Examination and Treatment of Peripheral Vestibular Disorders

By
Janine Hatch, PT, DPT, MS
Geriatric Clinical Specialist
Certified Vestibular Rehabilitation Specialist

Upon successful completion of this course, continuing education hours will be awarded as follows:
Physical Therapists: 4 Contact Hours
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Janine Hatch has disclosed that she has no significant financial or other conflicts of interest pertaining to this course book.

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Rebecca English has disclosed that she has no significant financial or other conflicts of interest pertaining to this course book.

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EXAMINATION AND TREATMENT OF  
PERIPHERAL VESTIBULAR DISORDERS  

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OBJECTIVES: After completing this course, I am able to:
1. Identify the anatomy and peripheral vestibular physiology, and pathways underlying gaze stabilization and postural control.
2. Differentiate between the types of peripheral vestibular disorders.
3. Select appropriate examination measures based on patient report and presentation.
4. Design appropriate treatment interventions based on interpretation of examination findings.

COURSE CONTENT
5. The course content was presented in a well-organized and clearly written manner.
6. The course content was presented in a fair, unbiased, and balanced manner.
7. The course content presented current developments in the field.
8. The course was relevant to my professional practice or interests.
9. The final examination was at an appropriate level for the content of the course.
10. The course expanded my knowledge and enhanced my skills related to the subject matter.
11. I intend to apply the knowledge and skills I’ve learned to my practice.
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continued on next page
COURSE RATING

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   A. Poor       B. Below Average       C. Average       D. Good       E. Excellent

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2. Compare your answers with the answers in the PRETEST KEY located at the end of the pretest. The pretest key indicates the page where the content of that question is discussed. Make note of the questions you missed, so that you can focus on those areas as you complete the course.

3. Read the entire course and complete the exam questions at the end of the course. Answers to the exam questions should be logged on the FasTrax test sheet included with the course.

Note: Choose the one option that BEST answers each question.

1. The purpose of the vestibular system is to
   a. detect movement in the environment to maintain postural control.
   b. detect head movement to maintain gaze stabilization and postural control.
   c. integrate sensory cues to maintain postural control.
   d. generate smooth coordinated movement to maintain postural control.

2. The most common etiology of dizziness in persons over 60 years of age is
   a. acoustic neuroma.
   b. cerebral vascular accident.
   c. benign paroxysmal positional vertigo (BPPV).
   d. vestibular neuronitis.

3. The clinical profile of a person with peripheral vestibular disorder includes
   a. dizziness, wide base of support with ambulation, tremor.
   b. dizziness, wide base of support with ambulation, ataxia.
   c. dizziness, loss of balance with head turn with ambulation, diminished gaze stabilization.
   d. dizziness, loss of balance with head turn with ambulation, headache.

4. Which of the following is the appropriate clinical examination method or tool to diagnose BPPV?
   a. The Berg Balance Scale
   b. Gaze stabilization testing
   c. The Modified Clinical Test of Sensory Interaction and Balance (mCTSIB)
   d. Positional testing

5. A targeted treatment approach for post-acute unilateral vestibular hypofunction includes
   a. adaptation exercises.
   b. single-limb stance and tandem stance training.
   c. pharmacological vestibular suppressants.
   d. canalith repositioning maneuvers.

PRETEST KEY
1. B page 2
2. C page 14
3. C page 15
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5. A page 35
INTRODUCTION

COURSE OBJECTIVES

After completing this course, the learner will be able to:

1. Identify the anatomy and peripheral vestibular physiology, and pathways underlying gaze stabilization and postural control.
2. Differentiate between the types of peripheral vestibular disorders.
3. Select appropriate examination measures based on patient report and presentation.
4. Design appropriate treatment interventions based on interpretation of examination findings.

Vestibular disorders are common in the adult population, affecting a vast number of persons over 40 years of age, with increased prevalence of vestibular dysfunction occurring with advancing age. Persons diagnosed with vestibular disorders have a higher likelihood of experiencing problems with balance and dizziness, leading to reduced physical function and falls; subsequent early nursing home placement and mortality make early diagnosis and effective management essential. There is strong research evidence to support the efficacy of vestibular rehabilitation to reduce or eliminate the symptoms and sequelae associated with common vestibular disorders (Fujino et al., 1994; Lee & Kim, 2010; Hall et al., 2016).

The vestibular system is a highly specialized sensory apparatus that integrates information regarding head movement and position relative to gravity with information from the visual and somatosensory systems to mediate upright posture, postural control, and gaze stabilization. Given the highly specialized nature of this system, physical therapy management of persons with vestibular dysfunction requires additional education and training beyond what is offered in a typical entry-level physical therapy program curriculum.

The vestibular system detects head motion to mediate gaze stabilization and postural control during head motion. Information from the vestibular end organs are integrated with sensory information from the visual and somatosensory systems in the central nervous system (CNS) to generate compensatory movements to maintain head and body postural stability and stabilize vision. Vestibular system impairments can result in a variety of symptoms, including vertigo and difficulties with gait and balance, which are the primary reasons patients with vestibular dysfunction seek care. Peripheral vestibular dysfunction is more common than centrally mediated vestibular disorders, with the two most common peripheral vestibular disorders being benign paroxysmal positional vertigo (BPPV) and vestibular neuritis, each of which is effectively rehabilitated through a problem-oriented treatment approach.

Detailed knowledge of the vestibular system is important for physical therapists to effectively manage balance and gait disturbances in patients with vestibular dysfunction. Differential assessment of the source of peripheral vestibular dysfunction depends on a comprehensive subjective account of symptom
provocation and severity, as well as a targeted clinical examination. The key components of a clinical examination include oculomotor and vestibular function testing, measures of balance, gait, and fall risk, and a review of musculoskeletal and neuromuscular systems.

The purpose of this course is to educate the practicing physical therapist on the management of patients with peripheral vestibular disorders in order to expand their current practice skill set into the subspecialty of vestibular rehabilitation. This will be accomplished through knowledge of functional anatomy of the vestibular system and current methods of evidence-based examination and the process of differential assessment to determine effective treatment of common peripheral vestibular disorders, or when referral is appropriate.

This intermediate-level course is targeted at practicing physical therapists who have experience managing patients with balance dysfunction and basic knowledge of standardized gait and balance measures.
OVERVIEW OF VESTIBULAR DYSFUNCTION

Dizziness is a common symptom in the older adult, with 20 to 35% of persons over age 65 reporting problems with dizziness in the previous 12 months (Lin & Bhattacharyya, 2012; Tinetti, Williams, & Gill, 2000). Symptoms of dizziness can have a catastrophic impact on wellness, often leading to unsteadiness with walking and increased incidences of falls and associated injuries. Persons experiencing difficulties with balance due to dizziness can develop a fear of falling, and subsequently reduce their mobility and participation in activities (Vellas, Cayla, Bocquet, De Pemille, & Albarede, 1987), with as many as 56% of older adults restricting activities due to this fear (Howland et al., 1998).

A systematic review of studies identifying the etiology of dizziness in the adult population revealed vestibulopathy, cardiovascular dysfunction, central nervous system (CNS) dysfunction or lesion, psychiatric, and other medical disorders (e.g., metabolic, polypharmacy) as the primary diagnostic classifications (Kroenke, Hoffman, & Einstadter, 2000). Of these, dizziness was attributed to peripheral vestibulopathy in 44% of the patients studied. Vestibular dysfunction affects 35% of adults over age 40, with the incidence rising to as much as 85% for persons 80 years of age and older (Agrawal, Carey, Della Santina, Schubert, & Minor, 2009). Hallmark symptoms of vestibular dysfunction are vertigo, dizziness, nausea, oscillopsia, and imbalance, which are the primary reasons for seeking medical care. Vertigo is the illusion of self-motion or object motion in a rotational manner, which the patient often describes as “spinning.” Patients with vestibular dysfunction can also report changes in cognition and a sense of feeling “foggy” or experiencing “brain fog.” Taken together, the consequences of these symptoms can have a profound negative impact on the patient’s state of physical, social, and emotional well-being.

Data from the U. S. National Health Interview Survey revealed the enormity of the impact on quality of life for individuals experiencing problems with dizziness and balance limitations (Lin & Bhattacharyya, 2012). Of the seven million persons surveyed, 61% reported not participating in their usual activities, such as exercise, 45% did not participate in social activities, and one-quarter required assistance with self-care and activities of daily living due to problems with their balance. Thus, in addition to the risk of injurious falls, there is a potentially dramatic impact on loss of independence and a subsequent emotional toll that ensues in persons with vestibular dysfunction, necessitating early diagnosis and effective management to reduce the decline in quality of life.
Of those individuals seeking care for their balance problems, the vast majority saw a general practitioner, neurologist, or cardiologist, 16% saw an otolaryngologist, and nearly 3% sought chiropractic care (Lin & Bhattacharyya, 2012). The source of dizziness complaints is not always easy to identify; it can range from polypharmacy to neurological disorders to vestibular pathology. Effective diagnosis and management of balance disorders requires detailed knowledge of the systems that mediate postural control, which includes the vestibular system. When peripheral vestibular dysfunction is the source of dizziness and balance limitations, as is true in nearly half of the cases, pharmacological or surgical management is not typically warranted. Rather, most cases of peripheral vestibular dysfunction require rehabilitative care to facilitate CNS compensation for the loss of vestibular function or to address the mechanical basis for the altered function. Compensation refers to the resolution of those symptoms associated with vestibular dysfunction, such as vertigo, nausea, and imbalance, despite permanent loss or damage to peripheral structures noted on objective tests (Herdman & Clendaniel, 2014). Given the vast numbers of individuals afflicted with peripheral vestibular disorders and that best practice dictates a focus on a problem-oriented treatment approach, physical therapists are well-positioned as the practitioner of choice to address the needs of this population.

**FUNCTIONAL AND STRUCTURAL ANATOMY OF THE VESTIBULAR SYSTEM**

The vestibular system is a highly specialized sensory apparatus that is responsible for sensing head motion and position relative to gravity. Information from the vestibular end organs is integrated with sensory information from the visual and somatosensory systems in the CNS to generate compensatory movements to maintain head and body postural stability and head-eye coordination. The anatomy of the vestibular system is composed of a peripheral portion, which includes the three semicircular canals, the two otolith organs, and the vestibulocochlear nerve (CN VIII), and a central portion that includes the vestibular nuclei and the cerebellum. Motor output from the vestibular system is mediated by the vestibulo-ocular and vestibulospinal white-matter tracts. The vestibulo-ocular pathway sends information regarding linear and angular head motion to the extra-ocular muscles to mediate gaze stabilization during movement. The vestibulospinal tract carries information to the skeletal muscles to drive compensatory antigravity balance reactions in response to head position changes.

