Dizziness

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Dizziness is the third most common complaint among outpatients [1]. Only chest pain and fatigue are more common. In 80% of these cases, the dizziness is severe enough to require medical intervention. Dizziness affects over 50% of the elderly population, and is the most common reason for visiting a physician after the age of 75 years (National Strategic Research Plan, NIDCD, 1999). The cause of dizziness in patients over the age of 60 years seen at the author’s Dizziness and Balance Center since 1995 is shown in Fig. 1. This article discusses the management of the most common causes of dizziness from the perspective of acute vertigo (vertigo <3 days); chronic dizziness (dizziness >3 days); and spells of dizziness.

Acute vertigo

Inner ear infections

Case example

A 56-year-old woman has had persistent sense of rotation, nausea, and dysequilibrium during the last 2 days. She has no hearing loss, tinnitus, or ear fullness. On examination, the patient has right-beating nystagmus, which markedly decreases when she fixates on a target. She is bothered by the oscillopsia (illusion of visual motion) from the right-beating nystagmus, and prefers to keep here eyes closed during examination. Her tympanic membranes are normal bilaterally. She can walk, has a negative Romberg’s test, but cannot stand with open or closed eyes during a tandem Romberg’s test. When she moves her head quickly to the left (head-thrust test) a decreased vestibular-ocular reflex (VOR) is detected; VOR to the right is normal. When 5 mL of ice water is inserted into the left ear, nystagmus does

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not change. When ice water is inserted into the right ear, low amplitude, left-beating nystagmus develops. MRI of the head is normal.

The patient’s history and findings are consistent with left-sided vestibular neuritis. She was given promethazine (Phenergan) suppositories for a few days and treated 10 days with oral prednisone. After 1 week she felt well enough to start vestibular enhancement exercises, which continued for 2 months.

**Pathophysiology**

Patients with acute, unilateral vestibular loss present with intense vertigo, nausea, and dysequilibrium, which persists for days. Within a few days these symptoms begin to resolve and the patient is left with a dynamic deficit in which vertigo and dysequilibrium are induced by rapid head movements. These dynamic deficits can last for weeks to months until central compensation occurs. The vestibular nerve is unique among the other 11 cranial nerves in that the neurons in this nerve on each side have a spontaneous firing rate of 100 spikes/s with the head still. With sudden loss of input from one side, there is a strong bias into the brainstem from the intact side (Fig. 2). This large bias in neural activity causes nystagmus. The direction of nystagmus is labeled according to the quick phase, but the vestibular deficit is actually driving the slow phase of the nystagmus. The intensity of peripheral vestibular nystagmus is increased when fixation is blocked. Nausea and vertigo also occur because of this unequal vestibular input into the brainstem. There are a number of causes for acute vertigo (Table 1). These causes can be divided into those that preserve hearing and those that impair hearing.
Fig. 2. (A) The membranous labyrinth consists of three semicircular canals (SCCs) labeled the anterior, posterior, and horizontal that lie 90 degrees to each other; and two otolith structures, the utricle and saccule. Within each SCC is an area of hair cells that protrude their processes into a gelatinous matrix called the cupula. (B) Central connections of the horizontal and anterior semicircular canals mediating the vestibular-ocular reflex. Primary afferents of the horizontal semicircular canal (HSC) project to the lateral vestibular nucleus (LVN) and medial vestibular nucleus (MVN). Neurons in MVN-LVN project across the midline to terminate in the 6th nerve nucleus (VI). There are two types of neurons in the 6th nerve nucleus: abducens neurons that project to the lateral rectus (LR) muscle; and interneurons that cross the midline and travel up the medial longitudinal fasciculus (MLF) to terminate the subnucleus of 3rd nerve nucleus (III) that innervates the medial rectus (MR). Primary afferents of the anterior semicircular canal (ASC) project to the superior vestibular nucleus (SVN), which in turn travel up the brachium conjunctivum (BC) and ventral tegmental tract (VTT) to terminate in subnuclei of the 3rd nerve nucleus (III) that innervates the superior rectus (SR) and inferior oblique (IO) muscles.
Vestibular neuritis. Vestibular neuritis is preceded by a common cold 50% of the time. The prevalence of vestibular neuritis peaks at 40 to 50 years of age [2,3]. Vestibular neuritis behaves similar to Bell’s palsy and is thought to represent a reactivated dormant herpes infection in the Scarpa’s ganglia within the vestibular nerve [4]. Vestibular neuritis primarily affects the superior division of the vestibular nerve, which innervates the anterior and horizontal semicircular canals (SCCs) (see Fig. 2) [5]. Vestibular neuritis is diagnosed primarily based on the clinical presentation.

Ramsay Hunt syndrome. Ramsay Hunt syndrome is caused by varicella zoster and is a variant of vestibular neuritis with involvement of cranial nerves VII and VIII. It causes facial paresis, tinnitus, hearing loss, and a vestibular defect [6]. It can also involve cranial nerves V, IX, and X. The incidence is 20 cases per 1,000,000 population per year.

Labyrinthitis. The labyrinth has several responses to infection and may be classified as serous (viral or bacterial); suppurative (bacterial); and chronic. There are several routes of entry of organisms into the labyrinth. Viral infection presumably invades the membranous labyrinth by a hematogenous route. Bacterial infection gains access to the perilymphatic space and produces a purulent inner ear infection. The route of bacterial spread may be from the middle ear through a bony fistula in the otic capsule or, more commonly, from the cerebrospinal fluid (meningitis) through the cochlear aqueduct or internal auditory canal. Acute or chronic ear infections can also cause labyrinthitis as a result of toxins and noxious enzymes entering the inner ear [7]. Signs and symptoms of otologic disease or meningitis support the diagnosis. Acute labyrinthitis usually presents with acute severe vertigo; hearing loss (sudden or progressive); nausea; vomiting; and fever. Patients with bacterial labyrinthitis are seriously ill and have severe auditory and vestibular impairment when compared with patients with serous labyrinthitis. Audiometry and vestibular tests demonstrate hypofunction of one or both labyrinths.

Management

Management of acute vertigo is summarized in Table 2. The patient should be admitted to the hospital only if extreme dehydration is present from vomiting or if a central disorder is suspected. Blood work should be obtained to rule out otic syphilis and vasculitis. A caloric test should be obtained 1 week after onset to document the extent of vestibular defect. Viral cultures are not necessary because they do not alter treatment.

A variety of medications are useful to treat vestibular neuritis in the acute stage (Table 3). Intramuscular promethazine (25 to 50 mg) can be used in the office at the onset of severe vertigo, and the patient can be sent home for 3 days of bed rest with promethazine suppositories to be taken as needed. This medication causes sedation and reduces nausea. Ondansetron (Zofran) may
also be appropriate for patients with severe vertigo and nausea, but currently this is only approved for chemotherapy-induced nausea [8]. In patients with vestibular neuritis, prednisone during the first 10 days of the attack may shorten the course of the illness [9,10]. The patient should be referred promptly for vestibular rehabilitation. Certain drugs including antianxiety, antihistamine, anticholinergic, and phenothiazines impair vestibular rehabilitation; these drugs should be stopped within a few days after onset of symptoms.

Patients with Ramsay Hunt syndrome are treated similarly to patients with vestibular neuritis, except acyclovir should be added during the first 10 days (see Table 3). These two drugs should be used promptly because reduced facial nerve degeneration and hearing loss only occurs if treatment is initiated within 3 days compared with more than 7 days after onset [11].

In patients with labyrinthitis, the underlying infection in either the middle ear, mastoid, or the cerebrospinal fluid should be treated [12,13].

**Brainstem infarct**

**Case example**

A 66-year-old man developed acute hearing loss and tinnitus on the right side, severe vertigo, nausea, and unsteadiness. On examination, he had a right-sided facial weakness, he was deaf on the right side, had a left-beat nystagmus in primary gaze and gaze to the right that changed to right-beat nystagmus on...

