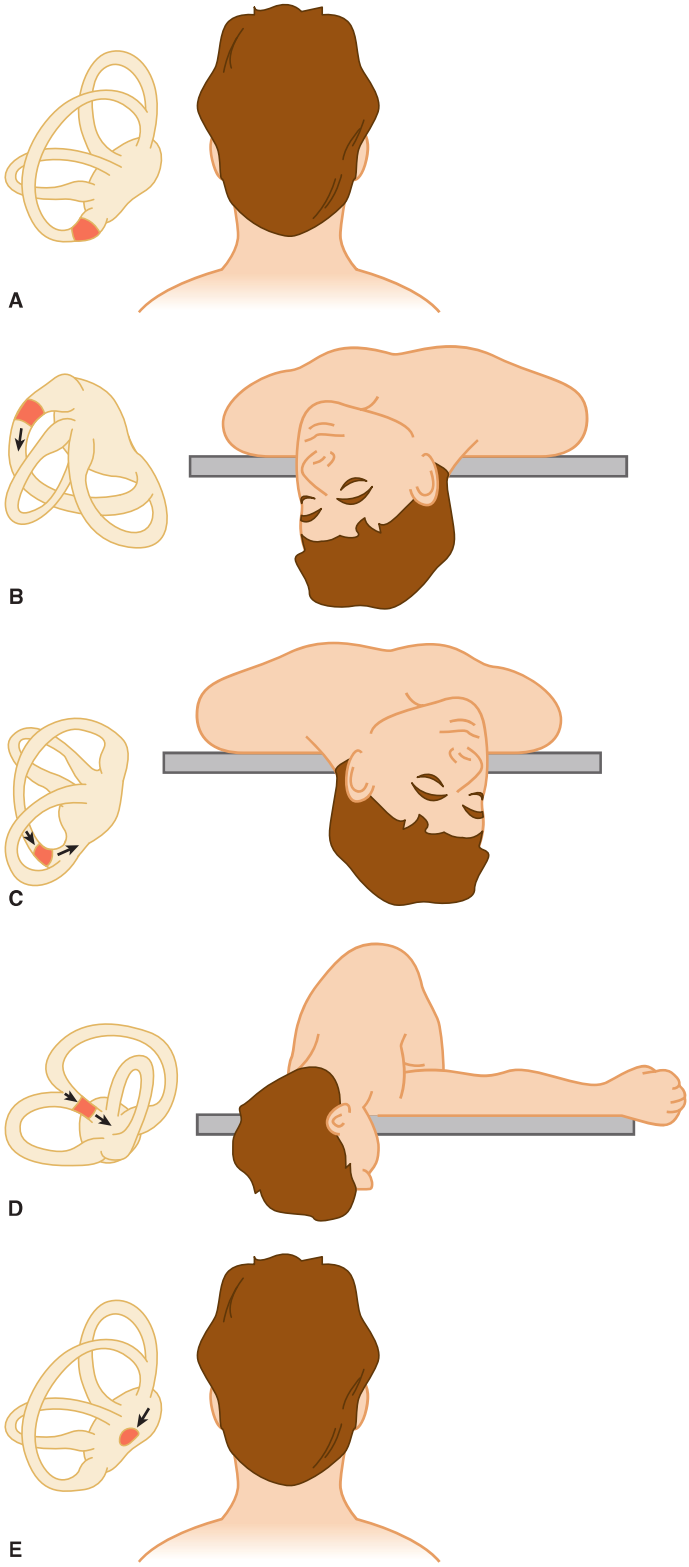

FIGURE 170-3. A through C. Dix-Hallpike position test; D, E. Epley Maneuver. Turn onto unaffected side. Hold position for 60 secs.