**Peripheral Vestibular Structures**

The peripheral vestibular system lies within the labyrinth of the inner ear, and is composed of a bony and a membranous component (see Figure 1). The bony labyrinth contains two main structures, the cochlea and the semicircular canals, which are adjoined by the vestibule.

The membranous labyrinth lies within the bony labyrinth and is suspended by connective tissue and perilymphatic fluid, which is similar in chemical nature to cerebrospinal fluid (see Figure 2). Within this membranous structure is the membranous portion of the semicircular canals and the two otolith structures, named the utricle and saccule. Within the membranous labyrinth is endolymphatic fluid, which is similar to intracellular fluid in its composition.

**Semicircular Canals**

The semicircular canals and otoliths are the motion sensors of the vestibular system, detecting angular head motion. The three semicircular canals are oriented in orthogonal planes,
which means that they lie at 90° angles from one another, and are named for their relative location in the inner ear: anterior, posterior, and horizontal, also known as superior, inferior, and lateral, respectively (see Figure 2).

Both ends of the semicircular canals are contiguous with the vestibule, with one end widened in a bulbous fashion, referred to as the ampulla. The ampullae serve an important anatomic role as they house the mechanoreceptors...
for the semicircular canals and create a barrier between the canal and vestibule. This configuration is the basis for the translation of head movement into neural firing. When angular head movement occurs, the endolymphatic fluid within the semicircular canals flows in the direction opposite to the direction of the head movement, creating a unidirectional displacement of the mechanoreceptors in the ampullae (Herdman & Clendaniel, 2014).

The mechanoreceptors within the ampullae are composed of two different types of hair cells; kinocilia and stereocilia. Kinocilia is the single, tallest hair cell, and the remaining smaller hair cells are called stereocilia. These hair cells sit on a bed of vascular and nerve fibers called the crista ampullaris, and are embedded in a gelatinous cone called the cupula (see Figure 3). This important configuration allows these mechanoreceptors to be bidirectionally sensitive, with deflection of the stereocilia toward or away from the kinocilia determining whether hair cell discharge frequency increases or decreases vestibular nerve firing. When the stereocilia are deflected toward the kinocilia (longer hair cells), there is a net increase in neural firing rate, or excitation. Conversely, when the stereocilia are deflected away from the kinocilia, there is a decrease in neural firing rate, or inhibition. In the horizontal canals, kinocilia are oriented in the ampullae away from the opening to the semicircular canal, so that displacement of the cupula, and hence, the kinocilia, toward the ampullae (away from the canal side) will result in excitation. With lateral head rotation to the right, kinocilia will be deflected toward the ampulla on the right, causing excitation on that side, and away from the ampulla on the left, causing inhibition on the left side. In the anterior and posterior canals, the kinocilia are oriented toward the semicircular canal side, so that posterior rotation of the head causes excitation in the posterior canals and inhibition in the anterior canals (see Figure 3).

In the semicircular canals, endolymph displacement is proportional to head velocity, thereby defining the role of the semicircular

FIGURE 3: ANATOMY OF THE CRISTA AMPULLARIS

Note. From Western Schools, 2018.
canals as “rate sensors.” This 1:1 ratio of velocity of head movement and endolymph flow, or gain, maintains the image stably on the retina during high-velocity movements. Without this, oscillopsia (blurred images) would result. The anatomic organization of the ampullae underlies the dynamic characteristics of the semicircular canals’ response to prolonged rotation at constant velocity. At constant velocity of head rotation, steady state is achieved as inertia is eventually overcome, and the ampullae return to resting position, so excitation ceases to exist. These dynamics serve an important functional role in everyday life. Consider the example of riding in an amusement ride, where the rider initially perceives the change in motion with some disorientation and vertigo. That is not a sensation that we usually want to persist! After a brief period of substance velocity, the rider then experiences the sense of steady state, and adapts to the motion. Of course, once the ride stops the cupula will deflect in the opposite direction consistent with the properties of inertia, and the rider will once again experience a brief period of vertigo and disorientation.

The semicircular canals are oriented as three coplanar pairs between the labyrinths, meaning that the right and left horizontal canals are one pair, the right posterior and the left anterior canals are another pair, and the left posterior and the right anterior canals are the third coplanar pair. When head movement occurs in a paired plane, the endolymph is displaced on each side in opposite directions relative to their ampullae, causing an increase in neural firing in one canal and a decrease in firing in the opposite paired canal. This coplanar pairing is responsible for the “push-pull arrangement” that drives CNS detection of head movement. This arrangement of coplanar pairing allows for redundancy, so that in the event of vestibular canal disease or dysfunction, the CNS will still receive information regarding head movement from the vestibular system in that plane of motion from the other canal of that coplanar pair. Comparison of the rate and magnitude in change of neural firing allows the CNS to detect velocity and direction of head movement so that it may effectively elicit compensatory motor responses to maintain postural control and gaze stabilization.

Vestibular afferent neurons have a tonic (baseline) firing rate of about 90 to 100 pulses per second (pps). Excitation of vestibular afferents is correlated with head movement, such that a head velocity of 50° per second results in a change in firing rate of the afferent nerves of 50 pps. Specifically, that would translate to a firing rate of 150 pps (baseline plus 50 pps) on the excitatory side and 50 pps (baseline minus 50 pps) on the inhibitory side. However, the vestibular system does not respond as well to high-velocity head movement due to an asymmetry in the excitation-inhibition firing responses. The basis for this phenomenon, referred to as “inhibitory cutoff,” is that although increased firing on the excitatory side correlates with head velocity, hyperpolarization (decreased firing) on the contralateral side can be reduced only to zero. This is defined by Ewald’s Second Law, which states that responses to rotation that excite a semicircular canal are greater than responses to rotations that inhibit a canal (Herdman & Clendaniel, 2014). The firing rate of the vestibular nerve on the inhibitory side can be driven to 0 pps with head velocities of as little as 180° per second. Therefore, the inhibitory cutoff phenomenon will occur with head velocities greater than this. Under the conditions of normal activities that require rapid head motion, and certain sporting activities, the velocity of head movement can be as great as 550° per second, requiring the vestibular system to depend on the excitatory side to detect the velocity of head motion in order
to elicit effective postural responses. We can quickly see why persons with unilateral loss of vestibular function have loss of balance or difficulty maintaining gaze stabilization when they turn their head quickly to the affected side.

**Otoliths**

The two otolith structures, the saccule and the utricle, are contained within the vestibule. The saccule has a vertical orientation, whereas the utricle is oriented horizontally (see Figure 2). The otoliths are the gravity-sensitive structures of the vestibular system, responding to linear motion and head position. The vertically oriented saccule senses motion predominantly in the sagittal plane, such as forward tilt (pitch) and vertical linear acceleration, while the horizontally oriented utricle senses motion predominantly in the horizontal plane, such lateral tilt (roll) or horizontal linear acceleration of the head. Together, they are able to detect linear acceleration in all possible vectors of the vertical, horizontal, and transverse planes of motion (Herdman & Clendaniel, 2014; Jacobson & Shepard, 2008).

A classic example of otolith function is that of riding in an elevator. Although from a visual perspective it appears as though the environment is stationary, the otoliths are able to register the vertical linear acceleration that is taking place, thus making it possible to appreciate that vertical movement is occurring.

Unlike the semicircular canals that rely on a hydrodynamic system to translate head movement into neural firing, calcium carbonate crystals (otoconia) embedded in the gelatinous macula of the otoliths detect linear acceleration and static head tilt with respect to gravity (see Figure 4).

As a result, neural firing is elicited from a shifting or shearing force of the otoconia on the macula, perpendicular to the hair cells, causing the hair cells to deflect in response to gravity-referenced head movement. Also in contrast to mechanoreceptors of the semicircular canals, which are organized in clusters, the hair cells of the otoliths are organized in curved sheets, bisected by a central strip of hair cells called a striola (see Figure 5). The kinocilia in the utricle are oriented toward their striola, whereas kinocilia in the saccule are oriented away from their striola, creating a push-pull arrangement in the otoliths in response to static head tilt and linear acceleration. This anatomical organization also results in excitatory and inhibitory neural firing within each macula, creating redundancy in the push-pull arrangement, which is thought to be the reason the otoliths may have less vulnerability to unilateral vestibular loss than the semicircular canals (Baloh & Halmagyi, 1996; Herdman & Clendaniel, 2014).

**Vestibular Nerve**

Neural projections carrying afferent information from the vestibular apparatus arise from the hair cells of the semicircular canals and otoliths and join with the cochlear nerve to become the CN VIII. The cell bodies of the primary vestibular afferents are located in the vestibular ganglion, also known as Scarpa’s ganglion. The vestibulocochlear nerve passes through the internal auditory canal, along with the facial nerve (CN VI) and labyrinthine artery, to synapse on the lateral, medial, inferior, and superior vestibular nuclei at the pontomedullary junction of the brainstem, located at the cerebellopontine angle. Projections are also sent to the cerebellum to monitor vestibular performance and make necessary adjustments for posture and coordination of limb movements. Inputs to the vestibular nuclei also include sensory information from spinal cord afferents, inputs from the cerebellum, and projections from contralateral vestibular nuclei.

Outputs from the vestibular nuclei include projections to the cerebellum, the lateral and
FIGURE 4: ANATOMY OF THE MACULA OF THE OTOLITHS

Otoconia
Gelatinous layer
Hair tufts from hair cells
Basilar membrane
Support cells
CN VIII nerve fibers

Note. From Western Schools, 2018.

FIGURE 5: OTOLITH ORGANIZATION

Superior
Striola
Saccule
Parasagittal plane
Utricle
Horizontal plane
Lateral
Anterior

Note. From Western Schools, 2018.
medial vestibulospinal tracts, reticulospinal tract, thalamus, motor nuclei of the extra-ocular muscles (ascending medial vestibulospinal tract), cerebral cortex, and the vestibular apparatus. The role of these connections in maintaining postural control will be discussed in later sections.

**Central Vestibular Structures**

The CNS components of the vestibular system are the cerebellum and the vestibular nuclei. The cerebellum is the central adaptive processor of the vestibular complex, readjusting and recalibrating signals from the vestibular system to drive smooth coordinated movement and postural control. It does this through integrating inputs from the vestibular system and spinal cord afferents with motor output neurons, maintaining head-eye coordination, postural control during static and dynamic activities, and coordination of limb movements (Herdman & Clendaniel, 2014; Jacobson & Shepard, 2008). Contextual orientation to upright and the environment is achieved through cerebellar integration of somatosensory, visual, and vestibular information, which drives appropriate motor responses for effective maintenance of balance and postural control.

The four vestibular nuclei (superior, inferior, medial, and lateral) are located in the brainstem at the level of the pons and medulla, and serve as the cranial nuclei of the vestibular nerve. The pathways for the vestibulo-ocular reflex (VOR) arise from the superior and medial nuclei. The lateral vestibular nucleus gives rise to the vestibulospinal pathways with some contribution from the medial vestibular nucleus. The inferior vestibular nucleus is connected to all other vestibular nuclei and the cerebellum, but has no primary motor output.

