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■ ■ FURTHER READING Brignole M et al: 2018 ESC Guidelines for the diagnosis and manage

ment of syncope. Eur Heart J 39:1883, 2018. Brignole M et al: Cardiac pacing in severe recurrent reflex syncope and tilt-induced asystole. Eur Heart J 42:508, 2021. Cheshire WP et al: Electrodiagnostic assessment of the autonomic nervous system: A consensus statement endorsed by the American Autonomic Society, American Academy of Neurology, and the International Federation of Clinical Neurophysiology. Clin Neurophysiol 132:666, 2021. Freeman R et al: Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. Auton Neurosci 161:46, 2011. Freeman R et al: Orthostatic Hypotension: JACC state-of-the-art PART 2 Cardinal Manifestations and Presentation of Diseases review. J Am Coll Cardiol 72:1294, 2018. Gibbons CH et al: The recommendations of a consensus panel for the screening, diagnosis, and treatment of neurogenic orthostatic hypotension and associated supine hypertension. J Neurol 264:1567, 2017. Sheldon RS, Raj SR: Pacing and vasovagal syncope: Back to our physiologic roots. Clin Auton Res 27:213, 2017. Sheldon R et al: Midodrine for the prevention of vasovagal syncope: A randomized clinical trial. Ann Intern Med 74:1349, 2021. Shen WK et al: 2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Circulation 136:e60, 2017. Mark F. Walker, Robert B. Daroff*

Dizziness and Vertigo Dizziness is an imprecise symptom used to describe a variety of common sensations that include vertigo, light-headedness, faintness, and imbalance. Vertigo refers to a sense of spinning or other motion that may be physiological, occurring during or after a sustained head rotation, or pathological, due to vestibular dysfunction. The term lightheadedness is classically applied to presyncopal sensations resulting from brain hypoperfusion but as used by patients has little specificity, as it may also refer to other symptoms such as disequilibrium and imbalance. A challenge to diagnosis is that patients often have difficulty distinguishing among these various symptoms, and the words they choose do not reliably indicate the underlying etiology. There are many causes of dizziness. Vestibular dizziness (vertigo or imbalance) may be due to peripheral disorders that affect the labyrinths or vestibular nerves, or it may result from disruption of central vestibular pathways. It may be paroxysmal or due to a fixed unilateral or bilateral vestibular deficit. Acute unilateral lesions cause vertigo due to a sudden imbalance in vestibular inputs from the two labyrinths. Bilateral lesions cause imbalance and instability of vision

when the head moves (oscillopsia) due to loss of normal vestibular reflexes. **Ocular Motility** The range of eye movements and whether they are equal in each eye should be observed. Peripheral eye movement disorders (e.g., cranial neuropathies, eye muscle weakness) are usually disconjugate (different in the two eyes). One should check pursuit (the ability to follow a smoothly moving target) and saccades (the ability to look back and forth accurately between two targets). Poor pursuit or inaccurate (dysmetric) saccades usually indicate central pathology, often involving the cerebellum. Alignment of the two eyes can be checked with a cover test: while the patient is looking at a target, alternately cover the eyes and observe for corrective saccades. A vertical misalignment may indicate a brainstem or cerebellar lesion. Finally, one should look for spontaneous nystagmus, an involuntary back-and-forth movement of the eyes. Nystagmus is most often of the jerk type, in which a slow drift (slow phase) in one direction alternates with a rapid saccadic movement (quick phase or fast phase) in the opposite direction that resets the position of the eyes in the orbits. Except in the case of acute vestibulopathy (e.g., vestibular neuritis), if nystagmus is *Deceased. Presyncopal dizziness occurs when cardiac dysrhythmia, orthostatic hypotension, medication effects, or another cause leads to brain hypoperfusion. Such presyncopal sensations vary in duration; they may increase in severity until loss of consciousness occurs, or they may resolve before loss of consciousness if the cerebral ischemia is corrected. Faintness and syncope, which are discussed in detail in Chap. 23, should always be considered when one is evaluating patients with brief episodes of dizziness or dizziness that occurs with upright posture. Other causes of dizziness include nonvestibular balance and

