Update

Central Causes of Dizziness

Vestibular physical therapy has become a mainstay in the management of patients with balance disorders manifesting as dizziness and disequilibrium. Numerous concept papers,[1–20] case studies,[21–29] case series with no controls,[30–44] and controlled studies[45–49] support the use of rehabilitation techniques for patients with peripheral vestibular disorders. There is less evidence supporting the use of rehabilitation techniques for patients with central vestibular disorders. Several concept papers,[6,9,17,50–54] case studies,[4,23,55–59] and case series with no controls[17,23,30,31,33,36,38–40] have been published regarding central vestibular disorders. In addition, 2 textbooks on the subject of vestibular rehabilitation[60,61] provide ample evidence that this treatment for patients with balance disorders appears to be very promising (Tab. 1). Only a few controlled studies,[45–49] however, have addressed the efficacy of physical therapy for patients with peripheral vestibular disorders, and none of these controlled studies have addressed patients with central vestibular pathology. Articles that specifically addressed benign paroxysmal positional vertigo[62–65] are not included in Table 1. Most of the studies cited in the case series group in Table 1 had few patients with central dysfunction, and rarely were these patients differentiated from the patients with peripheral vestibular disorders, making it difficult to reach any definitive conclusions.

Overall, patients with central vestibular disorders have worse outcomes of rehabilitation than do patients with peripheral vestibular disorders.[53] Patients with central disorders often cannot be progressed as quickly as patients with peripheral vestibular dysfunction; thus, overall treatment times are longer for patients with central disorders.[53] Outcomes of rehabilitation in patients with central vestibular disorders, for example, following head trauma, are not as good as outcomes following peripheral vestibular disorders.[39] Patients with only central vestibular disorders, however, have better outcomes than do patients with combined peripheral and central dysfunction.[39]

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Joseph M Furman

Susan L Whitney

Table 1.
References for Concept Papers, Case Studies, Case Series With No Controls, and Controlled Studies Regarding Physical Therapy for Patients With Peripheral or Central Vestibular Disorders

	Peripheral Vestibular Disorders	Central Vestibular Disorders
Concept papers	1–20	6, 9, 17, 50–54
Case studies	21–29	4, 23, 55–59
Case series/no controls	30–44	17, 23, 30, 31, 33, 36, 38–40
Controlled studies	45–49	

Much of the published material regarding physical therapy for balance disorders concerns the response to injury of the inner ear structures that provide information to the central nervous system regarding angular acceleration, linear acceleration, and orientation of the head with respect to gravity. This update is particularly concerned with a different aspect of balance function, namely, central nervous system abnormalities that cause dizziness directly or that affect a patient's response to or recovery from an inner ear abnormality.

Clinicians often categorize patients with vestibular system abnormalities as those with peripheral vestibular disease versus those with central vestibular disease. The peripheral vestibular system consists of the vestibular end organs, including the 3 semicircular canals and the 2 otolith organs in each ear, and the vestibular portion of the eighth cranial nerve. Conversely, the central vestibular system consists of those structures and pathways listed in Table 2. The root entry zone of the vestibular portion of the eighth cranial nerve, although within the substance of the brain itself, is considered a peripheral vestibular structure. One of the most important structures in the central vestibular system is the vestibular nuclear complex. This structure, which consists of 4 vestibular subnuclei, is located in the medulla and caudal pons and receives inputs not only from the peripheral vestibular system but also from other sensory modalities, including vision and somatosensation.[66] The vestibular nuclear complex is a sensory integration center with neural outputs that are important for controlling eye movements and postural movements and for spatial orientation. Another critical central vestibular structure is the cerebellum. Whereas the vestibular nuclei process and relay vestibular signals, the vestibulocerebellum (ie, the flocculonodular lobe) is particularly important for modulating vestibular responses and allowing the vestibular system to adapt to injury, disease, and changes in sensorimotor demands.[67] The integrity of the central vestibular system, and especially the vestibulocerebellum, affects a person's ability to recover from a peripheral vestibular ailment.[68]

Central nervous system abnormalities can cause dizziness or can affect a person's response to or recovery from an inner ear abnormality.