**Vascular Supply**

The vascular supply for the peripheral and central portions of the vestibular system arises from the vertebral-basilar arterial system. The two vertebral arteries travel up the right and left side of brainstem to give off branches at the level of the medulla, called the posterior inferior cerebellar artery (PICA), which supplies the inferior aspect of the cerebellar hemispheres as well as the inferior aspect of the vestibular nuclear complex. As the vertebral arteries become the vertebral-basilar artery at the level of the pons, the anterior inferior cerebellar arteries (AICA) branch out, supplying the peripheral vestibular system, via the labyrinthine artery, and ventrolateral aspects of the cerebellum (see Figure 6).

The labyrinthine artery bifurcates into the superior or anterior vestibular artery and the common cochlear artery, which eventually splits off to give rise to the posterior vestibular artery. The anterior vestibular artery supplies the anterior and horizontal semicircular canals and the otoliths, whereas the posterior vertebral artery supplies the posterior semicircular canals and the saccule. It is important to appreciate that the AICA is the sole circulatory supply for the peripheral vestibular system, as an AICA infarct would result in complete unilateral loss of peripheral vestibular function.

**MOTOR OUTPUT OF THE VESTIBULAR SYSTEM**

The purpose of the vestibular system is to maintain head-eye coordination to stabilize gaze during head movement, such as when we walk, run, and turn, and to maintain an upright position during motion. It does this through neural pathways to the ocular muscles as well as the spinal cord that mediate the VOR and the vestibulospinal reflex (VSR), respectively.

**Vestibulo-ocular Pathways and Vestibulo-ocular Reflex**

The VOR is responsible for generating rapid reflexive eye movement in response to head
movement to maintain gaze stability while the head is moving. To maintain stable gaze, the VOR must generate eye movements in the opposite direction and with equal velocity of head movement. This 1:1 relationship of head-eye movement is expressed as the “gain” of the VOR system. The motor pathways of the VOR originate at the level of the vestibular nuclei, carrying information to the nuclei of the ocular motorneurons (CN III, CN IV, CN VI) via two white-matter tracts to control head-eye coordination. These vestibulo-ocular tracts are the ascending tract of Deiters and the medial longitudinal fasciculus (MLF). The ascending tract of Deiters carries output from the horizontal canals to the abducens nuclei (CN VI) to drive motor activity of the ipsilateral lateral rectus muscles of the eyes during horizontal head motion. Recalling the push-pull neurodynamics of the coplanar pairs of the semicircular canals, when the head is rotated to the right, excitation occurs in the mechanoreceptors of the right canal, and hyperpolarization (decreased firing) occurs in the mechanoreceptors of the left canal. The VOR pathways carry excitatory inputs generated in the right semicircular canal to the left lateral rectus and right medial rectus to elicit leftward eye movement corresponding to rightward head movement (see Figure 7). At the same time, the VOR pathways also carry inhibitory inputs generated from the left horizontal canal to the right lateral and left medial rectus muscles. This antagonistic arrangement of extra-ocular muscle activity is referred to as the Law of Reciprocal Innervation (Leigh & Zee, 1991) and describes the correlate relaxation (inhibition) of a muscle while the opposing muscle is contracting. In this example, the medial and lateral rectus muscles of each eye are undergoing reciprocal inhibition. This response results in lateral eye movement that is equal and opposite to lateral head rotational movement.
Outputs from the anterior and posterior semicircular canals are carried to the other nuclei of the extra-ocular muscles (CN III and CN IV) via the MLF, mediating reciprocal excitation and inhibition of the superior and inferior rectus and oblique muscles, resulting in reflexive rotary eye movement with an upward or downward component motion. In this instance, the contralateral posterior and anterior canals are working as coplanar pairs. The superior rectus muscles direct eye movement in a vertical plane (up and down). The oblique muscles drive rotational eye movement, with the superior oblique generating movement down and in, and the inferior oblique generating eye movement up and out. When head movement occurs in the plane of the right posterior canal (head tipped back with rotation to the right) VOR pathways from the right posterior canal carry excitatory responses to the ipsilateral superior oblique muscle and contralateral inferior rectus muscle, and simultaneous inhibition to the ipsilateral
inferior oblique muscle and contralateral superior rectus muscle. At the same time, the VOR pathways for the contralateral anterior canal, being the coplanar pair of the posterior canal, mediate the same pattern of muscle activity. In other words, head tilt in the plane of the right posterior canal, and subsequent left anterior canal, results in excitation of the right superior oblique and superior rectus muscles, and the contralateral correlate of left inferior oblique and inferior rectus muscles. Inhibition occurs in the opposite pairs of eye muscles. The net effect is a downward and left torsional eye movement. Thus, eye movement occurs in the opposite direction of head movement to maintain eye position fixed on the target.

Vestibulospinal Pathways and Vestibulospinal Reflex

The VSR utilizes three neural pathways as outputs from the vestibular system to the anterior horn cells of the skeletal muscles. These pathways are the medial vestibulospinal tract (MVST), which innervates muscles of the trunk and neck, the lateral vestibulospinal tract (LVST), innervating the extremities, and the reticulospinal tract. The reticulospinal tract is a highly collateralized, yet poorly defined region of the spinal cord that receives input from all vestibular nuclei, as well as other sensory and motor inputs responsible for postural control (Herdman & Clendaniel, 2014).

The LVST and MVST receive inputs from the otoliths and semicircular canals to mediate antigravity motor responses to maintain upright postural control and vertical orientation of the head in response to head position changes with respect to gravity. The LVST primarily drives reactionary balance responses, such as ankle, hip, and stepping strategies, in response to changes in the center of mass. It is also responsible for eliciting protective extension responses, such as extending legs or arms, when the center of mass is displaced enough to generate head movement toward the ground. In addition, increased flexor activity is stimulated on the opposite side. For example, head and body tilt (or loss of balance) to the right would elicit right arm and leg extension, and left arm and leg flexion. It is clear to see that the primary role of the VSR is to protect the head from hitting the ground – a feature that is very compatible with surviving a fall!

CERVICAL CONTRIBUTION TO POSTURAL CONTROL

Cervical reflexes (cervico-ocular, cervico-spinal, and cervicocollic) are mediated through cervical afferent inputs to aid the vestibular system in maintaining gaze stabilization and postural orientation.

Cervico-ocular Pathway and Cervico-ocular Reflex (COR)

Inputs from neck proprioceptors interact with the VOR system to aid in stabilizing images on the retina during slow head movements, or very low gain. The functional significance of this mechanism is not well understood, but it may be tapped into as a secondary gaze stabilization system when vestibular damage has occurred.

Cervicospinal Pathway and Cervicospinal Reflex (CSR)

Like the COR, the CSR (also known as the tonic neck reflex) receives inputs from neck proprioceptors to augment the role of the VSR in stabilizing the body during head motion. The pathways that mediate the CSR are an excitatory pathway from the lateral vestibular nucleus, and an inhibitory pathway from the reticular formation. The fundamental role of the CSR is to provide information to the CNS to
differentiate head motion that occurs from head-on-neck versus head-on-body. For example, when the body tilts forward, the vestibular system registers forward head motion in the vertical gravity-referenced direction, subsequently eliciting a protective extension response through the VSR in an effort to maintain postural control. Conversely, when the head is tilted forward on the neck, the same vestibular inputs register forward head motion, but in this case neck proprioceptors also register neck flexion motion. As a result, CSR and VSR inputs cancel one another and the head moves on the neck without unnecessary balance responses, thereby maintaining postural stability.

**Cervicocollic Pathway and Cervicocollic Reflex (CCR)**

The CCR stabilizes the head on the body. Changes in neck position creating a stretch in neck muscles will result in a reflexive contraction of appropriate neck muscles to elicit head righting in the vertical plane.

**SENSORY CONTRIBUTION TO POSTURAL CONTROL**

Postural control is defined as the body’s ability to maintain the center of mass (COM) within the base of support (BOS) during quiet postures or positions (static postural control) as well as during movement (dynamic postural control; Shumway-Cook & Woollacott, 2011). Normal postural control requires integration of sensory information regarding body position and motion with respect to the environment to elicit effective motor responses. Specific information from vestibular, visual, and somatosensory systems provides the CNS with a different frame of reference regarding body position and motion. The effect of these systems working together triangulates sensory data, allowing the CNS, primarily the cerebellum, to minimize sensory conflict, thereby gaining a clear schema of the body’s orientation in space in order to drive appropriate postural responses.

**Vestibular System**

Up to this point, we have focused on the role of the vestibular system in maintaining postural control. It provides the CNS with information regarding rotational and linear head motion and position, and is the only sensory structure that provides gravity-referenced information, to stabilize the head and trunk, achieve and maintain vertical orientation, and perceive self-motion. However, the vestibular inputs alone cannot help the CNS distinguish the environmental context in which motion is occurring to determine if the body is moving through the environment (e.g., walking) versus moving with the environment (e.g., riding in a train), in order to elicit the appropriate postural responses. For that, the CNS requires information from the visual and somatosensory systems.

**Visual System**

The visual system provides information to the CNS regarding head and body motion and position with respect to the environment, as well as referencing verticality. It also contributes to maintaining postural control during quiet stance, as is seen with the occurrence of increased postural sway when the eyes are closed as compared with eyes open. Another example of the influence of visual inputs for postural control comes from an experiment by Lee and Lishman (1975) where they provided continual environmental oscillations (movement of walls and ceiling) and noted that the neurologically intact subjects exhibited an increased sway in response to movement of the environment. Consider a familiar example of standing on a train platform and watching a moving train pass in front of you. There is a sense that you are moving, despite maintaining a standing
position on the platform, which comes from the visually mediated sway that is occurring. This example also illustrates that the visual system is unable to accurately distinguish self-motion from motion of the environment.

**Somatosensory System**

The somatosensory system provides the CNS with information regarding body position and motion with respect to the supporting surface. It also provides information about the relative position and relationship of body segments to one another. Somatosensory inputs register body and segment motion when moving along a horizontal surface, to detect changes in the conditions or motion of the surface to maintain verticality with respect to the surface. An example of this is walking along a cobblestone path or sandy beach, where ankle motion accommodates the uneven or compliant, shifting surface. However, when surface conditions change so that they are not horizontal, such as with a rocking ship or a ramp, orienting the body relative to the supporting surface is no longer effective. Rather, orienting with reference to gravity becomes the effective strategy, such as is mediated by the vestibular system.