gait disorders (e.g., loss of proprioception from sensory neuropathy, parkinsonism) and anxiety. When evaluating patients with dizziness, questions to consider include the following: (1) Is it dangerous (e.g., arrhythmia, transient ischemic attack/stroke)? (2) Is it vestibular? (3) If vestibular, is it peripheral or central? A careful history and examination often provide sufficient information to answer these questions and determine whether additional studies or referral to a specialist is necessary. **APPROACH TO THE PATIENT HISTORY** When a patient presents with dizziness, the first step is to delineate more precisely the nature of the symptom. In the case of vestibular disorders, the physical symptoms depend on whether the lesion is unilateral or bilateral, and whether it is acute or chronic. Vertigo, an illusion of self or environmental motion, implies an asymmetry of vestibular inputs from the two labyrinths or in their central pathways. Symmetric bilateral vestibular hypofunction causes imbalance but no vertigo. Because of the ambiguity in patients' descriptions of their symptoms, diagnosis based simply on symptom characteristics is typically unreliable. Thus, the history should focus closely on other features, including whether this is the first attack, the duration of this and any prior episodes, provoking factors, and accompanying symptoms. **Timing** Dizziness can be divided into episodes that last for seconds, minutes, hours, or days. Common causes of brief dizziness (seconds) include benign paroxysmal positional vertigo (BPPV) and orthostatic hypotension, both of which typically are provoked by changes in head and/or body position relative to gravity. Attacks of vestibular migraine and Ménière's disease often last hours. When episodes are of intermediate duration (minutes), transient ischemic attacks of the posterior circulation should be considered, although migraine and other causes are also possible. **Associated Symptoms** Symptoms that accompany vertigo may be helpful in distinguishing peripheral vestibular lesions from central causes. Unilateral hearing loss and other acute aural symptoms (ear pain, pressure, fullness, new tinnitus) typically point to a peripheral cause. Because the auditory pathways quickly become bilateral upon entering the brainstem, central lesions are unlikely to cause unilateral hearing loss unless the lesion lies near the root entry zone

of the auditory nerve. Symptoms such as double vision, numbness, and limb ataxia suggest a brainstem or cerebellar lesion. **EXAMINATION** Because dizziness and imbalance can be a manifestation of a variety of neurologic disorders, the neurologic examination is essential in the evaluation of these patients. Focus should be given to assessment of eye movements, vestibular function, and hearing.

TABLE 24-1 Features of Peripheral and Central Vertigo

- Nystagmus from an acute peripheral lesion is unidirectional, with fast phases beating away from the ear with the lesion. Nystagmus that changes direction with gaze is due to a central lesion.
- Transient mixed vertical-torsional nystagmus occurs in benign paroxysmal positional vertigo (BPPV), but pure vertical or pure torsional nystagmus is a central sign.
- Nystagmus from a peripheral lesion may be inhibited by visual fixation, whereas central nystagmus is not suppressed.
- Absence of a head impulse sign in a patient with acute prolonged vertigo should suggest a central cause.
- Unilateral hearing loss suggests peripheral vertigo. Findings such as diplopia, dysarthria, and limb ataxia suggest a central disorder.

easily seen in the light, it is probably due to a central cause. Two forms of nystagmus that are characteristic of lesions of the cerebellar pathways are vertical nystagmus with downward fast phases (downbeat nystagmus) and horizontal nystagmus that changes direction with gaze (gaze-evoked nystagmus). By contrast, peripheral lesions typically cause unidirectional horizontal nystagmus. Use of Frenzel eyeglasses (self-illuminated goggles with convex lenses that blur the patient's vision but allow the examiner to see the eyes greatly magnified) or infrared video goggles can aid in the detection of peripheral vestibular nystagmus, because they reduce the patient's ability to use visual fixation to suppress nystagmus. Table 24-1 outlines key findings that help distinguish peripheral from central causes of vertigo.

Head Impulse Test The most useful bedside test of peripheral vestibular function is the head impulse test, in which the vestibuloocular reflex (VOR) is assessed with small-amplitude (~20 degrees) rapid head rotations. While the patient fixates on a target, the head is rotated quickly to the left or right. If the VOR is deficient, the rotation is followed by a catch-up saccade in the opposite direction (e.g., a leftward saccade after a rightward rotation). The head impulse test can identify both unilateral (catch-up saccades after rotations toward the weak side) and bilateral (catch-up saccades after rotations in both directions) vestibular hypofunction.

Positioning Maneuvers All patients with episodic dizziness, especially if provoked by positional change, should be tested with the Dix-Hallpike maneuver. The patient begins in a sitting position with the head turned 45 degrees; holding the back of the head, the examiner then lowers the patient into a supine position with the head extended backward by about 20 degrees while watching the eyes. Posterior canal BPPV can be diagnosed confidently if transient upbeat-torsional nystagmus is seen. If no nystagmus is observed after 15–20 s, the patient is raised to the sitting position, and the procedure is repeated with the head turned to the other side. Again, Frenzel goggles may improve the sensitivity of the test.

Dynamic Visual Acuity This is a functional test that can be useful in assessing vestibular function. Visual acuity is measured with the head still and when the head is rotated back and forth by the examiner (about 1–2 Hz). A drop in visual acuity during head motion of more than one line on a near card or Snellen chart is abnormal and indicates vestibular dysfunction.