Patients with dizziness comprise a large portion of the patients seen in emergency departments (6.7%)[69] and by primary care physicians (2.6%).[70] Some of these patients appear to have a vestibular system abnormality to account for their dizziness, whereas other patients do not. A subset of patients with vestibular disease will be evaluated and treated by physical therapists. We believe that knowledge of how vestibular disorders may be manifested and, in particular, of the importance of central vestibular structures can improve a therapist's effectiveness. Table 3[71(p92)] shows some of the characteristic features in the medical history that can help distinguish patients with peripheral vestibular disease from those with central vestibular disease. Central vestibular disorders are more likely than peripheral vestibular ailments to cause imbalance, and central disorders are more likely than peripheral disorders to be associated with other neurologic symptoms. Additional information regarding the localization of the patient's pathology may be obtained from the duration of his or her episodes of dizziness (Tab. 4[71(p92)]). Although the duration of a patient's symptoms, if they occur in episodes, may provide clues to localization, there is a large overlap in this aspect of the history between central and peripheral disorders. Nonetheless, this aspect of the history is useful.

The most common peripheral vestibular disorder that is likely to be seen by a physical therapist is benign

JM Furman, MD, PhD, is Professor, Departments of Otolaryngology, Neurology, Bioengineering, and Physical Therapy, University of Pittsburgh, Pittsburgh, Pa. Address all correspondence to Dr Furman at Department of Otolaryngology, University of Pittsburgh School of Medicine, 203 Lothrop St, Suite 500, Pittsburgh, PA 15213 (USA) (furman@pitt.edu).

SL Whitney, PT, PhD, ATC, is Assistant Professor, Departments of Physical Therapy and Otolaryngology, University of Pittsburgh, and Director, Physical Therapy Vestibular Rehabilitation Program, Centers for Rehab Services, Pittsburgh, Pa.

Dr Furman and Dr Whitney provided concept/research design and writing.

Table 2.
Central Vestibular System

Vestibular nuclei
Vestibulo-ocular pathways
Vestibulospinal pathways
Vestibulocolic pathways
Vestibulo-autonomic pathways
Vestibulocerebral pathways
Vestibulocerebellum
Perihypoglossal nuclei

paroxysmal positional vertigo. This disease has been discussed recently in the physical therapy literature.[62–65] People with another common peripheral vestibular ailment, Ménière disease (ie, endolymphatic hydrops), typically are not seen by physical therapists because the episodes are self-limited. The most common central vestibular disorders occurring with dizziness are migraine-associated dizziness, the sequelae of trauma, ischemic disease (including vertebrobasilar insufficiency and brain-stem stroke), and degenerative neurologic disorders that affect the cerebellum. Failure to compensate for a peripheral ailment (eg, inadequate recovery from a unilateral peripheral vestibular loss) also should be considered a central vestibular disorder.

This update will discuss 4 disease entities that affect the central vestibular system. Because this update is meant to supplement recently published material concerning the presentation of peripheral vestibular disorders to physical therapists, we chose several diseases for discussion that include a range of underlying pathophysiologies and a diversity of signs and symptoms. Each of the disorders will be discussed primarily in terms of diagnosis. Information in the literature regarding physical therapy intervention for central balance disorders is quite limited (Tab. 1). Where possible, implications regarding physical therapy intervention will be provided.

Migraine

Migraine-associated dizziness[72–88] is an underrecognized disorder that is actually quite common with a prevalence of 6.5%.[88,89] Many people with a history of migraine headaches may have dizziness with some or all of their headaches. Other people with migraine headaches may have migraine-associated dizziness in isolation, that is, dizziness without headache.[87,88] Rarely, people who have no history of headache may experience migraine-associated dizziness. Migraine-associated dizziness can manifest itself as a spinning sensation preceding a migraine headache, as dizziness and imbalance during a migraine headache, or as dizziness without headache. Some people experience a sense of imbalance and dizziness between episodes of headaches. A recent study by Cass et al[87] has indicated the large variability in the duration of symptoms in people with migraine-associated dizziness.