The affected ear is determined by the side of the positive Dix-Hallpike position test. Antihistamines or antiemetics administered before the maneuver may improve patient comfort. Seat the patient in the Dix-Hallpike position test, and turn the head 45 degrees toward the affected ear. Gently bring the patient to the recumbent position with the head hanging roughly 20 degrees below the examining table. Gently rotate the head 45 degrees to the midline. Then rotate the head a further 45 degrees to the unaffected side. The patient rolls onto the shoulder of the unaffected side, at the same time rotating the head a further 45 degrees. Return the patient to the sitting position and return the head to the midline. Wait 5 minutes after each portion of the maneuver to permit the particles to traverse their intended course. Observe for nystagmus in the same direction as during Dix-Hallpike testing. Nystagmus in the opposite direction suggests the particles have moved back toward the cupula; this portends an unsuccessful maneuver. Repeat the maneuver until both the vertigo and the accompanying nystagmus have disappeared. A home treatment device has been found to enable patients with an established diagnosis of BPPV to perform the maneuver themselves safely and effectively.[24]

While the Epley maneuver is safe and effective for posterior canal BPPV, relapses are common. Adverse effects include light-headedness and exacerbation of vertigo.

Most episodes of BPPV resolve spontaneously after a few days. Refer patients with persistent symptoms to an otolaryngologist.

MÉNIÈRE'S SYNDROME

Ménière's syndrome is a disorder associated with an increased endolymph within the cochlea and labyrinth. It tends to occur in older men and women with equal prevalence. The disease is usually unilateral but may become bilateral over time. The precise pathogenesis is unknown, but evidence suggests that patients have difficulty regulating the volume, flow, and composition of endolymph. The onset is usually sudden, with associated nausea, vomiting, and diaphoresis, with attacks lasting from 20 minutes to 12 hours. The frequency of attacks varies from several times per week to several times per month. Other associated symptoms include tinnitus, diminished hearing, and fullness in one ear. Between attacks, the patient is usually well, although decreased hearing may persist.

Ménière's syndrome is managed symptomatically with antihistamines and betahistine; combination therapy with triamterene and hydrochlorothiazide is also recommended in confirmed cases. Ménière's syndrome is the only condition for which betahistine has proven efficacy. A high-dose regimen of at least 48 milligrams three times daily has been shown to provide long-term prophylaxis.[25] Calcium channel blockers may also be used (Table 170-5). None of these drug treatments improves hearing. Intratympanic gentamicin administration may provide significant immediate and long-term relief.[26] Refer patients for treatment to an otolaryngologist. Attacks of vertigo are generally controllable, but tinnitus and hearing loss tend to be unresponsive to therapy.[27]

PERILYMPH FISTULA

A perilymph fistula is an opening in the round or oval window that permits pneumatic changes in the middle ear to be transmitted to the vestibular apparatus. Trauma, infection, or a sudden change in the pressure inside the ventricular system may cause the tear. The diagnosis is suggested by the sudden onset of vertigo associated with flying, scuba diving, severe straining, heavy lifting, coughing, or sneezing. Associated symptoms may include hearing loss. **The diagnosis is confirmed by nystagmus elicited by pneumatic otoscopy (Hennebert sign).**

Perilymph fistula is managed with symptomatic treatment and bed rest and referral to an ear, nose, and throat specialist for surgical repair (emergent referral for patients with acute associated hearing loss).

VESTIBULAR NEURONITIS

Vestibular neuronitis, a disorder of suspected viral etiology, is the second most common cause of peripheral vertigo. Unlike BPPV and Ménière's syndrome, vestibular neuronitis typically lasts several days and does not recur. The onset is sudden, often with a current or recent viral illness. Intense vertigo may require bedrest for several days. Unilateral loss of hearing and tinnitus may occur. Positive findings on physical examination include positive head thrust and horizontal or mild torsional nystagmus. The Romberg test is negative; however, the gait tends to be slow, cautious, and widely based. The condition remits spontaneously with no recurrence. Treatment is symptomatic. Both methylprednisolone and valacyclovir have been recommended, but there is insufficient evidence that these agents enhance recovery.[28]

VESTIBULAR GANGLIONITIS

Vestibular ganglionitis is believed to be caused by a neurotrophic virus such as varicella zoster reactivated years following initial infection. Herpes zoster oticus, also known as the Ramsay Hunt syndrome, is a neuropathic disorder thought to be associated with vestibular ganglionitis. It is characterized by deafness, vertigo, and facial nerve palsy. **The diagnosis is confirmed by the presence of grouped vesicles on an erythematous base inside the external auditory canal.** Treat this disorder with antiviral therapy started within 72 hours of the appearance of vesicles along with symptomatic treatments.