**SENSORY INTEGRATION**

Understanding the role of the vestibular, visual, and somatosensory systems in detecting motion and position elucidates their relative contributions to the CNS to create a schema, or map, of the body with respect to the environment, which underlies postural control. We share many experiences where we have discovered how these systems work together. A common example of sensory integration is one where you are sitting in your car next to another car at the red light. Suddenly, the car next to you starts to move forward to get a jump on the green light, which your peripheral vision picks up as net motion, but it cannot distinguish whether you are rolling backward or the car next to you is moving forward. Since we tend to rely on visual information, despite its inaccuracies relative to movement, our brain misinterprets this information as self-motion and drives the motor response of pushing your foot down harder on the brake pedal. Unfortunately, that does not change the net movement we are perceiving since we are not in motion. That information is fed back to the CNS (no change), and sensory inputs are reinterpreted. Although the visual system is sending information to the CNS that movement is taking place, since we are not moving relative to the surface the somatosensory system is not detecting movement, and since there is no head movement the vestibular system is also sending information to the CNS that we are not moving. Thus, the sensory inputs are being compared and “reweighted” to resolve this sensory conflict. As a result, the CNS shifts its reliance on information from the two corroborating sensory systems, and we quickly come to the realization that the car next to us is in motion. These adaptations, occurring in a fraction of a second, are essential to successfully maintaining postural control under changing task conditions, and are the basis for the rehabilitation of peripheral vestibular deficits.

**TYPES OF PERIPHERAL VESTIBULAR DISORDERS**

Causes of peripheral vestibular dysfunction can arise from pathology of the semicircular canals, otoliths, or the CN VIII. Peripheral vestibular pathology is classified into three categories: distorted function, reduced function, and fluctuating function. Establishing the nature and character of complaints of dizziness is an essential first step in the clinical examination process as it helps to categorize the pathologic basis
of the patient’s symptoms. This is achieved by gaining an accurate description of the frequency, onset, and duration of episodes of dizziness through systematic differential questioning on intake interview. Determining the category of pathology plays an important role in guiding intervention and indicating whether referral for additional services may be required.

**Distorted Function**

Benign paroxysmal positional vertigo (BPPV) is the sole pathology that falls into this category. BPPV is the most common cause of dizziness symptoms in the older adult (van Leeuwen & Bruintjes, 2014). The incidence of BPPV is greater for adults 60 years of age as compared to younger adults, with the incidence of BPPV peaking in the sixth and seventh decades of life (Hilton & Pinder, 2003; von Brevern et al., 2007). Symptoms associated with BPPV are characterized as brief periods of vertigo, lasting less than 60 seconds, and brought on by stereotypical head positions relative to gravity. The pathological mechanism that underlies BPPV is caused by otoconia that have been dislodged from the macula in the utricle either through natural degenerative changes or by trauma, such as a fall. The displaced otoconia eventually find their way to the semicircular canals, resulting in free-floating debris within the canal, or debris that has adhered to the cupula. The term **canalithiasis** refers to the condition in which debris is floating freely in the semicircular canal. The presence of otoconia in the affected semicircular canal changes the fluid dynamics of that canal in response to head movement. In this condition, the otoconia create a hydrodynamic drag of endolymph when the affected canal is moved into the direction of gravity, creating an increased magnitude of response in the cupula. The term **cupulolithiasis** refers to the condition in which otoconia have adhered to the cupula, thus increasing the mass of the cupula, making it more sensitive to gravity (Lee & Kim, 2010). Otoliths will most often migrate to the posterior semicircular canal due to its more inferior orientation in the ear relative to gravity, with the prevalence of posterior canal BPPV reported to be upwards of 96%. The horizontal canal is the next most affected canal, with studies showing that horizontal canal BPPV occurs approximately 2 to 16% of the time. BPPV of the anterior canal is rare (Fife, 1998; Honrubia, Baloh, Harris, & Jacobson, 1999; Jacobson & Shepard, 2008; Macias, Lambert, Massingale, Ellensohn, & Fritz, 2000).

The chief symptom of BPPV is vertigo that is provoked with changes in head position. Some patients will also experience lightheadedness, nonspecific dizziness, postural instability, and nausea (Blatt, Georgakakis, Herdman, Clendaniel, & Tusa, 2000). Symptoms are brought on by rapid changes in head position that orient the affected canal in a gravity-dependent position. For the posterior canal, that is typically head extension and rotation toward the affected side, such as looking up to a high shelf, or when the patient gets into or out of bed transitioning toward the affected ear. Provocation of horizontal canal BPPV symptoms is common with rolling from one side to the other in bed, or lateral head tilt movements.

Once provoked, symptoms of vertigo will have a latent onset of 1 or more seconds as the gravity-referenced otoconia change position, inducing displacement of the cupula. The patient will then experience transient symptoms of dizziness or vertigo that will fatigue within 1 to 2 minutes while maintaining the provoking head position. In canalithiasis, the duration of symptoms is typically less than 60 seconds, while patients with cupulolithiasis will experience symptoms for as long as 2 minutes. Another characteristic feature of vertigo and dizziness associated with BPPV is the crescendo-decrescendo nature of the symptoms, meaning
that the patient will experience an increasing intensity of symptoms at onset, reaching a peak intensity before subsiding (Herdman & Clendaniel, 2014). It is important for the clinician to appreciate that symptoms associated with BPPV have a mechanical basis, whereas other vestibular disorders are caused by loss of function. Thus, the patient with BPPV will generally not experience symptoms if they avoid the offending head position.

**Reduced Function**

This category describes loss of vestibular function due to infection, degenerative changes, or injury affecting the CN VIII or membranous labyrinth, resulting in diminished or absent transmission of signals from the semicircular canals and otoliths to the CNS. The profile of symptoms commonly associated with reduced vestibular function include dizziness or vertigo, imbalance, gait disturbances, and difficulties with gaze stabilization that are provoked with movement through space, either through head movement, walking, or transitions. Symptoms may be chronic and persistent, being present even when movement is ceased, in contrast to BPPV, where symptoms are provoked with particular head position, triggering transient dizziness (Herdman & Clendaniel, 2014).

Neuronitis and labyrinthitis are the second most commonly seen peripheral vestibular disorders after BPPV (Herdman and Clendaniel, 2014; Kroenke et al., 2000). Neuronitis is inflammation of the CN VIII, affecting the superior branch of the vestibular nerve, which supplies the utricle and anterior and horizontal semicircular canals. Inflammation of the superior branch of the vestibular nerve will result in reduced transmission of information from the horizontal canal (horizontal canal paresis), and can result in utricular degeneration, causing posterior canal BPPV as a secondary complication. Labyrinthitis defines an inflammatory condition of the membranous labyrinth, which is essentially an infection of the entire inner ear, causing symptoms of neuronitis in addition to hearing loss and tinnitus. Multiple infectious processes have been implicated in causing vestibular neuronitis or labyrinthitis, the most common being an upper respiratory infection. Typically, the original illness precedes the onset of vestibular symptoms by as much as several weeks. Symptoms of dizziness or vertigo are generally self-limiting, with spontaneous recovery occurring in 1 to 3 days. The patient may also experience problems with gaze stabilization or blurred vision. When new onset of hearing loss is part of the presentation, infections such as mononucleosis, herpes zoster, Lyme disease, mumps, and measles must be considered, as well as the possibility of other noninfectious lesions such as acoustic neuroma (Baloh & Halmagyi, 1996; Herdman & Clendaniel, 2014). This profile of symptoms and findings necessitates a physician referral for further differential examination, including an audiogram, due to the likelihood of a medical pathology underlying the presentation.

Acute vestibular syndrome (AVS) is a condition in which there is a rapid onset of severe vertigo, nausea, vomiting, and spontaneous nystagmus with head motion intolerance and imbalance. It is caused by infectious neuronitis, or infectious labyrinthitis when hearing is involved. Because of its similar presentation to a CNS infarct, a differential assessment must be undertaken (Hotson & Baloh, 1998). Although AVS is most likely caused by infectious neuronitis or labyrinthitis, the older adult with at least one risk factor for vascular disease is at high risk for a brainstem stroke, which would result in a similar presentation, but require different management. A study by Kattah, Talkad, Wang, Hsieh, and Newman-Toker (2009) evaluated older adult patients with \( \geq 1 \) risk factor for stroke presenting to the emergency department
with symptoms of nystagmus, vertigo, nausea/vomiting, and gait unsteadiness to determine a centrally versus peripherally mediated cause for their symptoms. A bedside clinical exam with the acronym “HINTS,” which will be detailed in the examination section of the course, was performed and findings clinically correlated with imaging studies upon admission. They found that the HINTS bedside assessment was 100% sensitive and 96% specific in diagnosing pontine stroke as the cause of the patient’s presentation that mimicked AVS.

Injury to the peripheral vestibular structures can be a result of direct trauma, but can also include an ischemic event in the PICA or AICA, medication toxicity, and acoustic neuroma. PICA or AICA infarct would present as persistent dizziness or vertigo, along with balance and gait disorders due to unilateral loss of semicircular canal inputs to the CNS with regard to head motion. Because the PICA and AICA supply central structures, such as the pons and cerebellum, signs of central pathology may also be present, including dysmetria and dyscoordination.

Medication toxicity is the result of sensitivity to aminoglycosides (e.g., gentamycin, streptomycin). High dose use of these medications to manage systemic bacterial infections can result in irreversible ototoxicity in 15% of patients receiving this therapy, permanently damaging hair cells in the vestibular apparatus. As ototoxicity generally affects vestibular structures bilaterally, the patient will present without symptoms of vertigo or dizziness due to the lack of “mismatch” in bilateral vestibular inputs (Balogh & Halmagyi, 1996; Huth, Ricci, & Cheng, 2011).

Acoustic neuroma (vestibular schwannoma) is a benign schwannoma that arises on the vestibular portion of CN VIII at the location of the cerebellopontine angle. The incidence of acoustic neuroma is rare, with an incidence of 1.09 per 100,000 in the United States (Kshettry, Hsieh, Ostrom, Kruchko, & Barnholtz-Sloan, 2015). The initial presentation is unilateral sensorineural hearing loss, tinnitus (high-pitched ringing in the ear) and a sense of aural fullness, typically evolving slowly over the course of several months or years. Presentation of vestibular impairments is not a main feature, as the slow growing nature of this tumor allows for ongoing compensation of the CNS to the gradual loss of vestibular function (Balogh & Halmagyi, 1996), but can be a chief complaint in more than 30% of patients (Olshan, Srinivasan, Landrum, & Sataloff, 2014). Given the location of the neoplasm and its proximity to the facial nerve at the level of the brainstem (CN VI), patient presentation can also include hemifacial paralysis (Balogh & Halmagyi, 1996; Olshan et al., 2014).

**Fluctuating Function**

Ménière’s disease and perilymphatic fistula are the vestibular system disorders in this category, with structural abnormalities in the vestibular apparatus underlying the cause of distorted vestibular function.