Establishing a diagnosis of migraine-associated dizziness can be difficult because it is largely a diagnosis of exclusion.[88] That is, migraine is a diagnosis that is reached when no other plausible diagnosis is available. Migraine-associated dizziness should be considered in all patients who have migraine headache or a positive family history of migraine without other diagnoses. Additional clues in the history may be a sense of imbalance in complex visual or motion environments. Physical examination findings are typically normal. The results of vestibular laboratory tests (including caloric testing, ocular-motor testing, rotational testing, positional testing, and posturography) may be abnormal, but they usually do not indicate a definite peripheral or central vestibular abnormality.[87] In particular, asymmetric nystagmus responses to rotational stimulation have been reported with migraine-associated dizziness.[72,87] Because physical examination findings are typically normal and no single laboratory test is available to establish a diagnosis of migraine-associated dizziness definitively, the diagnosis remains uncertain in most patients. Although the diagnosis remains uncertain, however, the disorder is quite common.[88,89]

Treatment for patients with migraine-associated dizziness (Tab. 5[87]) does not usually include physical therapy. Johnson[88] has indicated some success with physical therapy for patients with migraine-associated dizziness. Johnson's patients completed a vestibular rehabilitation program in addition to having pharmacologic management of their migraine-related dizziness. In our experience, patients with migraine who receive balance therapy also generally improve but may worsen transiently. Because migraine is so frequent, many patients referred for physical therapy for balance disorders may have migraines even if they are not the primary cause of their balance problem. The presence of migraine in addition to other vestibular ailments can complicate the therapist's choice of treatments because of possible transient worsening of symptoms. Moreover, many patients with migraine are medicated (eg, with an antidepressant), which may influence a patient's response to physical therapy. In our experience, in addition to other treatments (including physical therapy and pharmacotherapy), providing patients with informational brochures about migraine triggers can be extremely important in helping them to manage their dizziness and to reduce triggers (ie, foods, sensory stimuli, or behaviors that may lead to an increased likelihood of symptoms).

Trauma

Head trauma may cause central nervous system trauma (eg, a postconcussion syndrome), a labyrinthine concus-

Table 3.
Differentiation Between Peripheral (End-Organ and Nerve) and Central Causes of Vertigo[a]

Cause	Nausea and Vomiting	Imbalance	Hearing Loss	Oscillopsia	Neurologic Symptoms	Compensation
Peripheral	Severe	Mild	Common	Mild	Rare	Rapid
Central	Moderate	Severe	Rare	Severe	Common	Slow

[a] Adapted with permission from Baloh and Honrubia.[71(p92)]

Table 4.
Duration of Common Causes of Vertigo[a]

Cause	Duration
Benign positional vertigo	Seconds
Vertebrobasilar insufficiency	Minutes
Ménière syndrome	Hours
Vestibular neuritis, infarction of the labyrinth	Days

[a] Adapted with permission from Baloh and Honrubia.[71(p92)]

Table 5.
Treatment Options for Migraine-Related Vestibulopathy[a]

1. Avoid dietary triggers
2. Treat underlying migraine phenomenon
   - Tricyclic antidepressants (eg, amitriptyline 50–100 mg/d)
   - Beta-blockers (eg, propranolol 80–320 mg/d)
   - Calcium channel blockers (eg, verapamil 80–120 mg/d)
3. Treat movement-associated disequilibrium
   - Vestibular physical therapy
4. Treat space and motion discomfort
   - Phenergan/pseudoephedrine (25 mg/60 mg twice daily)
5. Treat associated anxiety or panic disorder
   - Behavioral therapy
   - Pharmacotherapy
     Tricyclic antidepressants
     Anxiolytic (eg, benzodiazepine)