LABYRINTHITIS

Labyrinthitis, an infection of the labyrinth, causes peripheral vertigo associated with hearing loss. Viral labyrinthitis (associated with measles and mumps) has a course that is similar to vestibular neuronitis. Serous labyrinthitis occasionally causes vertigo.

Bacterial labyrinthitis may be a sequela of otitis media, in which bacteria and toxins diffuse across the membrane of the round window. Possible antecedents for bacterial labyrinthitis include otitis media with fistula, meningitis, mastoiditis, cholesteatoma, and dermoid tumor. **The hallmarks of this disease include sudden onset of vertigo with associated hearing loss and middle ear findings.**

Patients with bacterial labyrinthitis are at risk for meningitis and require antibiotics and referral to an ear, nose, and throat specialist for admission and possible surgical drainage.

OTOTOXICITY

Various drugs have been found to be ototoxic (**Table 170-8**).[29] Aminoglycoside antibiotics produce hearing loss and peripheral vestibular dysfunction by accumulating inside the endolymph, causing the death of cochlear and vestibular hair cells. However, because both inner ears are affected, vertigo is uncommon. Clinical manifestations include ataxia and oscillopsia (defined as the inability to maintain visual fixation while moving). The damage is usually irreversible but is dose- and duration-dependent. Loop diuretics (furosemide and ethacrynic acid) also cause irreversible vestibular toxicity and ototoxicity. *N*-Acetylcysteine administered orally may help prevent aminoglycoside-induced ototoxicity in hemodialysis patients.[30] Cytotoxic agents such as vinblastine and cisplatin cause vestibular damage; however, newer platinums, such as carboplatin, are less likely to do so. The antiarrhythmic drug quinidine and antimalarial drugs derived from quinine, such as chloroquine and mefloquine, also can cause vestibular symptoms that may be irreversible.

Reversible causes of vestibular damage and ototoxicity include nonsteroidal anti-inflammatory drugs, salicylates, minocycline, erythromycin, and some fluoroquinolones. Isolated cases of unsteady gait have been observed with antiviral drugs such as abacavir as well as antiparasitic agents. Solvents and other chemicals such as propylene glycol, toluene, mercury, and hydrocarbons can cause both peripheral and central vestibular symptoms.

TABLE 170-8 Ototoxic and Vestibulotoxic Agents

Agent	Dose Dependent	Reversible
Aminoglycosides	Yes	Usually not; possible improvement with *N*-acetylcysteine
Erythromycin	No	Yes
Minocycline	No	Yes
Fluoroquinolones	No	Yes
Nonsteroidal anti-inflammatory drugs; salicylates	Yes	Yes
Loop diuretics	No	Can be irreversible
Cytostatic drugs	Yes	No
Antimalarials	No	Yes
Anticonvulsants	Yes	Yes

Drugs that sometimes induce a central vestibular syndrome include tricyclic antidepressants, neuroleptics, opiates, and alcohol. Anticonvulsants cause dizziness and ataxia, especially in older patients. Lamotrigine may cause less dizziness.[31] Phenytoin, toluene, and cancer chemotherapy agents can cause irreversible cerebellar toxicity. Phencyclidine is a recreational drug that causes central vestibular symptoms, including nystagmus and ataxia.

In general, most patients adapt to chronic vertigo by relying on intact proprioception and vision. However, **benzodiazepines and neuroleptics that are often used as antivertigo therapy may exacerbate symptoms by delaying or inhibiting such compensation.** Thus, avoid using this therapy on a long-term basis. Refer patients with suspected ototoxicity to an otolaryngologist.

EIGHTH NERVE LESIONS AND CEREBELLOPONTINE ANGLE TUMORS

Lesions of the eighth cranial nerve such as meningiomas and acoustic schwannomas may produce mild vertigo. The onset is usually gradual and remains constant until central compensation takes place. Hearing loss usually precedes the vertigo. Tumors of the cerebellopontine angle such as acoustic neuromas, meningiomas, and dermoids may also cause vertigo. **These usually present with deafness and ataxia, as well as ipsilateral facial weakness, loss of the corneal reflex, and cerebellar signs.** All such patients require urgent diagnostic imaging as well as referral to a neurosurgeon.