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Etiology of acute vertigo</th>
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<tbody>
<tr>
<td>Hearing Unchanged</td>
<td>Hearing Decreased</td>
</tr>
<tr>
<td>Vestibular neuritis</td>
<td>Ramsay Hunt syndrome labyrinthitis</td>
</tr>
<tr>
<td>Brainstem infarct</td>
<td>Brainstem infarct</td>
</tr>
<tr>
<td>Posterior inferior cerebellar artery or anterior vestibular artery</td>
<td>Anterior inferior cerebellar artery or labyrinthine artery</td>
</tr>
<tr>
<td>Vestibular schwannoma</td>
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<table>
<thead>
<tr>
<th>Table 2</th>
<th>Management of Acute vertigo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Days 1 to 3</td>
<td>After day 3</td>
</tr>
<tr>
<td>Administer medications (Table 3)</td>
<td>Stop vestibular suppressants</td>
</tr>
<tr>
<td>Prescribe bed rest (hospitalize if patient is dehydrated or suspect central defect)</td>
<td>Refer for vestibular adaptation exercises (Table 6)</td>
</tr>
<tr>
<td>If a central defect is suspected, obtain CT or MRI of head</td>
<td>Audiogram (obtain immediately if Méniere’s disease is suspected)</td>
</tr>
<tr>
<td>Electronystagmography</td>
<td>MRI with 8th nerve cuts to rule out vestibular schwannoma</td>
</tr>
<tr>
<td>Blood work (rheumatoid factor, sedimentation factor, antinuclear antibody, fluorescent treponemal antibody absorption)</td>
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</table>
right gaze. He was unable to walk because of imbalance and kept falling to the right side. He had a history of hypertension and had a myocardial infarction 5 years ago.

The examination contains deficits both in the inner ear and cranial nerve 8th (hearing loss, tinnitus) and brainstem (facial weakness, direction-changing nystagmus, severe imbalance to the point that he could not walk). A CT of the head did not reveal any abnormalities, but the patient was admitted because of the suspicion of a brainstem stroke. Because there was no blood on the CT scan, he was placed on intravenous heparin. An MRI of the head done on the next day revealed a pontine infarct on the right side. MR angiography did not reveal any significant stenosis. Because of the history of myocardial infarction, he had a Holter and transesophageal echocardiogram. The echocardiogram revealed dyskinesia in the left ventricle and mural thrombi. He was discharged on warfarin (Coumadin) and received vestibular rehabilitation.

**Pathophysiology**

Vertigo may occur from infarcts in posterior fossa structures that contain vestibular pathways. There are two common presentations based on the brainstem circulation.

**Anterior inferior cerebellar artery infarct.** The anterior inferior cerebellar artery perfuses the lateral cerebellum (cerebellar branch); the dorsolateral pons (pontine branch); and the labyrinth (labyrinth artery). The labyrinth artery in turns divides into two terminal vessels: the anterior vestibular artery, which perfuses the utricle, anterior SCC, and horizontal SCC; and the common cochlear artery, which perfuses the cochlea, saccule, and posterior

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### Table 3
**Medications for acute vertigo**

<table>
<thead>
<tr>
<th>Major action</th>
<th>Drug</th>
<th>Indications</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antianxiety</td>
<td>Alprazolam (Xanax)</td>
<td>Acute anxiety</td>
<td>0.25 mg prn</td>
</tr>
<tr>
<td>Antihistamine/</td>
<td>Meclizine (Antivert)</td>
<td>Acute vestibular neuritis,</td>
<td>25–50 mg q6h × 3 d</td>
</tr>
<tr>
<td>AntichoLINergic</td>
<td></td>
<td>labyrinthitis, Ramsay Hunt syndrome</td>
<td></td>
</tr>
<tr>
<td>Anti-inflammation</td>
<td>Prednisone</td>
<td>Acute vestibular neuritis,</td>
<td>60 mg q d, then taper over 10 d</td>
</tr>
<tr>
<td></td>
<td></td>
<td>labyrinthitis, Ramsay Hunt syndrome</td>
<td></td>
</tr>
<tr>
<td>Antiviral</td>
<td>Acyclovir (Zovirax)</td>
<td>Ramsay Hunt syndrome</td>
<td>400 mg 5×/d × 10 d</td>
</tr>
<tr>
<td>Phenothiazine</td>
<td>Promethazine (Phenergan)</td>
<td>Acute vestibular neuritis,</td>
<td>25 mg po, im or supp q12h</td>
</tr>
<tr>
<td></td>
<td>Prochlorperazine</td>
<td>labyrinthitis, Ramsay Hunt syndrome, severe nausea from central vertigo</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Compazine)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serotonin agonist</td>
<td>Ondansetron (Zofran)</td>
<td>Severe nausea from central vertigo</td>
<td>4 mg q8h for 3 d</td>
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</tbody>
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SCC. There are several types of anterior inferior cerebellar artery syndromes that result in acute vertigo. Infarct of the anterior vestibular artery presents with just peripheral symptoms (vertigo, nausea, vomiting, and imbalance). Infarct of the common cochlear artery or labyrinthine artery also results in these peripheral symptoms along with hearing loss and tinnitus. Infarct of the pontine branch results in central signs (dysarthria, peripheral facial palsy, trigeminal sensory loss, Horner’s syndrome, dysmetria, contralateral temperature, and pain sensory loss) [14]. The labyrinthine artery can originate directly from the basilar artery in approximately 15% of patients.

Posterior inferior cerebellar artery infarct. The posterior inferior cerebellar artery perfuses the posterior inferior cerebellum (cerebellar branch) and the dorsolateral medulla. Vertigo can occur from infarcts in the lateral medulla (Wallenberg’s syndrome) because of involvement of the vestibular nucleus. Characteristic signs include crossed sensory signs, ipsilateral lateropulsion, ataxia, and Horner’s sign. Nystagmus may be pure torsion or mixed torsion and horizontal. When the nystagmus contains a horizontal component, it reverses direction on gaze toward the lesion side, unlike nystagmus from peripheral vestibular lesions.

Management

MR arteriography can be performed to assess posterior circulation and transcranial Doppler may detect decreased flow in the basilar artery. Treatment includes reduction of risk factors for cerebrovascular disease and antiplatelet therapy. Warfarin is used when there is significant vertebral-basilar artery stenosis [15]. During acute vertigo, patients can be treated with ondansetron or one of the phenothiazines (see Table 3) [8]. Vestibular enhancement exercises should be started once spontaneous nausea and vertigo decrease, although the recovery process is much more prolonged compared with patients with peripheral vestibular defects. Central compensation from vestibular exercises may not occur if the infarct involves the vestibular portion of the cerebellum [16].

Vestibular schwannoma

Case example

A 66-year-old man was seen for acute vertigo and nausea. He had a 5-year history of tinnitus and progressive hearing loss in the left ear. On examination he had a mild right-beat nystagmus that increased when fixation was blocked. He had a moderate sensorineural hearing loss on the left side. He could not stand in tandem stance with eyes open or closed. Caloric test revealed severe vestibular loss on the left side. Because of the vestibular loss, an MRI of the head was ordered to rule out a vestibular schwannoma. This diagnosis was especially of concern because of the unilateral tinnitus and progressive hearing loss. No tumor was seen on the unenhanced scan, but with gadolinium, an
intracanalicular mass was found on the left side consistent with a vestibular schwannoma (Fig. 3). The tumor was removed and the patient was sent for vestibular rehabilitation.