[a] Adapted with permission from Cass et al.[87]

sion, a neck injury (eg, whiplash), or some combination of these injuries. Thus, physical therapists must be aware that patients who experience dizziness following head trauma may have a combination of central vestibular abnormalities, peripheral vestibular abnormalities, and neck injuries. Other conditions that may exist in patients who have experienced head trauma and who have dizziness include posttraumatic benign paroxysmal positional vertigo, posttraumatic Ménière disease, and perilymphatic fistula.[90] Moreover, a central nervous system injury may impair the process of compensating for a peripheral vestibular ailment.[68]

Central nervous system trauma can produce dizziness either on the basis of a postconcussion syndrome presumably resulting from cerebral injuries or due to a brain-stem injury.[91(pp244–245)] The most critical factor in the patient's history in arriving at a diagnosis of posttraumatic dizziness is the observation of a temporal association between the patient's head trauma and his or her dizziness. A complicating factor in the evaluation of a patient who has sustained head trauma is that the peripheral vestibular system or the neck also may have been damaged. The term "labyrinthine concussion" is used to describe peripheral vestibular dysfunction following head trauma.[71(pp244–245)] In such cases, caloric testing usually indicates a unilateral reduction in function.[90]

Head trauma resulting from automobile accidents often is associated with "whiplash" (ie, a flexion-extension injury). The neck plays a critical role in balance.[91] Although "cervical vertigo" is poorly described and nearly impossible to diagnose definitively,[92] the accurate detection by the central nervous system of the position of the head with respect to the torso is critical for normal vestibulospinal function.[93,94] To assist in determining whether a patient has cervical vertigo, the head-fixed–body-turned maneuver can be used to stimulate the neck without stimulating the labyrinth and can cause nystagmus.[95] To illustrate the importance of afferent information from the neck to central vestibular structures, a simple example is as follows: With the head turned to the left, a forward body movement results in head movement toward the right labyrinth and away from the left labyrinth. With the head turned to the right, however, a forward body movement results in a movement of the head toward the left ear and away from the right ear. Thus, the central vestibular system must reverse the signals sent to the postural control system as the head-on-torso position changes from left to right.

Any traumatic injury that impairs the ability of the central nervous system to locate accurately the head on the torso can produce dizziness and disequilibrium. Unfortunately, there is no definitive test for establishing a diagnosis of cervical (ie, neck) dizziness or vertigo.[92]

Patients with cervical vertigo may complain of dizziness-associated headaches. They also may complain of a "swimming sensation" in the head and often have restrictions in cervical range of motion. Difficulty sleeping because of neck pain is common. Patients may also have referred pain into their shoulders and scapular region.

Table 6.
Symptoms Associated With Vertebrobasilar Insufficiency[a]

Symptom	Percentage
Visual dysfunction	69
Drop attacks	33
Unsteadiness, incoordination	21
Extremity weakness	21
Confusion	17
Headache	14
Hearing loss	14
Loss of consciousness	10
Extremity numbness	10
Dysarthria	10
Tinnitus	10
Perioral numbness	5

[a] Adapted with permission from Grad and Baloh.[101]

The treatment of patients experiencing posttraumatic dizziness must be planned with the realization that both the peripheral and central vestibular systems may be involved simultaneously with or without concomitant neck problems. Intervention will vary depending on the individual patient's presentation. Assessing segmental movement of the cervical spine and treating the dysfunction may result in better functional outcomes.[96] Muscle relaxants and physical therapy are used to improve range of motion of the neck and to reduce neck muscle pain and spasm.[97,98] In our opinion, the use of a cervical collar should be limited to no more than 1 to 2 hours per day. In addition, in our experience, range of motion, gentle manual traction, and acupressure may help to relieve muscle spasm, decrease pain, and reduce dizziness.