POSTTRAUMATIC VERTIGO

Acute posttraumatic vertigo and unsteady gait are caused by a direct injury to the labyrinthine membranes. The onset of vertigo is immediate and is accompanied by nausea and vomiting. There may be a concomitant fracture of the temporal bone. Vertigo associated with a closed head injury warrants CT or MRI to exclude an intracranial hemorrhage. Vertigo due to direct labyrinthine trauma tends to resolve within several weeks. Closed head trauma also can displace otoconia from the utricular maculae, precipitating an attack of BPPV. Postconcussive syndrome can be associated with unsteadiness of gait and a vague sense of dizziness. These patients may be treated symptomatically, with referral to a specialist if symptoms fail to resolve.

VERTIGO AFTER COCHLEAR IMPLANTATION

Vertigo is a well-known complication of cochlear implantation. The etiology is likely multifactorial and includes the dislodging of otoconia and the introduction of bone dust into the labyrinth.[32]

DISORDERS CAUSING CENTRAL VERTIGO

Central vertigo is caused by disorders affecting the cerebellum and the brainstem. These include cerebrovascular disease, hemorrhage, migraine, demyelination, and neoplasms. Central vertigo is unlikely to be associated with tinnitus and hearing impairment. Nystagmus is more likely to be vertical than horizontal or rotatory and may be present in the absence of vertigo.

CEREBELLAR HEMORRHAGE AND INFARCTION

Cerebellar hemorrhage typically causes acute vertigo and ataxia along with headache, nausea, and vomiting. Instead of intense vertigo, patients tend to complain of a sense of side-to-side or front-to-back motion. Patients may have truncal ataxia and may not be able to sit without support. Romberg testing and tandem gait will be abnormal. Occasionally, there may be a sixth cranial nerve palsy or conjugate eye deviation away from the side with the hemorrhage. Cerebellar infarction has a similar clinical presentation. Such patients require emergent MRI, and those with cerebellar hemorrhage require emergent neurosurgical consultation.

WALLENBERG'S SYNDROME

A lateral medullary infarction (Wallenberg's syndrome) of the brainstem can cause vertigo as part of its clinical presentation. **Classic ipsilateral findings include facial numbness, loss of corneal reflex, Horner's syndrome, and paralysis or paresis of the soft palate, pharynx, and larynx (causing dysphagia and dysphonia).** Contralateral findings include loss of pain and temperature sensation in the trunk and limbs. Occasionally, lesions of the sixth, seventh, and eighth cranial nerves can occur, causing vertigo, nausea, vomiting, and nystagmus. These patients require emergent MRI and neurologic consultation.

VERTEBROBASILAR INSUFFICIENCY

Transient ischemic attack of the brainstem due to vertebrobasilar insufficiency can produce vertigo that may closely mimic peripheral vestibular disorders. Focal neurologic signs may be absent in more than half of patients.[3] Patients have typical risk factors for cerebrovascular disease. As with transient ischemic attacks in general, the vertigo may be of sudden onset and typically lasts from minutes to hours and should resolve completely within 24 hours. Vertebrobasilar insufficiency–induced vertigo may be accompanied by diplopia, dysphagia, dysarthria, bilateral long-tract signs, and bilateral loss of vision. Unlike other causes of central vertigo, vertebrobasilar insufficiency may be provoked by position. Turning the head partially occludes the ipsilateral vertebral artery. **If the contralateral artery is stenotic, head turning could cause transient ischemia to the brainstem, resulting in vertebrobasilar insufficiency.** Order a brain MRI and consult a neurologist in patients suspected of having vertebrobasilar insufficiency.

VERTEBRAL ARTERY DISSECTION

Vertebral artery dissection can lead to a stroke involving the posterior circulation. **The most common symptoms of vertebral artery dissection are dizziness, headache, and neck pain.** However, one in four patients with dissection present without headache.[3] Unlike vestibular migraine, the onset of headache is often sudden and severe. Recent head or neck trauma is a known risk factor. A history of trauma (e.g., motor vehicle crash, diving injury, or even coughing or sneezing) along with headache and dizziness should spark concern for underlying dissection. It is a rare but recognized complication of chiropractic neck manipulation.[33] The age of presentation is usually less than 50 years. Patients with suspected dissection require emergent diagnostic imaging and referral.