ANCILLARY TESTING The choice of ancillary tests should be guided by the history and examination findings. Audiometry should be performed whenever a vestibular disorder is suspected. Unilateral sensorineural hearing loss supports a peripheral disorder (e.g., vestibular schwannoma). Predominantly low-frequency hearing loss is characteristic of Ménière's disease. Videonystagmography includes recordings of spontaneous nystagmus (if present) and measurement of positional nystagmus. Caloric testing compares the

responses of the two horizontal semicircular canals, while video head-impulse testing measures the integrity of each of the six semicircular canals.

Vestibular evoked potentials assess otolith reflexes. The test battery often includes recording of saccades and pursuit to evaluate central ocular motor function. Neuroimaging is important if a central vestibular disorder is suspected. In addition, patients with unexplained unilateral hearing loss or vestibular hypofunction should undergo MRI of the internal auditory canals, including administration of gadolinium, to rule out a schwannoma. ■ ■

DIFFERENTIAL DIAGNOSIS AND TREATMENT Treatment of vestibular symptoms should be driven by the underlying diagnosis. Simply treating dizziness with vestibular suppressant medications is often not helpful and may prolong recovery. The diagnostic and specific treatment approaches for the most commonly encountered vestibular disorders are discussed below. Dizziness and Vertigo CHAPTER 24 ■

■ **ACUTE PROLONGED VERTIGO**

(**VESTIBULAR NEURITIS**) An acute unilateral vestibular lesion causes constant vertigo, nausea, vomiting, oscillopsia (motion of the visual scene), and imbalance. These symptoms are due to a sudden asymmetry of inputs from the two labyrinths or in their central connections, simulating a continuous rotation of the head. Unlike BPPV, continuous vertigo persists even when the head remains still. History and Examination When a patient presents with an acute vestibular syndrome, the most important question is whether the lesion is central (e.g., a cerebellar or brainstem infarct or hemorrhage), which may be life-threatening, or peripheral, affecting the vestibular nerve or labyrinth (vestibular neuritis). Attention should be given to any symptoms or signs that point to central dysfunction (diplopia, weakness or numbness, dysarthria). The pattern of spontaneous nystagmus, if present, may be helpful (Table 24-1). If the head impulse test is normal, an acute peripheral vestibular lesion is unlikely. A central lesion cannot always be excluded with certainty based on symptoms and examination alone; thus, older patients with vascular risk factors who present with an acute vestibular syndrome should be evaluated for the possibility of stroke even when there are no specific findings that indicate a central lesion. Treatment Most patients with vestibular neuritis recover spontaneously, although chronic dizziness, motion sensitivity, and disequilibrium may persist. The role of early glucocorticoid therapy is uncertain, as studies have yielded disparate results. Antiviral medications are of no proven benefit and are not typically given unless there is evidence to suggest herpes zoster oticus (Ramsay Hunt syndrome). Vestibular suppressant medications may reduce acute symptoms but should be avoided after the first several days because they may impede central compensation and recovery. Patients should be encouraged to resume a normal level of activity as soon as possible, and directed vestibular rehabilitation therapy may accelerate improvement. ■ ■

BENIGN PAROXYSMAL POSITIONAL VERTIGO BPPV is a common cause of recurrent vertigo. Episodes are brief (<1 min and typically 15–20 s) and are always provoked by changes in head position relative to gravity, such as lying down, rising from a supine position, and extending the head to look upward. Rolling over in bed is a common trigger that may help to distinguish BPPV from orthostatic hypotension. The attacks are caused by free-floating otoconia (calcium carbonate crystals) that have been dislodged from the utricular macula and have moved into one of the semicircular canals, usually the posterior canal. When head position changes, gravity causes the otoconia to move within the canal, producing vertigo and nystagmus. With posterior canal BPPV, the nystagmus beats upward and torsionally (the upper poles of the eyes beat toward the affected lower ear). Less commonly, the otoconia enter the horizontal canal, resulting in a horizontal nystagmus when the patient is lying with either

ear down. Superior (also called anterior) canal involvement is rare. BPPV is treated with repositioning maneuvers that use gravity to remove the otoconia from the semicircular canal. For posterior canal BPPV, the Epley maneuver (Fig. 24-1) is the most commonly used procedure. For