Pathophysiology

Acoustic neuroma is a misnomer because the tumor actually is composed of Schwann cells of the vestibular nerve. This tumor should be called vestibular schwannoma. The most common presentations of a vestibular schwannoma are progressive or sudden sensorineural hearing loss, but vertigo may be the presenting symptom in up to 38% of patients [17,18]. Patients with documented unilateral vestibular loss or unexplained, unilateral hearing loss or tinnitus should have a gadolinium-enhanced MRI with VIIIth nerve cuts. This type of scan reaches 100% sensitivity no matter what size the tumor and is the gold standard for detecting this tumor. An unenhanced MRI can miss a vestibular schwannoma.

Management

If a vestibular schwannoma is detected, patients should be imaged every 6 to 24 months depending on the growth of the tumor. Studies have shown no growth in 36% to 71% of tumors over the course of 3.5 years [17,19]. When tumor growth is detected on MRI of the head, there are two options. Surgical removal by a translabyrinthine approach is considered by many to be the treatment of choice for patients with nonserviceable hearing. The translabyrinthine approach is popular because of a low complication rate and the potential of total tumor removal [20]. In addition, it is safe and effective even with the largest of tumors. Facial neuropathy and cerebrospinal fluid leak are the two most frequent complications [21]. For patients with serviceable hearing, microscopic dissection of the tumor from

Fig. 3. Small acoustic neuroma revealed in an enhanced MRI of the head. The figure on the left shows a gadolinium-enhanced MRI scan. The intracanalicular portion of the VIIIth nerve on the right side enhances (arrow) consistent with an acoustic neuroma. The figure on the right shows the same section in an axial T2 MRI (no enhancement). The scan without enhancement does not show the tumor.
a suboccipital approach, and stereotactic delivered radiation treatments using gamma knife or fractionated radiotherapy are both useful to stop growth of the tumor [22]. Both approaches preserve hearing. There are no randomized trials in which microscopic surgery has been compared with radiation treatment. Following translabyrinthine or intracranial surgery, acute vertigo, nausea, and imbalance usually occur because there is usually functioning nerve present before surgery. These patients should be treated with vestibular rehabilitation. A randomized controlled trial has shown that vestibular adaptation exercises improve postural stability and diminished perception of dysequilibrium following vestibular schwannoma resection [23].

**Chronic dizziness**

**Vestibular hypofunction**

**Case example**

A 65-year-old man had developed chronic unsteadiness on his feet and oscillopsia during head movement (false illusion of motion in visual environment). The imbalance increases when he stands in the dark, walks on uneven surfaces, bends over, or makes quick turns. Physical examination reveals no spontaneous nystagmus and normal visual tracking eye movements. Bedside tests of his VOR find decreased dynamic visual acuity (visual acuity during head movements) and decreased VOR during head thrusts both to the left and right. He has a negative Romberg’s test, but could not stand in tandem with eyes open or closed. He is very cautious while walking for fear of falling. No nystagmus was induced during the caloric test.

The patient’s history and findings are consistent with bilateral loss of vestibular function. He was referred for vestibular physical therapy, in which he was taught exercises to compensate for loss of vestibular function. He went to physical therapy six times over the course of 3 months and continued to do the exercises on his own at home. Within a few months his dynamic visual acuity improved sufficiently for him to drive and balance improved where he could play golf again. Repeat caloric testing still showed an absent vestibular response. The recovery was primarily mediated by central compensation.

**Pathophysiology**

Table 4 lists the most common causes of chronic vestibular loss. There is an associated hearing loss in approximately half the causes of vestibular defects. The most common cause of bilateral loss is idiopathic (most likely degenerative). About 33% of the author’s cases of bilateral loss are caused by ototoxicity (usually gentamicin). The incidence of ototoxicity from gentamicin increases to nearly 20% in patients with renal failure. Gentamicin is selectively taken up by the hair cells in the cupula of the inner ear. This drug
consistently causes ototoxicity in high doses, although the condition can also occur idiosyncratically in normal doses in certain patients. Because gentamicin is much more toxic to the hair cells in the vestibular portion of the inner ear than the hearing portion, a loss of hearing should not be a criterion for diagnosing ototoxicity. Vertigo is infrequent in these patients because the vestibular loss is bilateral.

Management

Vestibular rehabilitation is extremely important in recovery of the VOR and vestibular-spinal reflexes in patients with vestibular hypofunction. To facilitate vestibular adaptation, the patient is encouraged to move their head while viewing a stimulus (Table 5). In addition, they are given exercises to improve their postural control at first while standing still and then with head and body movement through space. Patients usually improve faster and more completely if these exercises are coordinated by a physical therapist trained in vestibular rehabilitation. The treatment of unilateral vestibular loss is based on research in animal studies [24,25]. The chronic problem after a vestibular lesion is a dynamic deficit in which vertigo and unsteadiness occur during head movements. This can only be repaired by vestibular adaptation. Vestibular adaptation occurs when there is a mismatch between head motion sensed by the vestibular system and the visual system. To facilitate vestibular adaptation, the patient is encouraged to move the head while viewing a still target. Eventually, these exercises should be done with the target moving in the opposite direction to the head. In addition, postural control is improved by having the patient stand with feet together, then in tandem, and then with the head moving. Similarly, the patient is encouraged to walk normally, then tandem, and finally with the head moving back-and-forth. Most recovery occurs from exercises that facilitate the substitution of other ocular motor systems and of somatosensory and visual cues to facilitate the recovery of postural stability. Plateau in recovery should occur within 3 to 6 months. Several controlled studies have demonstrated that supervised exercises are significantly more effective in improving balance and perceived “dizziness” in patients with unilateral and bilateral vestibular deficits than giving patients an instruction sheet of exercises to perform on their own at home [26,27]. Controlled trials have demonstrated significant improvement in balance and reduced falls in patients with bilateral vestibular loss following vestibular rehabilitation [28].

Table 4
Etiology of chronic vestibular loss

<table>
<thead>
<tr>
<th>Defect</th>
<th>Etiology</th>
<th>Hearing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral vestibular</td>
<td>(See Table 1)</td>
<td>(See Table 1)</td>
</tr>
<tr>
<td>loss</td>
<td>Idiopathic or hereditary</td>
<td>Usually impaired</td>
</tr>
<tr>
<td>Sequential vestibular</td>
<td>Neuritis</td>
<td>Usually spared</td>
</tr>
<tr>
<td>loss</td>
<td>Ototoxicity</td>
<td>Usually spared</td>
</tr>
</tbody>
</table>
Table 5
Vestibular enhancement exercises for patients with loss of vestibular function

Exercises to enhance the vestibular-ocular reflex

Tape a business card on the wall in front of you so that you can read it. Move your head back and forth sideways keeping the words in focus. Move your head faster but keep the words in focus. Continue to do this for 1–2 min without stopping several times a day. Repeat the exercise moving your head up and down. After this exercise can be easily accomplished try to read portions of the newspaper or pages in a book while oscillating the head for 1–2 min several times a day.

Exercises to improve static balance
1. Stand with your feet placed as close together as possible with both hands touching the wall to help maintain balance. Take your hand off the wall for longer and longer periods of time while maintaining your balance.
2. Stand with feet placed shoulder width apart with eyes open, looking straight ahead at a target (an X) on the wall. Slowly narrow your base of support by moving your feet closer together. Start with your feet apart, then feet together, then one foot slightly forward but still next to the other, and then heel to toe (one foot directly in front of the other). You should change your foot position one inch at a time. Hold each position for 15 seconds and then move on to the next most difficult exercise. This exercise can be performed with arms outstretched, with arms close to the body, or with arms folded across the chest. The exercise can also be repeated with eyes closed, at first intermittently and then continuously all the while making a special effort mentally to visualize the surroundings.
3. Practices standing on a cushioned surface. Progressively more difficult tasks might be hard floor (linoleum, wood), thin carpet, shag carpet, thin pillow, or sofa cushion. Graded density foam can also be purchased.
4. Stand with your feet as close together as possible. Then turn your head to the right and left horizontally while looking straight ahead at a target (an X) on the wall for 1 min without stopping. The size of your head movement should be approximately ±30 degrees. As you improve, you can move your head more rapidly (always seeing clearly) and can move your feet closer together to make the exercise more difficult.

