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 therapy. Vestibular suppressant medications should not be used, as they will increase the imbalance. Evaluation by a neurologist is important not only to confirm the diagnosis but also to consider any other associated neurologic abnormalities that may clarify the etiology.

CENTRAL VESTIBULAR DISORDERS

Central lesions causing vertigo typically involve vestibular pathways in the brainstem and/or cerebellum. They may be due to discrete lesions, such as from ischemic or hemorrhagic stroke (Chap. 446), demyelination (Chap. 458), or tumors (Chap. 118), or they may be due to neurodegenerative conditions that include the vestibulocerebellum (Chap. 448). Subacute cerebellar degeneration may be due to immune, including paraneoplastic, processes (Chaps. 122 and 450). Table 28-1 outlines important features of the history and examination that help to identify central vestibular disorders. Acute central vertigo is a medical emergency, due to the possibility of life-threatening stroke or hemorrhage. All patients with suspected central vestibular disorders should undergo brain MRI, and the patient should be referred for full neurologic evaluation.

PSYCHOSOMATIC DIZZINESS/VERTIGO

Psychological factors play an important role in chronic dizziness. First, dizziness may be a somatic manifestation of a psychiatric condition such as major depression, anxiety, or panic disorder (Chap. 465e). Second, patients may develop anxiety and autonomic symptoms as a consequence or comorbidity of an independent vestibular disorder. One particular form of this has been termed variously *phobic postural vertigo*, *psychophysiologic vertigo*, or *chronic subjective dizziness*. These patients have a chronic feeling (months or longer) of dizziness and disequilibrium, an increased sensitivity to self-motion and visual motion (e.g., movies), and a particular intensification of symptoms when moving through complex visual environments such as supermarkets (visual vertigo). Although there may be a past history of an acute vestibular disorder (e.g., vestibular neuritis), the neurootologic examination and vestibular testing are normal or indicative of a compensated vestibular deficit, indicating that the ongoing subjective dizziness cannot be explained by a primary vestibular disorder. Anxiety disorders are particularly common in patients with chronic dizziness and contribute substantially to the morbidity. Thus, treatment with antianxiety medications (selective serotonin reuptake inhibitors [SSRIs]) and cognitive-behavioral therapy may be helpful. Vestibular rehabilitation therapy is also sometimes beneficial. Vestibular suppressant medications generally should be avoided. This condition should be suspected when the patient states, "My dizziness is so bad, I'm afraid to leave my house" (agoraphobia).

TREATMENT VERTIGO

Table 28-2 provides a list of commonly used medications for suppression of vertigo. As noted, these medications should be reserved for short-term control of active vertigo, such as during the first few days of acute vestibular neuritis, or for acute attacks of Ménière's disease. They are less helpful for chronic dizziness and, as previously stated, may hinder central compensation. An exception is that benzodiazepines may attenuate psychosomatic dizziness and the associated anxiety, although SSRIs are generally preferable in such patients.

TABLE 28-2 TREATMENT OF VERTIGO

Agent[a]	Dose[b]
Antihistamines	
Meclizine	25–50 mg 3 times daily
Dimenhydrinate	50 mg 1–2 times daily
Promethazine	25 mg 2–3 times daily (also can be given rectally and IM)
Benzodiazepines	
Diazepam	2.5 mg 1–3 times daily
Clonazepam	0.25 mg 1–3 times daily
Anticholinergic	
Scopolamine transdermal[c]	Patch
Physical therapy	
Repositioning maneuvers[d]	
Vestibular rehabilitation	
Other	
Diuretics and/or low-sodium (1000 mg/d) diet[e]	
Antimigrainous drugs[f]	
Methylprednisolone[g]	100 mg daily days 1–3; 80 mg daily days 4–6; 60 mg daily days 7–9; 40 mg daily days 10–12; 20 mg daily days 13–15; 10 mg daily days 16–18, 20, 22
Selective serotonin reuptake inhibitors[h]	

[a]All listed drugs are approved by the U.S. Food and Drug Administration, but most are not approved for the treatment of vertigo. [b]Usual oral (unless otherwise stated) starting dose in adults; a higher maintenance dose can be reached by a gradual increase. [c]For motion sickness only. [d]For benign paroxysmal positional vertigo. [e]For Ménière's disease. [f]For vestibular migraine. [g]For acute vestibular neuritis (started within 3 days of onset). [h]For psychosomatic vertigo.

Vestibular rehabilitation therapy promotes central adaptation processes that compensate for vestibular loss and also may help habituate motion sensitivity and other symptoms of psychosomatic dizziness. The general approach is to use a graded series of exercises that progressively challenge gaze stabilization and balance.

29 Fatigue

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Fatigue is one of the most common symptoms in clinical medicine. It is a prominent manifestation of a number of systemic, neurologic, and psychiatric syndromes, although a precise cause will not be identified in a substantial minority of patients. Fatigue refers to an inherently subjective human experience of physical and mental weariness, sluggishness, and exhaustion. In the context of clinical medicine, fatigue is most typically and practically defined as difficulty initiating or maintaining voluntary mental or physical activity. Nearly everyone who has ever been ill with a self-limited infection has experienced this near-universal symptomatology, and fatigue is usually brought to medical attention only when it is either of unclear cause or the severity is out of proportion with what would be expected for the associated trigger. *Fatigue* should be distinguished from *muscle weakness*, a reduction of neuromuscular power (Chap. 30); most patients complaining of fatigue are not truly weak when direct muscle power is tested. By definition, fatigue is also distinct from *somnolence* and *dyspnea on exertion*, although patients may use the word fatigue to describe those two symptoms. The task facing clinicians when a patient presents with fatigue is to identify an underlying cause if one exists and to develop