Step 1 Step 2 Step 3 Step 4 Step 5 Nose is pointed 45° PART 2 Cardinal Manifestations and Presentation of Diseases FIGURE 24-1 Modified Epley maneuver for treatment of benign paroxysmal positional vertigo of the right (top panels) and left (bottom panels) posterior semicircular canals. Step 1. With the patient seated, turn the head 45 degrees toward the affected ear. Step 2. Keeping the head turned, lower the patient to the head-hanging position and hold for at least 30 s and until nystagmus disappears. Step 3. Without lifting the head, turn it 90 degrees toward the other side. Hold for another 30 s. Step 4. Rotate the patient onto their side while turning the head another 90 degrees, so that the nose is pointed down 45 degrees. Hold again for 30 s. Step 5. Have the patient sit up on the side of the table. After a brief rest, the maneuver should be repeated to confirm successful treatment. (Reproduced with permission from Chicago Dizziness and Hearing (CDH). Figure adapted from <http://www.dizziness-and-balance.com/disorders/bppv/movies/Epley-480x640.avi>.)

more refractory cases of BPPV, patients can be taught a variant of this maneuver that they can perform alone at home. A demonstration of the Epley maneuver is available online (<http://www.dizziness-and-balance.com/disorders/bppv/bppv.html>).

■ ■ VESTIBULAR MIGRAINE Vestibular migraine is a common yet underdiagnosed cause of episodic vertigo. Vertigo sometimes precedes a typical migraine headache but more often occurs without headache or with only a mild headache. Some patients who have had frequent migraine headaches in the past present later in life with vestibular migraine as the predominant problem. In vestibular migraine, the duration of vertigo may be from minutes to hours, and some migraineurs also experience more prolonged periods of disequilibrium (lasting days to weeks). Motion sensitivity and sensitivity to visual motion (e.g., movies) are common. Even in the absence of headache, other migraine features may be present, such as photophobia, phonophobia, or a visual aura. Although data from controlled studies are generally lacking, vestibular migraine typically is treated with medications that are used for prophylaxis of migraine headaches (Chap. 441). Antiemetics may be helpful to relieve symptoms at the time of an attack.

■ ■ MÉNIÈRE'S DISEASE Attacks of Ménière's disease consist of vertigo and hearing loss, as well as pain, pressure, and/or fullness in the affected ear. Low-frequency hearing loss and aural symptoms are key features that distinguish Ménière's disease from other peripheral vestibulopathies and from vestibular migraine. Audiometry at the time of an attack shows a characteristic asymmetric low-frequency hearing loss; hearing commonly improves between attacks, although permanent hearing loss may eventually occur. Ménière's disease is associated with excess endolymph fluid in the inner ear; hence the term endolymphatic hydrops. The exact pathophysiological mechanism, however, remains unclear. Patients suspected of having Ménière's disease should be referred to an otolaryngologist for further evaluation. Diuretics and sodium restriction are typically the initial treatments. If attacks persist, injections of glucocorticoids or gentamicin into the middle ear may be considered. Nonablative surgical options include decompression and shunting of

Nose is pointed 45° the endolymphatic sac. Full ablative procedures (vestibular nerve section, labyrinthectomy) are seldom required. ■ ■ VESTIBULAR SCHWANNOMA Vestibular schwannomas (sometimes termed acoustic neuromas) and other tumors at the cerebellopontine angle cause slowly progressive unilateral sensorineural hearing loss and vestibular hypofunction. These patients typically do not have vertigo because the gradual vestibular deficit is compensated centrally as it develops. The diagnosis often is not made until there is sufficient hearing loss to be noticed. The

vestibular examination will show a deficient response to the head impulse test when the head is rotated toward the affected side, but nystagmus will not be prominent. As noted above, patients with unexplained unilateral sensorineural hearing loss or vestibular hypofunction require MRI of the internal auditory canals to look for a schwannoma. ■ ■

BILATERAL VESTIBULAR HYPOFUNCTION

Patients with bilateral loss of vestibular function also typically do not have vertigo, because vestibular function is lost on both sides simultaneously, and there is no asymmetry of vestibular input. Symptoms include loss of balance, particularly in the dark, where vestibular input is most critical, and oscillopsia during head movement, such as while walking or riding in a car. Bilateral vestibular hypofunction may be (1) idiopathic and progressive, (2) part of a neurodegenerative disorder, or (3) iatrogenic due to medication ototoxicity (most commonly gentamicin or other aminoglycoside antibiotics). Other causes include bilateral vestibular schwannomas (neurofibromatosis type 2), autoimmune disease, superficial siderosis, and meningeal-based infection or tumor. It also may occur in patients with peripheral polyneuropathy; in these patients, both vestibular loss and impaired proprioception may contribute to poor balance. Finally, unilateral processes such as vestibular neuritis and Ménière's disease may involve both ears sequentially, resulting in bilateral vestibulopathy. Examination findings include diminished dynamic visual acuity (see above) due to loss of stable vision when the head is moving, abnormal head impulse responses in both directions, and a Romberg sign. Responses to caloric testing are reduced. Patients with bilateral vestibular hypofunction should be referred for vestibular rehabilitation

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