Exercises to improve dynamic postural stability
1. Practices walking with a more narrow base of support. You can do this first touching the wall for support and then gradually touching only intermittently and then not at all.
2. Walk close to a wall, turning your head to the right and to the left while walking. You should try to focus on different objects while walking. As you improve, try to turn your head more often and faster, always seeing clearly. Try the same exercise moving your head up and down.
3. Practice turning around while you walk, at first making a large circle but gradually making smaller and smaller turns. Be sure to practice turning in both directions.
4. Practice standing and then walking on ramps, either with a firm surface or with more cushioned surface.
5. Play catch: at first without much movement but then being required to move in order to catch the ball. Try walking and tossing a ball while you walk.
6. Practice walking through an obstacle course. The course should include different surfaces, turns, small and large steps, and curbs or stairs. You can also walk this course while carrying a heavy bag or while playing catch.
7. Out in the community, practice walking in a mall before it is open and while it is quiet; practice walking in the mall while walking in the same direction as the flow of traffic; walk against the flow of traffic. Try walking up and down the aisles of a grocery store without a cart. Try walking in a grocery store while turning your head from side to side.
Disuse dysequilibrium and fear of fall

Case example

A 73-year-old woman fell from a 3-ft ladder 9 months ago. Since then, she has had chronic dizziness. She loses her balance occasionally. Before she fell, she walked 3 miles per day, but is now afraid to walk. On examination she has no significant orthopedic or neurologic problems. Her vestibular examination is normal. She cannot walk tandem and shows fear of fall when standing with eyes closed. Her Tinetti Fall Risk assessment was 27, which places her at moderate risk for fall. This assessment is excellent for patients at risk for fall [29].

The history and examination are consistent with disuse dysequilibrium and fear of fall. The patient was placed on a daily home exercise program coordinated by a physical therapist with a specialty in geriatrics. She saw the therapist in clinic once each week for 4 weeks. During each clinic visit with the physical therapist, her balance was assessed and her exercises were increased in difficulty. The exercises included progressive static balance with eyes opened and closed, progressive gait exercises with and without head movements, and eventually a walking program that increased from 1 to 3 miles per day. At the end of the fourth week, her Tinetti Fall Risk assessment improved to normal range (score, 35), and she was discharged from the clinic.

Pathophysiology

In elderly individuals, there is progressive decline in muscle bulk, joint range of motion, and reflex time with age. Increased exercise can decrease the rate of this decline. Lack of exercise in the elderly can lead to disuse dysequilibrium [30]. The patient may stop walking and exercising because of recent surgery, fatigue, or chronic illness or for some other reason. Fear of fall can occur as a result of disuse dysequilibrium [31]. Fear of fall can also exacerbate disuse dysequilibrium by reducing the patient’s willingness to participate in a home-exercise program.

Management

A daily home exercise program that increases endurance, balance, and lower extremity strength frequently resolves the problem [32]. Success depends on compliance by the patient and a supportive role by the family or friends.

Leukoaraiosis

Case example

The patient is a 60-year-old woman who complains of chronic imbalance. Her legs feel heavy or like lead. She has had several falls backward with injury. She has diabetes mellitus, hypertension, and elevated cholesterol. On examination she had a masked faces; mild rigidity; gait apraxia; and a poor
righting reflex (would fall like a “log” when given a mild push backward). She did not have tremor or cogwheel rigidity. She did not improve on a trial of carbidopa-levodopa (Sinemet). An MRI of her head revealed leukoaraiosis (Fig. 4). She was referred to physical therapy for gait and balance exercises.

Pathophysiology

Patients with significant ischemic white matter diseases (leukoaraiosis) have a number of symptoms including balance disorders. In severe cases, these patients develop gait apraxia, initiation defects, and severe retrogression. These gait and balance disorders overlap with normal pressure hydrocephalus (symptoms of cognitive decline, gait apraxia, and urinary incontinence). In the retropulsion test, the patient stands with feet slightly spread apart and is instructed to just take one step backward if they are pulled backward by the hips by a mild force. In a positive test the patient must take three or more steps backward or falls backward like a log. This test is positive in patients with basal ganglia disorders (progressive supranuclear palsy or Parkinson’s disease) and disorders that disrupt frontal lobe–basal ganglia projections (normal pressure hydrocephalus or leukoaraiosis). The cause of leukoaraiosis is believed to be significant small vessel disease. It occurs most frequently in patients with diabetes mellitus and hypertension.

Management

Because patients with leukoaraiosis have features that overlap with Parkinson’s syndrome, a 2- to 4-week trial of low-dose carbidopa-levodopa
should be tried to improve gait and balance. There are no trials to date to
determine if improved blood flow to the brain stabilizes or improves func-
tion, but pre-existing disease (hypertension, diabetes mellitus, and elevated
cholesterol) should be controlled. These patients are at high risk for falls,
especially backward. They should be referred to physical therapy and be
given a fall-risk assessment. A home health evaluation may also be neces-
sary to reduce the risk for falls at home. Some balance improvement may
occur with physical therapy, but many patients have to be given an aid (cane
or walker) when ambulating inside or outside the house.

**Psychogenic**

**Case example**

A 15-year-old girl was referred by her mother for inability to walk for 2
weeks. She could only take a few steps before she had to sit down or her
knees would buckle. She started attending a new school 3 weeks before her
illness. She was doing very well until the dizziness started. There is an older
daughter who is excelling in the same school. A head CT and audiograms were
normal. She had a positive tilt table to isoproterenol suggesting possible
orthostatic hypotension and was placed on medication and salt tablets. This
may have initially helped but for only a week. Her physical examination was
normal except for her stance and gait. There was significant sway at the hips
with eyes open and closed, but she did not fall. While walking, she had sudden
buckling of the knees but was able to still walk. There was much side-to-side
swaying and waste of muscular energy.

This patient had a conversion disorder. She had a deficit that suggested
a neurologic disorder and yet there was no disorder found. She had a
psychogenic stance and gait disorder with several of the features described in
Table 6. Her symptoms began temporally with the stress of starting
a new school, the same school that was attended by her sister who was an
over achiever. There was no evidence for external economic gain as one
expects for malingering. She did not have a history of assuming a sick role
motivated by psychologic need as one expects for a factitious disorder. As
in several cases of conversion disorder, this case prompted extensive
evaluations and an organic diagnosis (orthostatic hypotension) that proved

<table>
<thead>
<tr>
<th>Clinical finding</th>
<th>% Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moment to moment fluctuations in the level of impairment</td>
<td>51</td>
</tr>
<tr>
<td>Excessive slowness or hesitation</td>
<td>51</td>
</tr>
<tr>
<td>Exaggerated sway on Romberg vs test, often improved by distraction</td>
<td>32</td>
</tr>
<tr>
<td>Uneconomical postures with waste of muscular energy</td>
<td>30</td>
</tr>
<tr>
<td>Extreme caution with restricted steps (walking on ice)</td>
<td>30</td>
</tr>
<tr>
<td>Sudden buckling of the knees, typically without falling</td>
<td>27</td>
</tr>
</tbody>
</table>
later to be wrong. The tilt table especially with isoproterenol has a number of false-positive outcomes. This diagnosis was not discussed with the child. It was discussed with the mother, but in a way that she could “save face.” The social problems with starting a new school attended by an over-achieving sister were discussed. School counseling was recommended. Her gait disorder slowly resolved after she was placed in a school that was not attended by her sister.

Pathophysiology
Six features have been identified on examination for the diagnosis of psychogenic balance and gait disorders based on review of videotapes from 37 patients with this disorder [33]. Table 6 lists the prevalence of each feature.