Ménière’s disease is a function of malabsorption of endolymph (endolymphatic hydrops) in the endolymphatic duct and sac within the semicircular canals, creating an altered flow of endolymph. The clinical feature of Ménière’s disease is an acute spontaneous onset of disabling vertigo, postural imbalance, nausea, and vomiting, persisting for 24 hours. Recovery is also spontaneous, with no residual impairments. However, symptoms of balance limitations can persist for several days or weeks for some patients (Herdman & Clendaniel, 2014). Ménière’s disease is primarily managed through medical, surgical, and pharmacological means, with physical therapy care addressing any residual postural control deficits that may present after persistent attacks.
A perilymphatic fistula is an abnormality or patency that occurs in the round and oval windows of the middle ear, allowing leakage of perilymph fluid from the semicircular canals to the middle ear. The perilymphatic fistula may be a result of chronic pathological elasticity of the bony labyrinth, leading to episodic changes in fluid pressure and results in fluctuating symptoms of vertigo and imbalance, as well as hearing changes. Often, it is a result of an injury, such as a closed head injury, penetrating injury to the tympanic membrane, barotrauma, or vigorous straining, creating a sudden onset of vertigo, imbalance, and tinnitus. If diagnosed immediately, the patient is placed on bedrest with the head elevated for 5 to 10 days to allow healing to take place. The symptoms will often subside, and come on only with forceful maneuvers such as straining or sneezing, or changes in pressure in the inner ear. Persons with chronic or worsening symptoms may go on for surgical management.

CLINICAL EXAMINATION OF THE VESTIBULAR SYSTEM

The clinician begins a clinical or “bedside” examination of vestibular system function by achieving an accurate description of the patient’s complaints. Often, the term dizzy is used to describe a host of symptoms not related to vestibular system dysfunction, such as difficulty with walking, unsteadiness, headache, and lightheadedness. Symptoms of vertigo strongly indicate a vestibular system contribution to the patient’s complaints, while complaints of “lightheadedness” are suggestive of migraine, cardiac, and anxiety among other nonvestibular disorders as the source of the patient’s symptoms. Nausea and vomiting along with dizziness or vertigo is indicative of either central or peripheral vestibular lesions.

The temporal nature, onset, and duration of symptoms of dizziness also guide the clinician’s differential assessment process. The clinician should establish whether the patient is experiencing an acute attack of dizziness, described as onset within the last 3 days, is experiencing chronic dizziness, lasting more than 3 days, or if the patient’s dizziness is episodic. Understanding whether the onset was abrupt or insidious as well as particular provoking factors or preceding illness events is also diagnostic. For episodic dizziness, the provoking factor and duration of symptoms in terms of seconds, minutes, or hours are important to elucidate for the purposes of differentiating the source of symptoms and vestibular system impairment.

Clinical examination of vestibular function includes a review of systems, specific testing of the oculomotor and vestibular systems, assessment of postural control, and the use of standardized tools to measure the impact the disorder has on the patient’s life.

Review of Systems

A review of systems (ROS) is an important component of a comprehensive clinical examination. The ROS, in conjunction with the patient’s subjective report and medical history, gathers information that is vital to the process of differential assessment. In an ROS, the examiner performs a systematic screening of all body systems to identify the source of the patient’s symptoms. Of equal importance is identifying symptoms that are unrelated to the patient’s reason for seeking care. For the patient who presents with symptoms that implicate vestibular system dysfunction, such as dizziness and difficulty with balance, screening for symptoms stemming from the cardiac system, vascular system, neurological system,
psychological disorders, and polypharmacy are of utmost importance.

When assessing the presence of neurological system impairment as the possible etiology of symptoms of dizziness and impaired postural control, the examiner should look for changes in muscle strength and coordination, muscle tone, sensation, vision, hearing, speech, and cognition. Cardiac and vascular disorders may cause dizziness upon exertion, postural hypotension, and dizziness provoked with cervical extension motions due to vertebral circulation insufficiency. For the patient with a history of psychological disorders, the onset of psychogenic-related dizziness and balance dysfunction will have a situational trigger, be associated with palpitations, trembling, and shortness of breath, or be motivated by some aspect of secondary gain. A comprehensive review of the patient’s medications, both prescribed and over-the-counter, is essential to determine whether possible medication side effects or medication interactions are contributing to the patient’s primary complaints. Of particular importance is to determine whether the patient has been prescribed antihistamines (such as meclizine, Antivert) to dampen symptoms of dizziness, nausea, and vomiting as these medications will blunt the patient’s responses on vestibular testing, reducing the diagnostic value of findings on clinical examination. Furthermore, the patient with nonvestibular-related symptoms will demonstrate a pattern of clinical findings that do not characterize vestibular pathology. The differential assessment process should provide the clinician with enough evidence to determine whether to treat the patient or refer him or her to a more appropriate specialist for further diagnostic workup and management.

**Oculomotor System**

Examination of the oculomotor system establishes baseline function of eye muscles and central oculomotor pathways. Since the vestibular system mediates head-eye coordination, it is important to determine whether full active ocular movement is present in order to accurately interpret findings on vestibular testing. Furthermore, abnormalities seen on oculomotor testing indicate the possibility of a centrally mediated disorder as these tests assess the function of central oculomotor pathways that are independent of the vestibular system. The components of the oculomotor examination are observation of ocular motility and alignment, presence of nystagmus, tests of smooth pursuit and saccade, and vergence.

**Ocular Motility and Alignment**

The examination should start with observation of ocular motility and alignment. The patient is asked to actively look up, down, side-to-side, and across the diagonal, with the head stationary, to assess motor function of the medial and lateral rectus, trochlear, and superior and inferior oblique ocular muscles. Any abnormalities in resting alignment of the eyes in the orbits should also be appreciated. Observation of differences in vertical alignment of the eyes is called skew deviation. Skew deviation is a vertical misalignment of the eyes. Although typically a sign of CNS lesion, vertical skew deviation at rest can be seen in acute unilateral vestibular loss. The loss of utricular inputs on the side of the lesion results in the ipsilateral eye resting lower in the orbit, and the contralesional eye appearing higher in the orbit due to the loss of inhibitory input from the opposite (lesioned) side. If this finding is associated with acute peripheral vestibular dysfunction, resolution of skew deviation alignment will occur within 3 to 14 days due to spontaneous rebalancing of the tonic firing rate (Herdman & Clendaniel, 2014).

However, skew deviation is most often caused by a centrally mediated lesion, typically
in the cerebellum or brainstem. Further assessment of underlying skew deviation is achieved through the Cross-Cover Test, also called the Alternate Cover Test. The examiner alternately covers one eye and then the other, looking for changes in ocular position. In most cases of skew deviation, the covered eye will migrate either up or down, and when rapidly uncovered a vertical correction (corrective saccade) will be observed. This correction repositions the eye in the center of the orbit as aided by visual fixation once the eye is uncovered. The skew is named for the side that is elevated (migrates downward with visual fixation), with the other side being the side of the lesion.

**Nystagmus**

Assessing the presence of nystagmus is another important component of the oculomotor examination. Nystagmus is a rapid repetitive involuntary movement of the eyes that occurs under both normal and pathological conditions. Under normal conditions, nystagmus is elicited through vestibular or visual stimuli. It is seen in central or peripheral vestibular pathology due to an imbalance in vestibular outputs caused by a unilateral loss of peripheral vestibular function or disruption of central vestibular pathways. Reduced input to the ocular muscles from the lesioned side results in a slow conjugate eye deviation (slow phase) toward the side of the lesion, and a fast corrective movement (fast phase) away from the side of the lesion. For example, the presence of nystagmus with a vestibular lesion on the right would be seen as a conjugate horizontal jerking motion of the eyes, starting with a drift of the eyes off the visual target toward the right, followed by an immediate quick correction toward the left back onto the target. The direction of the nystagmus is named for the fast phase of eye movement. In this example, this would be defined as left beating nystagmus.

Two types of physiological, or normal, nystagmus are optokinetic and post-rotary nystagmus. Optokinetic nystagmus is induced by looking at a moving visual stimulus, such as watching a moving train while standing on the train platform. Visual responses for gaze fixation will cause the eye to re-establish fixation on the target as the target moves, resulting in a lateral nystagmus. Post-rotary nystagmus can be seen after a patient is subjected to continuous rotary motions, such as spinning in a chair, or that famous teacup ride in the amusement park, stimulating semicircular canal responses. Upon abrupt cessation of the spinning motion, the patient will exhibit a short duration of nystagmus until the endolymph flow and cupula of the semicircular canals return to resting state.

Nystagmus that occurs in the absence of visual or vestibular stimulus is found in peripheral and central vestibular pathology. Spontaneous nystagmus is observed while the patient is in a stationary position, without head movement. Acute loss of unilateral peripheral vestibular function, such as with vestibular neuritis or postoperative vestibular schwannoma resection, results in spontaneous nystagmus due to the asymmetry in baseline firing from semicircular canals, which is interpreted by the CNS as head movement. The presence of nystagmus due to acute unilateral vestibular loss will resolve within 1 to 2 weeks without intervention due to spontaneous compensation in the CNS, which rebalances tonic firing rates even when CN VIII does not undergo recovery (Mantokoudis, Schubert, Saber Tehrani, Wong, & Agrawal, 2013; Smith & Curthoys, 1989). However, spontaneous nystagmus due to CNS lesion is persistent.

Three features of nystagmus differentiate peripheral vestibular from central causes. First, nystagmus caused by peripheral vestibular dysfunction can be suppressed by visual fixation,
Examination and Treatment of Peripheral Vestibular Disorders

while centrally mediated nystagmus will not diminish or abate when the patient fixates on a target. Second, the direction of nystagmus is quite differentiating, with a peripheral lesion demonstrating movement in a mixed plane, usually horizontal with a torsional component, and central lesions demonstrating nystagmus in a single plane with either a torsional or vertical direction. Finally, the effect of gaze also distinguishes peripheral versus central causes of nystagmus. With peripheral vestibular lesions, the intensity of the nystagmus will increase when the patient looks into the direction of the quick phase, and decrease when the patient looks into the direction of the slow phase. This phenomenon is known as Alexander’s law (Leigh & Zee, 2006). With centrally mediated nystagmus, direction of gaze will have no effect or will elicit a change in direction of the nystagmus (direction-changing nystagmus) in that right beating nystagmus will occur with gaze to the right, changing to left beating nystagmus with gaze to the left (see Table 1).

In acute stages of peripheral lesions, spontaneous nystagmus can be seen with center gaze, as well as gaze to the right and left. This is called third-degree nystagmus. Within the first few days of recovery, nystagmus will be seen only at center gaze and gaze away from the side of the lesion – in the direction of the fast phase (second-degree nystagmus). As recovery continues over the course of a week or so, nystagmus can be seen only during gaze away from the side of the lesion (first-degree nystagmus). As nystagmus can be suppressed with visual fixation in the patient with peripherally mediated nystagmus, the use of Frenzel lenses or infrared goggles during examination will help the examiner appreciate the presence of the nystagmus.

