

Symptoms that accompany vertigo may be helpful in distinguishing peripheral vestibular lesions from central causes. Unilateral hearing loss and other aural symptoms (ear pain, pressure, fullness) 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 important in the evaluation of these patients. Particular focus should be given to assessment of eye movements, vestibular function, and hearing. 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 indicates central pathology, often involving the cerebellum. 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 primary position nystagmus is 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) can aid in the detection of peripheral vestibular nystagmus, because they reduce the patient's ability to use visual fixation to suppress nystagmus. **Table 28-1** outlines key findings that help distinguish peripheral from central causes of vertigo.

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 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 vestibular hypofunction (catch-up saccades after rotations in both directions).

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 upbeating-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* 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. Electronystagmography or videonystagmography includes recordings of spontaneous nystagmus (if present) and measurement of positional nystagmus. Caloric testing assesses the responses of the two horizontal semicircular canals. The test battery often includes recording of saccades and pursuit to assess 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 magnetic resonance imaging (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 make the symptoms worse and prolong recovery. The diagnostic and specific treatment approaches for the most commonly encountered vestibular disorders are discussed below.

#### 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.

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 28-1). 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.

Most patients with vestibular neuritis recover spontaneously, but glucocorticoids can improve outcome if administered within 3 days of symptom onset. 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.

**TABLE 28-1** 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 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.