Brain-Stem Stroke and Vertebrobasilar Insufficiency

Establishing the diagnosis of a brain-stem stroke in a patient with dizziness is usually straightforward because there is a sudden onset of dizziness in addition to other neurologic symptoms, including alterations of vision, strength, coordination, or sensation.[99] Establishing the diagnosis of vertebrobasilar insufficiency, however, is more problematic because symptoms are transient. Dizziness alone is infrequently the presenting sign of vertebrobasilar insufficiency.[100] Moreover, with time, the diagnosis of vertebrobasilar insufficiency becomes increasingly unlikely in the absence of associated symptoms such as those listed in Table 6.[101]

Probably the most easily recognized central vestibular disorder is that of Wallenberg syndrome, which is caused by infarction of the lateral medulla (which includes the vestibular nuclei).[102,103] Most often this disorder is due to ischemia in the distribution of the posterior inferior cerebellar artery (PICA).[102,103] Damage to surrounding central nervous system structures produces a highly characteristic complex of symptoms and neurologic signs. A closely related syndrome is the anterior inferior cerebellar artery (AICA) syndrome.[102,104] The distinction between these 2 syndromes (ie, occlusion of PICA versus AICA) is probably unimportant. Table 7[105] lists the symptoms, signs, laboratory abnormalities, and pathophysiology of Wallenberg syndrome (caused by occlusion of the PICA) and of the AICA syndrome. Because of the central rather than peripheral localization of these syndromes, patients with an infarction of the vestibular nuclei might be expected to have a very poor prognosis and respond poorly to physical therapy. That is, the central vestibular structures that are important for vestibular compensation, sensory-sensory interaction, and adaptation to altered sensory inputs may have been damaged. Despite these obvious potential limitations, such patients often respond well to physical therapy. The reason for this somewhat paradoxical finding may be that patients with brain-stem strokes are likely to have unilateral lesions, with preserved function contralaterally. In addition, there may be sufficient redundancy in central vestibular pathways to allow partial recovery of balance function.

Therapists should be careful not to compromise vertebrobasilar circulation. If the physical therapist suspects vertebrobasilar insufficiency, the patient's physician should be notified immediately. Whether extension and rotation of the neck[106] should or should not be performed in an attempt to diagnose vertebrobasilar insufficiency is controversial.[107–111] In our opinion, such a maneuver should not be performed because it may not be a valid screening test.[107] Such maneuvers also may reduce blood flow in the vertebrobasilar circulation and lead to an infarction.[108,110] In any case, patients should be warned about the perils of excessive rotation and extension of the cervical spine. A neck collar might be warranted.

Cerebellar Degeneration

There are several well-defined, genetically determined causes of cerebellar degeneration. Some patients have nonsyndromal adult-onset cerebellar degeneration.[112(pp243–253)] Older individuals may have symptoms and signs consistent with cerebellar dysfunction but no clear family history of spinocerebellar degeneration or any obvious etiology for cerebellar dysfunction such as excessive chronic ethanol intake, vasculitis, a paraneoplastic process, or a structural abnormality (eg, a Chiari malformation).[113] Patients with cerebellar degeneration typically have a gradual decline in balance function that is especially prominent while walking on uneven surfaces.[57,114] These patients typically have no otologic symptoms, including no hearing loss, tinnitus, or vertigo.[112(pp189–194)] Physical examination may uncover abnormal ocular pursuit, improperly sized saccades, nystagmus (especially with downward gaze or with

Table 7.
Comparison of Posterior Inferior Cerebellar Artery (PICA) and Anterior Inferior Cerebellar Artery (AICA) Syndromes[a]

	Seen in Both PICA and AICA Syndromes	Typically Seen Only in PICA Syndrome	Typically Seen Only in AICA Syndrome
Symptoms	Vertigo, lateropulsion, unusual visual illusions, facial numbness, limb numbness, disequilibrium, dysphagia, and incoordination	Hoarseness	Tinnitus, hearing loss, and facial weakness
Signs	Vestibular nystagmus, decreased facial sensations ipsilaterally, dissociated sensory loss to pain and temperature contralaterally, Horner syndrome, ipsilateral limb ataxia, and gait ataxia	Saccadic lateropulsion, skew deviation, and vocal cord paralysis	Hearing loss, facial weakness, and gaze palsy
Laboratory abnormalities	Abnormal imaging, spontaneous nystagmus, and decreased hearing	Saccadic lateropulsion	Caloric reduction ipsilaterally
Pathophysiology	Damage of fifth nerve nucleus, spinothalamic tract, and vestibular nuclei	Damage of nucleus ambiguus and dorsal motor nucleus	Damage of inner ear, eighth cranial nerve, seventh cranial nerve, seventh and eighth cranial nerve root-entry zones, sixth nerve nucleus, flocculus, and middle cerebellar peduncle