Pressure-induced nystagmus should also be assessed. In this test, the examiner looks for drift of the eyes or the presence of mixed vertical and torsional nystagmus while pressure is induced in three ways: The patient puts pressure against the tragus of the ears (with his or her fingers) and exerts an external pressure (Hennebert’s sign), the patient closes his or her glottis and bears down (valsalva), or the patient attempts to blow out through pinched nostrils. A positive Hennebert’s sign demonstrates conjugate eye movement away from the affected ear with positive pressure (pressure against tragus), and conjugate eye movement away from the affected ear with negative pressure. Positive findings are suggestive of perilymphatic fistula, and sometimes Ménière’s disease.

Smooth Pursuit, Saccadic Eye Movements, and Vergence

Once baseline ocular motility and alignment have been established, the examiner assesses smooth pursuit and saccadic eye movements as well as vergence. Smooth pursuit testing assesses the quality of eye movement while the patient tracks a slowly moving target in all directions. The smooth pursuit system is used to maintain a moving target on the fovea of the retina during head movement at low velocities. While the vestibular system acts to maintain gaze at higher velocities, this system is driven by cortical regions in addition to the cerebellum.

<table>
<thead>
<tr>
<th>TABLE 1: PERIPHERAL VERSUS CENTRALLY MEDIATED NYSTAGMUS</th>
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<tr>
<td><strong>Peripheral Mediated</strong></td>
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<td>Direction</td>
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*Note: From Western Schools, 2018.*
and brainstem (Leigh & Zee, 2006). The examiner asks the patient to track a discreet target (pencil, finger) from a distance of 18 to 24 inches away. The object should move 30° from center in each direction at a speed of about 20° per second while the patient maintains a stationary head position. The examiner is looking for a smooth trajectory of oculomotor motion in all directions (lateral, vertical, diagonal).

Saccadic eye movement is rapid conjugate eye movement to place the fovea on a target. The examiner holds two discreet targets within the patient’s peripheral field of vision and asks the patient to look quickly between the two targets while keeping the head in a stationary position. If the patient’s eyes do not meet the target and a refixation on the target is observed, this is called a corrective saccade. The patient should normally be able to reach the target in one movement. Abnormal findings in saccadic and smooth pursuit eye movements are indicative of lesions in the brainstem or cerebellum and would warrant referral to a physician for further diagnostic consultation.

To examine vergence, the examiner holds a discreet object (finger, pencil) 2 feet away from the patient’s nose and slowly brings it toward the bridge of the nose until the target becomes double. The eyes should converge and the pupils should constrict. Normal convergence occurs at about 4 inches. Patients having difficulty with convergence will experience double vision of the target when it is farther than 4 inches from the bridge of the nose.

**Vestibular Function**

Tests of the functional integrity of the vestibular system are designed to incorporate movements that stimulate vestibular system activity in order to elicit the VOR. As the motor output of the vestibular system is reflected in the oculomotor system, measuring VOR responses will give information about vestibular system function. Initial assessment of VOR function is to have the patient fix the vision on a target while moving the head side-to-side, stimulating the horizontal semicircular canals to elicit a VOR response through the MLF to maintain gaze stabilization. The examiner tilts the patient’s head down 30° to place the horizontal semicircular canals in the horizontal plane of movement, and then slowly moves the patient’s head side-to-side approximately 30° in both directions. The patient can look at the examiner’s nose as the target, while the examiner assesses the patient’s ability to maintain visual fixation on that target. The examiner then assesses the vestibulo-ocular reflex cancellation (VORc). In this test, the patient follows a moving target with the head and eyes, maintaining ocular position in the center of the orbits. This requires input from higher cortical centers to override the VOR.

A more quantitative assessment of VOR is the Dynamic Visual Acuity (DVA) test. In this test, the examiner uses a Snellen eye chart to assess the patient’s baseline visual acuity. The patient can be either sitting or standing, as appropriate, the proper distance from the eye chart, and should wear his or her glasses if needed for distance correction. The patient recites the lowest line (smallest font size) they can read clearly on the chart. The examiner stands behind the patient, firmly grasping the head with both hands on either side of the head, tipping the head down 30° so that the horizontal semicircular canals are aligned in the horizontal plane. The patient’s head is then moved side-to-side at a frequency of 2 Hz (two full side-to-side cycles per second) while the patient recites the lowest line on the Snellen chart he or she can read clearly. If the VOR is functioning normally, the patient’s eyes will move in the opposite direction of head movement at the same
frequency, allowing for visual fixation on the target (eye chart). Normally, a patient’s visual acuity will be somewhat reduced under these conditions as head-eye movement is not a perfect 1:1 relationship, and he or she can demonstrate up to a two-line degradation (increase in font size, moving up on the chart) over the baseline visual acuity line. DVA is abnormal if there is a three-or-more-line degradation, indicating difficulty with gaze stabilization (Herdman & Clendaniel, 2014). It is important for the examiner to maintain a smooth horizontal trajectory of head movement to ensure that any degradation of visual acuity can be attributed to diminished VOR function and not to distortion of the target. Using a metronome, which can be accessed online, will ensure that the test is performed at the proper testing frequency of 2 Hz. It is important to ensure that the frequency is at or above 2 Hz to limit the use of pursuit eye movements, which would be able to track the target at lower frequencies.

The Head Thrust Test (HTT), also called the Head-Impulse Test (HIT), examines VOR function and can identify unilateral or bilateral vestibular loss. In this test, the patient fixes his or her gaze on the examiner’s nose while the examiner quickly rotates the head to one side through a small amplitude of movement, maintaining the end point position while observing for the patient’s ability to maintain visual fixation (see Figure 8). Noting the need for a corrective saccade to refixate vision on the target is indicative of loss of vestibular function on the side to which the head is being turned. Before performing this test, it is important to “clear” the neck by assessing passive range of neck motion, determining any restrictions due to loss of cervical spine mobility, pain, or guarding. This test has been found to have a 35% sensitivity and 95% specificity for detecting unilateral vestibular loss (Harvey, Wood, & Feroah, 1997). Thus, we can interpret a positive finding on the HTT as the patient having loss of vestibular function with 95% confidence. However, it also incurs a high percentage of false negative results (sensitivity). In other words, there is only a 35% chance that the patient does not have loss of vestibular function if the test is negative. The degree of loss has been determined to be a factor in the sensitivity of this test, with sensitivity being as much as 87% in patients with severe loss of vestibular function (Beynon, Jani, & Baguley, 1998). Positioning the patient’s head in a 30° downward tilt to place the horizontal semicircular canals in the plane of motion and making the timing and direction of the head thrust motion unpredictable further improves the sensitivity of the test (Schubert, Tusa, Grine, & Herdman, 2004).

The Head-Shaking Nystagmus Test also assesses imbalance in bilateral vestibular function as seen in the presence of nystagmus after rapidly oscillating the patient’s head in a small amplitude to stimulate the VOR. In this test, the patient’s head is tipped down 30° to position the horizontal semicircular canals in the horizontal plane, the patient’s eyes are closed, and the examiner oscillates the patient’s head 20 times. The patient is then asked to open the eyes immediately upon cessation of the movement while the examiner observes the presence of nystagmus, which indicates a vestibular imbalance. A negative test (no nystagmus) will occur in a normally functioning vestibular system, or when bilateral loss is present as there will be no imbalance between each side to generate the nystagmus. The patient is wearing Frenzel lenses or infrared goggles during this test to obliterate visual fixation, while the examiner can see the patient’s eye movement. Visual fixation would suppress any nystagmus that may be present; thereby the test result would be falsely interpreted as negative or normal. The sensitivity and specificity of the test is similar to
the HTT for determining loss of vestibular function (Harvey et al., 1997).

The patient with acute vestibular syndrome will typically present to the emergency department due to the acute onset and severity of the symptoms. This vestibular presentation must be differentiated from stroke in order for appropriate and timely intervention to take place. The acronym “HINTS” helps in the differential assessment process, using the Head Impulse test, assessing Nystagmus, and Test of Skew. Findings of a negative HIT, the presence of direction-changing nystagmus, and skew deviation has been found to have 100%
sensitivity and 98% specificity in identifying a central cause (Kattah et al., 2009).

Positional Testing

The previous section presented tests that assess the functional integrity of the vestibular system. Positional testing assesses for vestibular system dysfunction due to BPPV (distorted function). The gold standard for diagnosing BPPV of the posterior canal is the Hallpike-Dix test. In this test, the patient is seated on a treatment table with the examiner holding the patient’s head in both hands, positioning the head in 45° of cervical rotation toward the affected ear, and 20° of cervical extension. The patient is then rapidly brought to a supine position with the examiner supporting the head hanging off the end of the table, maintaining the 45° of rotation and 20° of cervical extension. In this position, the affected posterior semicircular canal is placed in a gravity-dependent position, creating movement of the otoliths in the canal (see Figure 9).

If the test is positive, the examiner will see a torsional and upward beating nystagmus toward the ear in the dependent position within 10 seconds, extinguishing within 60 seconds. In conjunction with these findings, the patient must also experience the familiar symptoms of vertigo in order to confirm a positive test. Symptoms persisting for as long as 2 minutes before fatiguing suggest cupulolithiasis as the basis of the BPPV. The patient may experience a brief episode of symptoms upon being brought up to the seated position at the end of the test.

The Roll Test assesses BPPV in the horizontal canal. The patient assumes a supine position and the head is quickly rolled to one side. The examiner assesses for the presence and direction of nystagmus and symptoms of vertigo. The head is then slowly brought back to center, and then quickly rolled to the opposite side with nystagmus and vertigo again being assessed. For unilateral horizontal canal BPPV, nystagmus will be seen with rolls in both directions as the affected ear is still moving in the testing plane. The velocity of the nystagmus must be appreciated to determine the affected ear, and the direction of the nystagmus determines whether the BPPV is due to canalithiasis or cupulolithiasis. If otoconia are free-floating (canalithiasis), the velocity and duration of the nystagmus will be higher when the patient is rolled toward the affected side. If otoconia are adhered to the cupula (cupulolithiasis), the velocity will be higher when the affected ear is up, and the duration persists. In horizontal canal BPPV, the direction of nystagmus is always lateral, but can be either geotropic (fast phase beating toward the ground/earth), or ageotropic (fast phase beating away from the ground/earth). In horizontal canalithiasis, nystagmus is geotropic and fatigues, while with cupulolithiasis nystagmus is ageotropic and does not fatigue. There is no doubt that interpreting findings on the Roll Test can be confusing considering that the examiner must simultaneously diagnose which side is affected and which form of BPPV is present. Using a patient case example to clarify, if the velocity of nystagmus is higher when the patient’s left ear is down (as compared to right ear down) and the direction of the nystagmus determines whether the BPPV is geotropic, then the diagnosis is left horizontal canalithiasis. In contrast, if the velocity is higher when the left ear is down, but the direction is ageotropic, then the diagnosis is right horizontal cupulolithiasis. In more simplified terms, when the patient is in the position that elicits the strongest response, the direction of the nystagmus will point to the affected side, while the geotropic and ageotropic characteristic lends diagnostic value in determining the type of BPPV.