Management
In 5 years, 47% of the patients with psychogenic gait disorders may have a favorable outcome [34]. Counseling is frequently helpful. Psychogenic dizziness may also occur in patients who are malingering. This is more frequent in patients who have potential monetary gain from the illness. In these cases, counseling may not be of any benefit.

Medications that can cause dizziness or be harmful to the dizzy patient
Several medications may cause subjective symptoms of “dizziness,” especially in those patients over the age of 65 years [35,36]. Table 7 lists the more common medications along with their primary effects. Certain drugs cause dysequilibrium and lightheadedness. These include anticonvulsants, antidepressants, antihypertensives, anti-inflammatory agents, hypnotics, muscle relaxants, tranquilizers, and chronic use of vestibular suppressants. Sensitization may occur to meclizine and scopolamine after a few days of continuous use, and withdrawal symptoms occur when the medication is discontinued. This may be misinterpreted as recurrence of the disorder itself, so that physicians should be cautious about restarting these medications. It is suggested that meclizine, scopolamine, and other vestibular suppressants only be used for a few days during acute vestibular hypofunction caused by vestibular neuritis and labyrinthitis. These drugs should then be discontinued because they interfere with central compensation within the denervated vestibular nucleus. Patients with brainstem medullary lesions may have nausea lasting for weeks and may require medication for a longer period of time. Certain drugs may cause vestibular ototoxicity and spare hearing, yet lead to dysequilibrium. These include certain aminoglycosides (streptomycin, gentamicin, and tobramycin); furosemide; and ethacrynic acid. The other aminoglycosides affect hearing primarily. Treatment of bilateral vestibular defects should include avoidance of all ototoxins that
Table 7
Drugs that can induce dizziness or be harmful to the dizzy patients

<table>
<thead>
<tr>
<th>Drug</th>
<th>Drugs that can cause dizziness</th>
<th>Drugs that interfere with vestibular compensation</th>
<th>Ototoxic drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiarrythmics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone, quinine,</td>
<td></td>
<td></td>
<td>X (synergistic)</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barbiturates,</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>carbamazepine,</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>phenytoin ethosuximide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antidepressants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>amitriptyline,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imipramine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antihypertensives</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furosemide, ethacrynic acid</td>
<td></td>
<td>X (synergistic)</td>
<td></td>
</tr>
<tr>
<td>a1-Blockers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prazosine, terosine</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b-Blockers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atenolol, propranolol</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium antagonists</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nifedipine, verapamil</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anti-inflammatory drugs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ibuprofen, indomethacin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>X</td>
<td></td>
<td>X (reversible)</td>
</tr>
<tr>
<td>Antibiotics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Streptomycin, gentamicin</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Tobramycin</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Chemotherapeutics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cisplatinum</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Hypnotics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flurazepam, triazolam</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle relaxants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyclobenzaprine,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>orphenidrine</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methocarbamol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tranquilizers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chlordiazepoxide,</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>meprobamate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vestibular suppressants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mecilizine, scopolamine,</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>chloridiazepoxide,</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>diazepam</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

may cause further permanent peripheral vestibular damage (gentamicin, streptomycin, tobramycin, ethacrynic acid, furosemide, quinine, and cisplatin) and avoidance of drugs that may transiently impair balance (sedative, antianxiety, antiepileptics, and antidepressants). Vestibular rehabilitation may be very helpful for these patients.
Spells of dizziness

Spells of dizziness that last for seconds are characteristic of benign paroxysmal positional vertigo (BPPV), central positional vertigo, perilymphatic fistula, superior canal dehiscence, and orthostatic hypotension (Table 8). Spells that last for minutes may be caused by transient ischemic attacks, migraine, and anxiety attacks. Meniere’s disease and hydrops can induce dizzy spells that last for hours to days.

Benign paroxysmal positional vertigo

Case presentation

A 67-year-old woman presents with a history of episodic positional vertigo lasting several seconds. It usually occurs in the morning when she turns over onto her right side or sits up. Occasionally, it occurs when she tilts her head back to look up to a high shelf or to wash her hair. She has stopped going to the dentist and beauty parlor because of these dizzy spells and now only sleeps on her left side. She also complains of imbalance. There has been no change in her hearing, but she has chronic tinnitus in both ears. On examination, there is no spontaneous nystagmus. All bedside VOR tests and visual tracking eye movements are normal. During the Dix-Hallpike test on the right side, however, she develops upbeat and right torsional nystagmus with vertigo a few seconds after her head is tilted back. The induced nystagmus and vertigo last 15 seconds. The Dix-Hallpike test on the left side is normal.

The patient’s history and findings are consistent with a diagnosis of right-sided BPPV. She was treated with the canalith repositioning maneuver. When she was seen 2 days later, she no longer had any positional dizziness or imbalance, and her Dix-Hallpike test on the right side was normal.

Pathophysiology

The most common cause of spells of dizziness in the elderly is BPPV (see Fig. 1) and the incidence increases with age. The diagnosis can often be

<table>
<thead>
<tr>
<th>Duration</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seconds</td>
<td>Benign paroxysmal positional vertigo</td>
</tr>
<tr>
<td></td>
<td>Central positional vertigo</td>
</tr>
<tr>
<td></td>
<td>Perilymphatic fistula</td>
</tr>
<tr>
<td></td>
<td>Superior canal dehiscence</td>
</tr>
<tr>
<td></td>
<td>Orthostatic hypotension</td>
</tr>
<tr>
<td>Minutes</td>
<td>Transient ischemic attacks</td>
</tr>
<tr>
<td></td>
<td>Migraine</td>
</tr>
<tr>
<td></td>
<td>Anxiety attack</td>
</tr>
<tr>
<td>Hours to days</td>
<td>Meniere’s disease and hydrops</td>
</tr>
</tbody>
</table>
made solely on the patient’s history. The key features in this patient’s case were the duration of the spells, the circumstances in which they occurred, and the findings during the Dix-Hallpike test. BPPV can cause dizziness with these same positional changes, but also while lying down or turning over in bed. Nystagmus from BPPV is a jerk-type that consists of slow phases and quick phases. The movement of otoconia in the SCC generates the slow phases. The quick phases are reflexive. Classically, nystagmus direction is based on the direction of the quick phase. For posterior SCC BPPV, the nystagmus is primarily upbeat and torsion (superior poles of each eye torts toward the dependent or inferior ear while lying). The nystagmus associated with BPPV is transient and correlates with the dizziness. BPPV is usually idiopathic, but can also occur after head trauma, vestibular neuritis, and ischemia in the distribution of the vestibular artery. Fig. 5 shows the pathophysiology underlying BPPV. Debris (otoconia) from the utricle floats into the posterior semicircular canal when the patient lies down (see Fig. 5A, B). When the patient sits back up again, the debris remains in the canal (see Fig. 5C). Occasionally, the debris attaches to the cupula of the semicircular canal (cupulolithiasis). Whenever the patient moves their head vertically, debris moves within the canal and stimulates the vestibular sense organ (cupula) to cause vertigo and nystagmus (eg, Dix-Hallpike test) (Fig. 6).