MULTIPLE SCLEROSIS

Demyelinating disease can present with vertigo that lasts several hours to several weeks and is usually nonrecurrent. The vertigo is mild, with nystagmus the most prominent finding on physical examination. Such patients require confirmatory testing with MRI as well as vestibular evoked myogenic potentials plus urgent referral to a neurologist.

NEOPLASMS

Neoplasms of the fourth ventricle can cause brainstem signs and symptoms, including vertigo. Such tumors include ependymomas in younger patients and metastases in older patients. These patients require diagnostic imaging while in the ED and should be referred to a neurosurgeon.

VESTIBULAR MIGRAINE

There is an epidemiologic link between vertigo and migraine. A disproportionate number of patients presenting to a dizziness clinic have a history of migraine, and the prevalence of vertigo is increased in patients with migraine. Vertigo can be a symptom of an aura, an analog or equivalent of the headache phase itself, or an associated symptom with the migraine prodrome. Basilar migraine is a migraine variant in which the aura has clinical manifestations similar to those of vertebrobasilar insufficiency. Vestibular migraine is the most common cause of recurrent spontaneous vertigo and should be considered in almost any patient with dizziness and headache.

The diagnostic criteria for migraine-related vertigo include a history of vertigo not attributable to other known conditions and a present or past history of migraine or a strong family history. In a first episode, cerebrovascular disease may have to be excluded.

Acute episodes are treated with acetaminophen and antiemetics. Frequent attacks may be managed with prophylactic agents such as metoprolol, topiramate, or valproic acid. **Avoid using ergotamine preparations or sumatriptan in patients with basilar migraine.**

Patients with Ménière's syndrome also have an increased prevalence of migraine. Therefore, patients who fail to respond to therapy specific for Ménière's syndrome may benefit from therapy for vertigo associated with migraine headaches.

SPECIAL CONSIDERATIONS

DISEQUILIBRIUM OF AGING

Disequilibrium of aging (sometimes referred to as "psychomotor disadaptation syndrome") manifests as ill-defined dizziness and gait unsteadiness. It is associated with age-related loss of hearing, balance, proprioceptive input, and vision, resulting in an alteration of postural reactions, reactional hypertonia, gait modifications, and fear of falling.[34] Other factors include a decline in central integration and processing, as well as a decrease in motor responses. Symptoms may be precipitated or exacerbated by diminished ambient light (with worsening of symptoms at night), unfamiliar surroundings, and the use of benzodiazepines and drugs with anticholinergic effects such as tricyclic antidepressants and neuroleptic agents. Refer these patients to an internist or gerontologist.

CONVULSIVE DISORDERS

Nonconvulsive status epilepticus, which is characterized by altered mental status without loss of consciousness or tonic-clonic phenomena yet associated with electroencephalographic evidence of seizure activity, may also produce nonvertiginous dizziness. Symptoms may last for hours to days. Refer patients with suspected convulsive disorders to a neurologist.

HYPERVENTILATION SYNDROME

Patients with primary hyperventilation may experience nonvertiginous dizziness or near-syncope during an episode. Diagnostic clues include paresthesias and carpopedal spasm.

PSYCHIATRIC DIZZINESS

Psychiatric dizziness presents as part of a recognized psychiatric disorder or symptom complex that is not related to known vestibular disorders. Dizziness, which can be chronic, is often reported by patients with primary and secondary anxiety disorders, especially panic disorder.[35] The diagnosis is made by exclusion of other more serious causes of dizziness.

DISPOSITION AND FOLLOW-UP

Discharge patients with peripheral vertigo from the ED once symptoms are controlled. Patients can be referred for vestibular rehabilitation therapy, an exercise-based program that promotes CNS compensation for peripheral vertigo. **Vestibular exercises are indicated for patients with BPPV, chronic vertigo, and psychiatric dizziness.** A Cochrane systematic review concluded that vestibular rehabilitation is safe and effective for unilateral peripheral vestibular vertigo.[33]

Refer all patients with a first episode of peripheral vertigo to their primary care physician or an otolaryngologist for further testing. Refer patients with BPPV who have had a particle-repositioning maneuver to an otolaryngologist for follow-up.

All patients with central vertigo require neuroimaging and other diagnostic testing to establish the diagnosis; life-threatening central causes of vertigo (e.g., stroke, cerebellar hemorrhage) require emergent diagnostic