In any positional tests, the examiner must be cautious not to interpret all oculomotor
responses as a positive test of BPPV. Findings of nonfatiguing vertigo or nystagmus while the patient is in the testing position, direction-changing nystagmus when the head is moved to another position, or nystagmus that does not coincide with the plane of the dependent canal (e.g., a pure downbeating nystagmus), indicates a CNS lesion, necessitating referral.

**Postural Control**

As the vestibular system is also responsible for mediating postural control through the VSR, the examiner must employ tests that measure balance and mobility in the patient with vestibular dysfunction who also reports difficulty with balance and walking. Clinical examination should include tests of static and dynamic balance, ambulation, and fall risk.

Static balance is assessed in both sitting and standing, determining the extent to which the patient is able to maintain the position independently. The surface on which the patient is seated or standing (bed, chair, foam/compliant surface, etc.) should also be considered as those features impact the level of challenge of the postural control activity. Tests of dynamic balance assess the ability to weight shift and move the center of mass within and to the edges of the base of support. This can be accomplished

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**FIGURE 9: HALLPIKE-DIX TEST**

1. **Patient’s head**
   - is turned 45 degrees
eastern side to be tested

2. **Patient moved into**
supine quickly

*Note. From Western Schools, 2018.*
through measuring the direction and distance of reaching in both sitting and standing. Again, the surface conditions must be appreciated.

Standardized tests of balance that can be used in patients with vestibular dysfunction include the Berg Balance Scale (BBS), the Four Square Step Test (FSST), and the Modified Clinical Test of Sensory Interaction and Balance (mCTSIB). The BBS is a 14-item tool that assesses both static and dynamic balance in sitting and standing, as well as identifying risk for falls. Items include a transfer, reach, turn, standing in tandem, and standing on one leg, and are scored on a 0 to 4 scale with a maximum score of 56. It has been validated for use in the community-dwelling older adult population (Berg, Maki, Williams, Holliday, & Wood-Dauphinee, 1992). The BBS has been shown to have a sensitivity of 91% for predicting fall risk with a score of $\leq 42/56$ in persons without a history of imbalance, and $\leq 51/56$ in persons with a history of imbalance (Shumway-Cook, Baldwin, Polissar, & Gruber, 1997).

The Four Square Step Test assesses multidirectional dynamic standing balance that is predictive for identifying fall risk. The patient is asked to step over four canes set up like a cross with the tips of the canes touching in the middle. The time it takes for the patient to move through the sequence of stepping over the canes forward, sideways, and backward in a clockwise and then a counterclockwise direction while facing forward is measured. Assessing the ability to make quick changes of direction and walk backward is important for the patient with vestibular disease. Patients taking greater than 15 seconds to complete the test are identified as being at risk for multiple falls with a sensitivity of 94% and specificity of 86% (Ditte & Temple, 2002). A cutoff score of 12 seconds was found to have 80% sensitivity and 92% specificity for identifying fall risk in patients with vestibular dysfunction (Whitney, Marchetti, Morris, & Sparto, 2007).

The mCTSIB assesses the patient’s ability to maintain static standing balance under altered sensory and surface conditions. The four conditions of the test are (1) standing on the floor with eyes open, (2) standing on the floor with eyes closed, (3) standing on a compliant foam mat with eyes open, and (4) standing on foam with eyes closed. The mCTSIB is a modified version of the familiar “Foam and Dome” test (CTSIB), where the two conditions of using a dome to create visual conflict are eliminated. The four conditions systematically alter somatosensory inputs and eliminate visual inputs to determine whether the patient is able to utilize remaining sensory inputs to maintain balance. Patients with vestibular loss often lose their balance under condition 4, where both visual and somatosensory information has been altered, and the patient must rely only on vestibular information to maintain balance. The conditions where the patient is most challenged help to guide treatment strategies. However, this test should not be interpreted as diagnostic for vestibular dysfunction (Herdman & Clendaniel, 2014).

The Timed Up and Go test (TUG) is a measure of functional mobility and has been validated to predict fall risk in patients with vestibular disease. The time it takes for a patient to rise from a standard chair, walk 3 meters, turn, and return to sitting is measured. Generally, community-dwelling adults taking greater than 13.5 seconds to complete the test are identified as having a risk for falls (Shumway-Cook, Brauer, & Woollacott, 2000). The TUG has a sensitivity of 80% and specificity of 56% for identifying fall risk for patients with vestibular disease who take greater than 11.1 seconds to complete the test (Whitney, Marchetti, Schade, & Wrisley, 2004).
Other standardized assessments of postural control incorporate functional tasks during locomotor activity, such as walking with head turns and negotiating obstacles, and provide a scoring system to quantify the degree to which the patient is able to maintain postural stability while performing these functional mobility tasks. The Dynamic Gait Index (DGI) and the Functional Gait Assessment (FGA) are two such tests that are useful to measure postural control in patients with vestibular disease. The DGI assesses the patient’s ability to maintain balance during walking with head turns, negotiating obstacles, and changing direction; speed of walking is measured. Eight items are scored on a 0 to 8 scale with a maximum score of 24. A DGI score of less than 19/24 was found to be able to predict fall risk in patients with vestibular disease (Whitney, Hudak, & Marchetti, 2000).

The FGA is a modification of the DGI, including more challenging tasks to reduce the ceiling effect seen in the DGI for higher functioning individuals with balance limitations. The FGA has 10 items scored on a 0 to 3 scale, with a maximum score of 30 points. Some of the modifications include walking backward and with eyes closed, which is particularly difficult for persons with vestibular dysfunction. The DGI has been validated for use in patients with vestibular dysfunction (Wrisley, Marchetti, Kuharsky, & Whitney, 2004) with a score of 22/30 or less to be 100% sensitive and 72% specific in predicting fall risk in the community-dwelling adult population (Wrisley & Kumar, 2010). This test can also provide information to help the clinician develop treatment strategies based on the components of functional mobility found to be most difficult for the patient.

Observational gait assessment is another important component of examination of postural control. The presence of deviations such as asymmetrical step length, slowed cadence, guarded movements, diminished reciprocal movement (en-bloc), widened base of support, or veering from a straight path are all indicative of difficulty with postural control.

**Patient Self-Report Measures**

Self-report measures are standardized assessment tools that aim to quantify the degree of limitation the patient is experiencing. Measurement of health status regarding level of function, disability, and quality of life provides valuable insight to understanding the impact a condition has on the individual patient, and helps the clinician develop a tailored intervention program to address those individual barriers to recovery. Information from self-report measures should be cross-referenced with information gathered on clinical examination to create a comprehensive clinical picture, and because the patients’ perception of their condition can lead to over- or underestimating their level of limitations. These outcome measures can offer information regarding the amount of change the patient has experienced in self-management, returning to their previous roles, and quality of life through rehabilitation.

Self-report measures can be generic, in that they measure general function or quality of life in any patient group. Examples of such measures are the Short-Form-36 Health Survey (SF-36) and the Functional Independence Measure (FIM). The limitation of using generic measures in patients with vestibular disease is that they are not sensitive enough to capture the impact of the patients’ condition on their everyday lives. For that reason, condition-specific outcome measures are used. These measures often assess different aspects of disability, so the clinician’s selection of specific tools should be guided by the measurement properties of the tool as well as the particular domains of the condition the tool aims to measure. Self-report measures widely
used by physical therapists in patients with vestibular disease include the Motion Sensitivity Quotient (MSQ), the Vertigo Symptoms Scale (VSS), the Dizziness Handicap Inventory (DHI), the Vertigo Handicap Questionnaire (VHQ), the Vestibular Disorders Activities of Daily Living (VADL) scale, the Falls-Efficacy Scale (FES), and the Activities-specific Balance Confidence (ABC) scale.

**Measures of Symptom Level**

The MSQ and VSS are tools that measure the degree of symptoms the patient is experiencing. The MSQ quantifies the severity of symptoms provoked by stereotypical movements that were designed for treatment. Seventeen movements such as sitting to supine, coming up to sit from forward bending, and standing and turning, which are typically provocative in patients with vestibular dysfunction, are assessed in terms of the patient’s report of intensity and duration of symptoms. The calculated percentage score provides a level of severity of sensitivity to motion. Items on the MSQ are used to guide treatment programs and document outcomes (Shepard, Telian, & Smith-Wheelock, 1990; Smith-Wheelock, Shepard, & Telian, 1991).

The VSS assesses the relationship of emotional and anxiety symptoms with vestibular symptoms. The scale consists of 36 items that ask the patient how often they experienced particular symptoms in the past 12 months. Tally scores of subscale items help to discriminate patient symptoms due to severity of vertigo, autonomic symptoms, and severity of symptoms due to somatic anxiety. The vertigo subscale scores were found to be well correlated with presence of vestibular disease, and anxiety subscale scores were significantly correlated with other measures of anxiety (Yardley, Masson, Verschuur, Haake, & Luxon, 1992; Yardley, Todd, Lacoudraye-Harter, & Ingham, 1992).

**Measures of Activity Restriction**

The DHI, VADL, and ABC are all measures that focus on determining the level of limitation of activity in daily life in patients with vestibular dysfunction. The DHI is the most widely used self-assessment tool in patients with vestibular disorders. It assesses the emotional, functional, and physical impact of vestibular disease over 25 items using a 3-point scale. The total score is obtained by summing the subscale scores, to a maximum score of 100 points, with higher scores indicating more severe disability (Jacobson & Newman, 1990). Although the DHI cannot guide treatment because it does not indicate specific tasks or activities that cause the patient’s symptoms, it has been shown to be well correlated with level of functional impairment as measured by the Five Times Sit-to-Stand test, ABC, and incidence of falling (Whitney, Wrisley, Brown, & Furman, 2004).

The VADL assesses the impact of vestibular dysfunction on activities of daily life. It uses 28 items to measure level of disability in the domains of basic self-care, mobility, and instrumental activities of daily living (Cohen & Kimball, 2000). The items on the VADL do not address the symptoms or emotional component of vestibular dysfunction, but rather focus on domains of function. The VADL has been shown to be moderately correlated with the DHI, and is able to detect changes in function after vestibular rehabilitation (Cohen & Kimball, 2003), although the Minimally Clinically Important Difference (MCID) psychometrics have not been established.