Management

Treatment of BPPV consists of a maneuver that moves the otoconia in the SCC back to the utricle. Once it is in this location it is reabsorbed into the macule of the utricle. Vestibular suppressant drugs do not have a role in the treatment of BPPV unless the patient refuses to do the maneuvers because of excessive vertigo and nausea. In these cases, promethazine or prochlorperazine suppository 30 minutes before the maneuver can be used. Furthermore, there is no medical treatment that can prevent or cure the disease forever (ie, there is no treatment that can prevent the recurrence of the release of particles from the utricle to the SCC). Some surgery has been used for positional vertigo, including section of the nerve that innervates the posterior SCC, or occlusion of the posterior SCC [37–39]. Surgery is not optimal because it results in a permanent deficit. There are two basic bedside maneuvers for BPPV: canalith repositioning treatment (canalith repositioning maneuver or Epley) and the liberatory treatment [40,41]. The author prefers the term canalith repositioning maneuver because it is descriptive. Canalith repositioning maneuver is the treatment the author prefers to use for severe canalithiasis (free-floating otoconia). It is effective in 85% to 95% of patients with one treatment [40,42–45]. Canalith repositioning maneuver can also resolve imbalance from BPPV [46]. During Canalith repositioning maneuver the patient is first moved into the Dix-Hallpike position toward the side of the affected ear and kept down for 20 seconds (Fig. 7). Then, the head is slowly rotated through moderate extension of the neck toward the
unaffected side and kept in the new position for 20 seconds. The patient is then rolled on a side-lying position with the head turned 45 degrees down (toward the floor) and kept there for 20 seconds. Keeping the head deviated toward the unaffected side and the head pitched down, the patient then slowly sits up. To make certain the otoconia stays in the utricle after the treatment, the patient is fitted with a soft collar and told not to bend over, lie back, move their head up or down, or tilt their head to either side for the rest of the day. The traditional follow-up treatment is to have the patient not bend the head back more than 45 degrees for 2 days even during sleep, and

Fig. 5. Mechanism for BPPV. The labyrinth contains two gravity detectors labeled the utricle and saccule. These structures contain a local region of hair cells that protrude their processes into a gelatinous matrix called the macule, which is covered by a surface of calcium carbonate crystals called otoconia. BPPV is caused by free-floating otoconia in the SCC (canalithiasis) or otoconia attached to the cupula (cupulolithiasis), which have become displaced from the macule of the utricle. Otoconia break free from the utricle (A), fall into the posterior SCC when the patient lies down (B), and then stay in the most dependent portion of this SCC when the patient sits up (C). Whenever the patient moves the head backward or forward, the movement of otoconia in the SCC deflects the cupula and causes vertigo.
then not to sleep on the affected ear until follow-up assessment 2 days later. The author has recently found that the outcome is the same regardless how many days the patient stays upright. In 50% of patients, a recurrence of BPPV occurs. The author has recently begun to teach the patient the treatment using a handout (see Fig. 7). They are instructed to do this in the morning and keep their head up the rest of the day. They are not asked to sleep upright at night. If they still develop vertigo when they lie down, they are instructed to repeat the treatment each morning until they no longer have vertigo during the maneuver.

Central positional vertigo

Case presentation

The patient is a 30-year-old woman with 1-year history of severe positional vertigo. Her chief complaint was “when I move my head or body, I feel a spinning sensation. I am feeling like I am rolling, spinning and turning in circles in all directions. The room looks like it is blurry and
bouncy. Nausea sets in as well. After I stop feeling like I am spinning, I feel like I am on a boat.” There was no history of head trauma or headaches. The patient brought in a copy of a normal, unenhanced MRI of the head. The examination was normal except for position testing. During the Dix-Hallpike test with either the left or right ear down, the patient developed severe positional vertigo associated with transient downbeat nystagmus without a torsional component. The nystagmus and vertigo did not resolve after treatment with the canalith repositioning maneuver, which was done twice. Positional downbeat nystagmus with vertigo is often caused by a central problem, especially if it is bilateral and there is no torsional component to the nystagmus. Another MRI of the head was ordered with gadolinium (Fig. 8). It shows enhancement of a small mass in the cerebellar nodulus. The mass was removed and it was found to be a low-grade glioma. The patient’s symptoms did not resolve following surgery.

**Pathophysiology**

Central positional vertigo consists of marked vertigo, nausea, and nystagmus induced by a position change in the head (lying down or sitting up).
It is caused by a lesion near the 4th ventricle often near the cerebellar nodulus. Etiology includes tumors, multiple sclerosis, and strokes. The key features that distinguish BPPV from central positional vertigo are listed in Table 9. In BPPV, the nystagmus is upbeat and torsional; the vertigo and nystagmus take a few seconds to develop after the head is placed in a head-hanging position (latency); the vertigo and nystagmus last 5 to 20 seconds; and if the Dix-Hallpike test is repeated several times, the nystagmus and vertigo are no longer elicited for several minutes to an hour (fatigue). Most of these features differ from the nystagmus and vertigo seen in central positional vertigo (see Table 9).

**Management**

All patients with central positional vertigo need a gadolinium-enhanced MRI of the head. Further work-up depends on the findings of the MRI. If the scan is normal it should be repeated in 6 months. If the scan shows multiple periventricular white matter signal intensities, a demyelinating process, such as multiple sclerosis, should be considered. If a mass is identified, the patient should be referred to neurology or neurosurgery for

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Peripheral (BPPV)</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direction of nystagmus</td>
<td>Upbeat and torsional</td>
<td>Up- or down-beat</td>
</tr>
<tr>
<td>Latency of vertigo and nystagmus</td>
<td>Seconds</td>
<td>No latency</td>
</tr>
<tr>
<td>Duration of vertigo and nystagmus</td>
<td>5–20 s</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Vertigo and nystagmus fatigues</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

*Abbreviation: BPPV, benign paroxysmal positional vertigo.*
further evaluation. Removal of the mass does not stop the symptoms. Because the nystagmus and vertigo do not fatigue or habituate, physical therapy is not effective. Some medications may be used to decrease symptoms including clonazepam (Klonopin) (Table 10). Patients usually learn to move their head slowly when lying down or sitting up from bed.

Perilymphatic fistula

Case presentation

A 33-year-old man experiences severe vertigo, nausea, vomiting, and imbalance for several hours after getting out of bed. He also notes vertigo and oscillopsia when he lifts weights or clears his middle ear with Valsalva’s maneuver. Four months ago he noticed slight dizziness and right ear fullness for several days after scuba diving down to 60 ft. During the Dix-Hallpike test on the left side, the patient developed vertigo and right-beat nystagmus when he lied down, and downbeat nystagmus when he sat up. The same was found for the test on the right side. When the patient blew out through pinched nostril, he developed right-beat nystagmus. Right-beat nystagmus

<table>
<thead>
<tr>
<th>Drug</th>
<th>Major action</th>
<th>Indications</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetazolamidie (Diamox)</td>
<td>Diuretic</td>
<td>Méniere’s disease</td>
<td>250 mg</td>
</tr>
<tr>
<td>Acyclovir (Zovirax)</td>
<td>Antiviral</td>
<td>Acute vestibular neuritis</td>
<td>400 mg 5×/d × 10 d</td>
</tr>
<tr>
<td>Alprazolam (Xanax)</td>
<td>Benzodiazepam</td>
<td>Acute anxiety</td>
<td>0.25 mg prn</td>
</tr>
<tr>
<td>Fludrocortisone (Florinef)</td>
<td>Mineral corticoid</td>
<td>Orthostatic hypotension</td>
<td>0.1–0.6 mg/d</td>
</tr>
<tr>
<td>Klonopin (clonazepam)</td>
<td>Benzodiazepam</td>
<td>Central positional vertigo</td>
<td>0.5–10 mg bid</td>
</tr>
<tr>
<td>Midodrine (proAmatine)</td>
<td>α1-adrenergic stimulant</td>
<td>Orthostatic hypotension</td>
<td>10 mg tid</td>
</tr>
<tr>
<td>Ondansetron (Zofran)</td>
<td>Serotonin agonist</td>
<td>Severe nausea from central vertigo</td>
<td>4 mg q8h for 3 d</td>
</tr>
<tr>
<td>Paroxetine (Paxil)</td>
<td>SSRI (antidepressant)</td>
<td>Chronic anxiety</td>
<td>10–20 mg q AM</td>
</tr>
<tr>
<td>Prednisone</td>
<td>Anti-inflammation</td>
<td>Acute vestibular neuritis</td>
<td>60 mg d, then taper over 10 d</td>
</tr>
<tr>
<td>Promethazine (Phenergan)</td>
<td>Phenothiazine</td>
<td>Méniere’s disease, severe nausea from central vertigo</td>
<td>25 mg PO, IM or supp q12h for &lt;3 d</td>
</tr>
<tr>
<td>Prochlorperazine (Compazine)</td>
<td>Beta blocker</td>
<td>Migraine</td>
<td>At least 80 mg q d split up in several doses</td>
</tr>
<tr>
<td>Propranolol (Inderal)</td>
<td>Gaba Agonist</td>
<td>Migraine</td>
<td>250–500 mg PO bid</td>
</tr>
</tbody>
</table>

Abbreviations: SSRI, selective serotonin reuptake inhibitor; supp, suppository.
also occurred when he relaxed from sucking in through pinched nose. The patient had a perilymphatic fistula in the right ear (hole in round window). This hole was patched and the patient’s symptoms and signs resolved.