[a] Adapted with permission from Furman and Cass.[105]

Table 8.
Differential Features of the Common Ataxia Syndromes[a]

Syndrome	Clinical	Electronystagmography	Neuroimaging
Friedreich ataxia	Early onset, muscle atrophy, areflexia	Saccadic dysmetria, ocular flutter, decreased calorics	Mild cerebellar atrophy
Olivopontocerebellar atrophy	Early to midlife onset, spasticity, hyperreflexia	Slow saccades, impaired pursuit and fixation suppression of vestibulo-ocular reflex	Prominent atrophy of pons and cerebellum (diffuse)
Cerebellar cortical atrophy (Holmes, Marie, Foix, Alajouanine)	Late onset, predominantly truncal, dysarthria	Downbeat and rebound nystagmus, central positional nystagmus, impaired pursuit and fixation suppression of vestibulo-ocular reflex	Prominent cerebellar vermian atrophy
Familial periodic vertigo and ataxia	Early onset, episodes induced by exercise, stress	Usually normal, may have downbeat or rebound nystagmus	Usually normal, may have vermian atrophy

[a] Adapted with permission from Baloh and Honrubia.[71(p257)]

oblique downward and lateral gaze), incoordination of the arms and legs, a wide-based ataxic gait, and inability to tandem walk.[112(pp194–215),115] These patients often have uneven stride lengths and decreased gait speed. Brain imaging may uncover shrinkage of the cerebellum, which may be particularly prominent in the midline.[116]

Patients with cerebellar degeneration are likely to experience problems with both the vestibulo-ocular system and the postural control system. Patients with disorders affecting the vestibulocerebellum may have difficulty with sensory integration, particularly visual-vestibular interaction.[117] Patients may have difficulty performing rapid head movements and maintaining balance while walking, especially if simultaneously moving the head and attempting to walk on uneven surfaces. These patients' problems may be multifactorial because some syndromes (eg, Friedreich ataxia) may include peripheral vestibular involvement because of degeneration of the eighth cranial nerve.[118]

Patients with cerebellar degeneration may have associated brain-stem abnormalities if their degeneration syndrome is not localized solely to the cerebellum. Brain-stem dysfunction, in addition to cerebellar dysfunction, will increase disability related to abnormal corticospinal tract function.[113] The clinical features of some of the more common ataxia syndromes are listed in

Table 8.[71(p257)] Some rare forms of cerebellar degeneration are associated with periodic vertigo, which may be responsive to treatment with acetazolamide.[119]

In our opinion, patients with cerebellar dysfunction, in general, should not receive vestibular suppressant medications because most of these medications will act to worsen cerebellar function. In our opinion, treatment regimens for such patients should include equalizing step lengths, narrowing step width, balance exercises on complaint surfaces, and enhancing somatosensory inputs to the lower extremities. Additionally, providing an assistive device such as a cane or walker may be helpful. In one study,[57] improvement was noted in postural stability after a 6-week period of exercise in 2 cases of cerebellar disease. Patients with cerebellar disease are able to suppress visual stimulation over time and rely more on their vestibular and proprioceptive inputs.[120] Gait instability and ataxia are the chief complaints of many older patients with cerebellar degeneration. Assessing the patient's gait speed and assessing the patient's step symmetry are 2 easy clinical methods for determining whether a patient's gait is improving.[121]

Conclusion

Patients with central vestibular disorders are among those individuals who are evaluated and treated by physical therapists for balance disorders. This update has focused on 4 central vestibular ailments (ie, migraine, trauma, vascular insufficiencies, and cerebellar degeneration). Although numerous case studies of patients with central vestibular disorders suggest that such patients can improve with physical therapy intervention,[4,23,55–59] controlled, randomized studies remain to be performed.

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