**Pathophysiology**

A perilymphatic fistula is a hole between the inner and middle ear caused by barotrauma (scuba diving); a tumor in the middle ear (cholesteatoma); head trauma; or displacement of a prosthetic middle ear bone into the inner ear. Any pressure changes to the inner or middle ear cause flow of fluid between these two compartments and distort the utricle or semicircular canal. Distortion of these end organs frequently causes transient vertigo, nystagmus, or skew deviation. Diagnosis requires middle ear exploration. The oval and round windows are examined for the leak, which may be increased with Valsalva’s maneuver.

**Management**

Surgical repair by patching the round or oval window (site of leak) by an autogenous tissue followed by bed rest for 1 week is usually effective.

**Superior canal dehiscence**

**Case presentation**

The patient is a 46-year-old man with rumbling in his head when he talks or coughs and ear fullness on right side. He has had chronic imbalance for the past 7 years and has fallen twice in aerobic class. He complains of movement of world when his heart races. A petrous bone CT scan shows dehiscence of the superior SCC on the right side (Fig. 9A). When the patient blows out through a pinched nose, he forces air into the middle ear, which travels through the oval window, and distorts the utricle to cause ocular tilt response (Fig. 9B). The pressure wave then passes out through the superior SCC dehiscence. The ocular tilt response caused the right eye to move up and intort and the left eye to move down and extort. The patient had a craniotomy to expose the top of the superior SCC at the top of the petrous bone. A bone plate was inserted over the superior SCC, which stopped the symptoms and signs.

**Pathophysiology**

Superior canal dehiscence causes brief, episodic dizziness when patients change positions of their head (eg, lie down); during pressure changes between the middle and inner ear (eg, Valsalva’s maneuver); or in response to loud noises (Tullio’s phenomenon). This entity was first described by Minor [47], who has done most of the seminal work. Less than 1% of the population may be born with thinning of the bony top of the superior SCC. Relatively mild head trauma or middle-inner ear pressure change may expose the membranous canal of the superior SCC to the overlying dura, which then provokes the symptoms described previously.
Diagnosis is suggested by eliciting vertical nystagmus or an ocular tilt response with pressure changes across the middle-inner ear barrier. Diagnosis is confirmed by thin-cut petrous bone CT scans (preferably 0.5-mm cuts) through the superior SCC. There are several options for treatment. A bony patch can be placed over the site of the dehiscence. Another option is to plug the superior SCC to prevent pressure changes. A third option is avoidance of pressure changes and use of ear plugs in patients who have noise-induced dizziness (Tullio’s phenomenon).

**Management**

Fig. 9. Dehiscence of the superior SCC. (A) Coronal CT scan of the petrous bone. Arrow illustrates bony portion of the superior SCC without a bony roof. (B) Sketch of the ear, which illustrates loss of bone above the superior SCC (dehiscence).
Orthostatic hypotension or intolerance

Case presentation
A 63-year-old man experiences transient lightheadedness whenever he gets out of bed and occasionally when he stands up. He also reports chronic fatigue and severe fatigue, dizziness, and unsteadiness 1 hour after large meals. He had a myocardial infarction 5 years ago and coronary artery bypass surgery 2 years ago. His medications include propranolol, hydrochlorothiazide, and nitropaste. The neurologic and neuro-otologic examinations were normal. Blood pressure lying down for 10 minutes was 130/75 and pulse was 62. One minute after standing up, blood pressure was 115/70 and pulse was 65. There was no further change at 5 minutes of standing.

A diagnosis of orthostatic hypotension was made. His dizziness decreased after his cardiologist decreased the dose of propranolol. To prevent post-prandial hypotension, the patient ate small meals. To prevent relative supine hypertension, the patient slept with the head elevated 30 degrees on a wedge pillow at night.

Pathophysiology
Symptoms can range from lightheadedness when first standing up to chronic tiredness, mental slowing, dizziness, nausea, and impending syncope. Common causes include medications (diuretics, antihypertensive medication, prolong bed rest, and tricyclic antidepressants), and neurogenic (autonomic neuropathy from diabetes, amyloid, multisystem atrophy, and Parkinson’s disease). Diagnosis is made based on a drop in systolic pressure by 20 mm Hg or more when the patient stands associated with symptoms of lightheadedness. If there is no drop in blood pressure but the patient is symptomatic, then they may have orthostatic intolerance. It is possible to confuse BPPV with orthostatic hypotension. A key difference to keep in mind is that orthostatic hypotension only causes dizziness when the patient sits up or stands up.

Management
Management begins with removal of potentially offending drugs if possible and rehydration if necessary. Salt and fluid intake should be increased. The patient is asked to sleep with the head elevated to reduce supine hypertension. If necessary fludrocortisone, 0.1 to 0.6 mg each day, is used (see Table 10). If this fails then midodrine, 10 mg three times a day, is given. In a double-blind, placebo-controlled study, midodrine significantly increased standing systolic blood pressure by 22 mm Hg (P<0.001) and decreased orthostatic dizziness, fatigue, and weakness (P<0.05) [48].

Transient ischemic attacks

Case presentation
A 50-year-old man had vertigo and nausea for 2 minutes while driving home 2 weeks ago. This morning, he had a sense of tumbling, being pulled
to the left, diaphoresis, and nausea for 2 minutes while sitting and talking on the telephone. His past medical history is pertinent for a myocardial infarction in 1984 and 1985. He had cardiac angioplasty in 1995. He had a motor vehicle accident 14 months ago and had developed spasm in paracervical region. His mother had a myocardial infarction at age 72. His medications include propranolol, 80 mg long-acting, and aspirin, 325 mg. His neuro-otology and neurologic examinations were normal.

A diagnosis of vertebral-basilar transient ischemic attacks was made. He declined admission and was placed on ticlopidine (Ticlid), 250 mg twice a day, and scheduled for outpatient studies. Within the next few days he had three episodes of vertigo, diaphoresis, and nausea. One spell occurred while showering; two others while sitting and turning his head to the side. He was admitted and had a cerebral arteriogram, which showed a vertebral artery dissection on the left side with limited flow. Transcranial Doppler showed decreased flow in the basilar artery during head turn. He was placed on warfarin and had no further spells of vertigo. Six months later the arteriogram was repeated. The vertebral artery had recannulized and the warfarin was stopped.

Pathophysiology

Transient ischemic attacks from vertebrobasilar ischemia provoke episodes of dizziness that are abrupt and usually only last for a few minutes. Transient ischemic attacks frequently are associated with other vertebrobasilar ischemia symptoms, most commonly visual disturbance, drop attacks, unsteadiness, or weakness. A small percentage of patients with vertebrobasilar ischemia may present with isolated spells of vertigo, presumably caused by ischemia in the distribution of the vestibular artery. These patients usually have known cerebrovascular disease or risk factors for this disease.

Management

MR arteriography can be performed to assess posterior circulation vessels and transcranial Doppler may detect decreased flow in the basilar artery. Treatment includes reduction of risk factors for cerebrovascular disease and antiplatelet therapy. Warfarin is used when there is significant vertebral or basilar artery stenosis [15].

Migraine

Case example

A 45-year-old woman has weekly spells of dizziness, nausea, and imbalance that last for several hours. The dizziness is usually provoked by head movements. During these spells, she has a mild pressure discomfort in the head and prefers to lie down. The spells are more common just before her menses. She also has moderate motion sickness while riding in the car
and avoids all amusement rides. She has a history of migraine headaches, some of which are associated with a scintillating scotoma. Her examination is normal.

Because migraine-equivalent spells is the most likely diagnosis, she was placed on a migraine diet, stress reduction, and asked to come back during the day of a spell. When she came back a mild positional nystagmus with mild vertigo was noted during the Dix-Hallpike test. This nystagmus stopped the next day. With a change in her diet her spells were reduced to a few times each year.

Pathophysiology

The mechanism of dizziness or vertigo from migraine is unknown. These patients may have other migraine spells including visual disturbance. They frequently have increased motion sickness. Spells usually last 4 to 60 minutes and may or may not be associated with a headache. The International Headache Society criteria can be helpful for the diagnosis of migraine. Because this is a diagnosis of exclusion, the diagnosis is secured with a positive response to treatment.

Management

Management of spells of vertigo caused by migraine is the same as that used for headaches. After establishing the diagnosis and reassuring the patient, the author gives the patient a handout listing the risk factors and foods that may precipitate an aura [49]. Patients are encouraged to avoid hypoglycemia by eating every 6 to 8 hours, avoid nicotine, avoid or reduce exogenous estrogen, and try to maintain a regular sleep schedule. If strict avoidance of these risk factors does not significantly reduce their spells based on a diary they keep, then a daily antiserotonergic medication is used [50]. Beta-blockers are among the most effective prophylactic drugs for migraine, but exercise intolerance and orthostatic hypotension may be a problem. Valproic acid can also be used prophylactically for migraine.

Psychogenic (anxiety)

Case example

A 29-year-old electrician complained of “dizziness for the past 2 years.” By dizziness he means trouble walking, poor balance, linear movement, tilt, floating, rocking, and blurred vision as all equal terms for his dizziness. The dizziness started while he was working on a high lift for 2 hours. He was at a height that caused a sense of rocking on the platform. In addition, he attributes his dizziness to inhalation of fumes of a floor sealant on the job. Since then he has constant dizziness, which is severe when he first awakens in the morning. It is also severe when he is fatigued or while walking in a dark room. His head feels heavy. He denied vertigo, hearing loss, and
tinnitus. Because of his symptoms, he reduced his exercise program. In the last 6 months he has had loss of strength, energy, memory loss, paresthesias, muscle and joint aches, trouble sleeping and speaking, tremor, incoordination, and headaches. His past medical history included surgery to the knee years ago that required intravenous antibiotics, gonorrhea treated with antibiotics, and anxiety and panic attacks 2 years ago. His mother had been on chronic benzodiazepines for stress. In the last 6 months dizziness has interfered with the patient’s activities 95% of the time and currently it is moderately intense. It has markedly changed his ability to work or do household chores. Dizziness has markedly decreased the amount of satisfaction or enjoyment the patient gets in taking part in family-related or social activities. His physical examination was normal. He already had an MRI with and without gadolinium that included 8th nerve cuts and a caloric, which were normal.

This patient has several features consistent with chronic anxiety. His complaints are vague, numerous, and out of proportion to his findings. Complaints of floating and rocking are typical for anxiety or depression. He had a history of panic attacks 2 years ago. There is likely a family history of anxiety because his mother has been on benzodiazepines for “stress.” There is frequently a family history of stress, anxiety, or nervousness in patients with anxiety. Exercise is an excellent stress reducer. He stopped all exercise 2 years ago. A tentative diagnosis of chronic anxiety was made. The symptoms from stress and anxiety were discussed with the patient. He was told that these symptoms are very real and can be extreme. The role of his past medical and family history for anxiety was discussed. He was encouraged to restart a regular exercise program to help reduce stress. He was started on paroxetine, 10 mg every morning, and clonazepam, 0.5 mg every evening; the side effects were explained. He was asked to return in 3 weeks. When he returned to clinic, most of his symptoms had resolved. He was exercising on a regular basis. The clonazepam was tapered over a 3-week period, but the paroxetine was continued for 1 year.

**Pathophysiology**

Panic attacks are an anxiety disorder that causes intense fear or discomfort that reaches a crescendo within 10 minutes, and is frequently associated with dizziness, nausea, shortness of breath or chest tightness, paresthesia, and sweating. It may occur unexpectedly or be situational bound. Chronic anxiety may also present with dizziness. Helpful diagnostic criteria for anxiety can be found in the *Diagnostic and Statistical Manual-IV Options Book*.

**Management**

The selective serotonin reuptake inhibitors (paroxetine and others) have been approved for panic disorder and the author has also found it effective for treatment of dizziness from chronic anxiety (see Table 10). This may be an ideal medication because it is non–habit forming and many patients with
anxiety have concomitant depression. Alprazolam can be used sporadically if attacks are infrequent (see Table 10). Behavioral modification is also effective. In a clinical outcome study using this form of treatment, 96.1% remain in remission at 2 years and 67.4% for at least 7 years [51].

**Meniere’s disease and hydrops**

**Case example**

A 35-year-old man complained of one spell per month of vertigo, nausea, and imbalance lasting for a day. A day before each spell the patient noticed left ear fullness associated with hearing loss and roaring tinnitus. Gadolinium MRI of the head was normal including 8th nerve cuts. The patient’s clinical examination is normal. The patient was asked to come back during the day of a spell. When the patient did so, he was found to have a left-beat jerk nystagmus, imbalance, and significant hearing loss on the left side. Repeat audiogram that day showed significant hearing loss in the low frequencies. He was treated with promethazine suppository for a few days. His symptoms resolved.

A diagnosis of Meniere’s disease was made based on the report of episodic vertigo with fluctuating hearing loss and a normal MRI of the head. He was placed on a 2-g or less sodium diet and acetazolamide, 250 mg twice a day. His spells decreased in frequency to one to two per year. Repeat yearly audiograms showed no significant progression in his baseline hearing loss.

**Pathophysiology**

Spells caused by Meniere’s disease or hydrops usually involve a low-pitched form of tinnitus, ear fullness, and hearing loss (often associated with vertigo), which lasts for hours to days. With repeat attacks, a sustained low-frequency sensorineural hearing loss and constant tinnitus develop. The pathologic mechanism is believed to be decreased reabsorption of endolymph in the endolymphatic sac. This may be idiopathic (Meniere’s disease) or may occur following ear disease (hydrops) from trauma or viral infection. The diagnosis is facilitated by documenting fluctuating hearing loss on audiograms. During the acute attack, nystagmus may be excitatory (beats toward the involved ear).

**Management**

Management begins with restricting the patient’s sodium intake to no more than 2 g a day. The mechanism of this form of treatment is not known. It was originally based on patient observation that spells of Meniere’s were triggered by consumption of salty food. Less proved prophylactic therapy includes avoidance of alcohol and caffeinated products (including chocolate). These dietary changes may significantly reduce the frequency of dizzy
spells. Some patients also need a diuretic. Acetazolamide may be the best choice because this drug may decrease osmotic pressure within the endolymph, but chlorothalidone and other diuretics have also been found to be beneficial [52,53]. During acute spells of dizziness the patient is treated with phenothiazines to reduce nausea. Blood work and vestibular exercises are usually not necessary, however, because the patient typically recovers quickly. Medical therapy may not control Meniere’s or hydrops. Endolymphatic shunts may be used; however, they are not always effective or may stop working after a few years. Labyrinthectomy (by injection of gentamicin into the middle ear near the round window) may be used in patients who have severe, pre-existing hearing loss on the side of the Meniere’s disease. Vestibular neurectomy may be done in patients whose hearing is useable.

References