The FES assesses fear of falling during activities of daily living in the older adult population. The 10-item scale asks patients to rate their confidence in being able to perform activities without falling. Activities rated include getting out of bed, reaching into cabinets, and...
getting dressed (Tinetti, Richman, & Powell, 1990). The FES is an appropriate tool to use for the frail older adult, but scores have been shown to skew toward 100% confidence in the community-dwelling older adult due to the low physical demand of the items scored (Maki, Holliday, & Topper, 1991).

The ABC was established to assess self-perceived balance confidence in performing 16 common daily activities in the community-dwelling older adult population (Powell & Myers, 1995). The stem question asks patients to rate their level of confidence (“How confident are you that you will not lose your balance or become unsteady…”) in performing activities such as walking up and down the stairs, getting on and off an escalator, and walking in a crowded mall. The average of all scores is calculated, with higher percentages indicating greater confidence in balance. The ABC has been extensively studied, and has been found to have good correlation with the FGA (Wrisley & Kumar, 2010), and is strongly correlated with the BBS and TUG in the community-dwelling older adult population (Hatch, Gill-Body, & Portney, 2003). In patients with vestibular dysfunction, the ABC has also been shown to have excellent correlation with the DHI (Whitney, Hudak, & Marchetti, 1999) and good correlation with the DGI (Marchetti, Whitney, Redfern, & Furman, 2011). A cutoff score of 67% (and below) has been established to predict fall risk in the community-dwelling older adult population with 84.4% sensitivity and 87.5% specificity (Lajoie & Gallager, 2004). An important note to clinicians is the finding that although the ABC is intended to be administered as a self-report measure, it has been determined that patients find the stem questions difficult to interpret, responding in terms of whether they perform the activity, rather than their level of confidence in the event that they would encounter the activity. Thus, studies have determined that this tool would be best administered by interview (Hatch et al., 2003; Powell & Myers, 1995).

MEDICAL EXAMINATION OF VESTIBULAR DYSFUNCTION

In addition to the “bedside” clinical examination of vestibular function, there are a number of vestibular function tests that are performed in a laboratory setting. These are caloric tests, rotational chair testing, and vestibular evoked myogenic potentials. The caloric exam is considered part of the “gold standard” for identifying unilateral peripheral vestibular hypofunction. While clinical tests for peripheral vestibular hypofunction involve head movements, which essentially assess both sides simultaneously, caloric tests can determine the side of the deficit because each side is tested separately. These tests systematically introduce warm and cold air or water into the external auditory canals, creating a temperature gradient across the horizontal canal and subsequent endolymph flow. Warm temperatures cause an excitation response, generating lateral nystagmus with the fast phase beating toward the test ear. Conversely, cool temperatures create an inhibitory response, generating a lateral nystagmus away from the test side. A reduced or absent response (no nystagmus) is indicative of vestibular hypofunction or loss. The caloric test has inherent limitations. The first is that it stimulates only horizontal canal function, and thus assesses afferent activity only from the superior vestibular nerve. It also generates a low frequency of stimulation; thus it may not accurately measure vestibular function. Finally, it is not well tolerated by many patients (Jacobson & Shepard, 2008).
Rotational chair testing is another “gold standard” test to identify bilateral vestibular hypofunction. This is a sophisticated test to measure VOR gain (ratio of head-eye movement), VOR symmetry (right and left rotations), and VOR phase (relationship of head and eye position). In this test, the patient is securely seated in a mechanical chair that will rotate at a variety of velocities, typically 60°/second and 240°/second. Electromyogram (EMG) recordings track eye movement both during rotation and upon stopping to measure VOR metrics, which identifies presence and degree of vestibular hypofunction on each side. As with caloric testing, the rotational chair test only measures components of horizontal canal function.

Vestibular evoked myogenic potentials (VEMP) measure otolith function. The neurophysiological basis for VEMP testing starts with the knowledge that otoliths can be stimulated by vibration and high-level acoustical inputs. The cervical vestibular evoked myogenic potential (cVEMP) test uses loud clicks to evoke stimulation of the sacculus and inferior vestibular nerve. Thus, sound-evoked otolith activity will elicit postural responses through the VSR pathway. The EMG activity of the sternocleidomastoid muscles are used as a measure of otolith function. In the ocular VEMP (oVEMP), vibration over the center of the forehead is used to stimulate utricle activity, resulting in an excitatory response of the inferior oblique muscles (Jacobson & Shepard, 2008).

Although these tests provide more sophisticated and diagnostic information regarding vestibular function, they are not performed routinely given the need for highly specialized equipment and highly trained staff, and the time-consuming nature of the tests. However, the clinician working with patients with vestibular disease must be able to appreciate the diagnostic value of these test results that may be part of the patient’s initial work-up.

**EVIDENCE-BASED REHABILITATION FOR PERSONS WITH PERIPHERAL VESTIBULAR DYSFUNCTION**

The goals of treatment for the patient with peripheral vestibular dysfunction are to reduce dizziness, improve gait and balance dysfunction, improve gaze stabilization, increase activity level, and reduce disability. Treatment should follow a problem-oriented approach so that the patient receives an individualized program of care, thereby optimizing his or her recovery. Developing a problem-oriented approach to care takes into account the patient’s diagnosis and category of vestibular dysfunction (i.e., reduced function, distorted function), the problem areas identified during the examination, and the patient’s medical history. Thus, the patient with anxiety and resulting peripheral vestibular hypofunction due to repeated attacks of Ménière’s disease, the patient with BPPV, and the patient with unilateral vestibular hypofunction will each have a different treatment plan.

**BPPV**

Treatment of BPPV will depend on whether the otoconia are free-floating in the semicircular canal or adhered to the cupula, based on examination findings. Canalith repositioning maneuvers (CRM) have been shown to be effective treatments for canalithiasis and cupulolithiasis (Casani, Vanucci, Fattori, & Berrettini, 2002; Gans & Harrington-Gans, 2000; Hilton & Pinder, 2002; Nunez, Cass, & Furman, 2000; Steenerson, Cronin, & Marbach, 2005). Repositioning maneuvers aim to move the otoconia out of the affected canal and back to the utricle to eliminate their effect on endolymph flow and resulting provocation of vertigo with head movement. Repositioning maneuvers to treat posterior and anterior canal BPPV are
the Epley maneuver for canalithiasis and the Semont (or Liberatory) maneuver for cupulolithiasis. For horizontal canal BPPV, the maneuvers are the Barbeque Roll and the Appiani maneuver for canalithiasis and the Casani (modified Semont) maneuver for cupulolithiasis.

**Posterior and Anterior Canal**

The Epley maneuver for posterior canalithiasis moves the patient through a series of positions while lying on the treatment table, to move the otoconia through the posterior semicircular canal. The position of the head and the direction of roll that are the main components of this CRM take into account the anatomical orientation of the posterior canal. The first position of the Epley maneuver begins in the Hallpike-Dix position, with the patient in supine, the head rotated 45° to the affected side (affected side down) and in 20° of cervical extension off the end of the examination table. The clinician must be certain to quickly guide the patient down into position from a long-sit position, avoiding a slow transition, which may not effectively move the otoconia. The patient maintains that position, with the therapist supporting the patient’s head, for at least 30 seconds, or twice as long as the duration of the nystagmus and vertigo. The second position is simply guiding the patient’s head to roll to 45° of rotation to the other side, maintaining 20° of cervical extension. This position is also maintained for at least 30 seconds, or twice the duration of the nystagmus and vertigo. For the final position, the patient rolls to the side of cervical rotation while the therapist maintains the 45° of cervical rotation. This results in the patient in a sidelying position on the unaffected side, with the head turned with the nose to the floor, while the therapist is supporting the head. Again, this position is maintained for at least 30 seconds, or twice the duration of the nystagmus. Finally, the patient moves into a short-sit position over the edge of the mat, while keeping the chin tucked (see Figure 10). Treatment of anterior canalithiasis using the Epley maneuver is exactly the same as for the posterior canal, with the affected ear down as the first treatment position.

At the completion of the Epley canalith repositioning maneuver, the Hallpike-Dix can be repeated to determine whether there are any remaining symptoms of BPPV, and thus residual otoconia in the posterior canal. In that case, the Epley maneuver can be performed another time, but should not be repeated more than three times in the same session.

The Semont (or Liberatory) maneuver is used to treat posterior canal cupulolithiasis. In this treatment, the patient sits on the edge of a treatment table with the head rotated 45° toward the unaffected side (away from the affected side). From there, the patient is moved quickly into a sidelying position on the affected side in a nose-up position (see Figure 11). The rapid movement into this position aims to dislodge the otoconia from the cupula. In this position, the patient will be lying with the affected side down, in a nose-up position, with 20° of cervical extension. The posterior canal will be positioned perpendicular to the horizon. The patient remains in this position for 1 to 2 minutes, allowing the otoconia to migrate to the lowest point in the canal with respect to gravity (the upper portion of the canal) and is then moved rapidly through the initial sitting position to the opposite side, without turning the head, coming to sidelying on the opposite side in a nose-down position. This brings the otoconia around and out of the posterior canal. The patient holds this position for 2 minutes and then slowly returns to a sitting position. There are no post-treatment restrictions or precautions for either of these maneuvers.

The Semont maneuver is also used to treat anterior canal cupulolithiasis, with the starting position the only difference. The starting
position for the anterior canal is still sidelying on the affected side, but the head is rotated toward the affected side, resulting in a nose-down position. As with the posterior canal repositioning maneuver, the patient remains in this position for 1 to 2 minutes, allowing the otocoria to migrate to the lowest point in the canal with respect to gravity (the upper portion of the canal) and is then moved rapidly through the initial sitting position to the opposite side, without turning the head, coming to sidelying on the opposite side in a nose-up position.

**Horizontal Canal**

CRM for horizontal canalithiasis has become known as the “Barbeque Roll” because the original maneuver resembled the movement around a barbeque spit. The initial technique had the patient rolling on the bed 360° in the direction away from the affected ear, hence the
analogy of the barbeque spit. This technique has since been modified to primarily rotate the head through a 270° (3/4) motion, eliminating the need for the patient to perform a full 360° roll. However, the original 360° roll can be done if the patient lacks sufficient cervical mobility, as it maintains a neutral head-on-body alignment. The Barbecue Roll technique starts with the patient in supine, with the affected ear down. The patient or therapist then rotates the head to midline (nose up), then to the unaffected side. Finally, the patient rolls to prone with nose pointing to the floor and the chin tucked into 30° of cervical flexion before coming to sit. The result of this sequence is the rotation of the head through a 270° arc of motion. Each position is maintained for 30 seconds, or until the dizziness stops, in order to allow the otoconia to move through the canal.

If the patient is unable to tolerate the movements or positions of either version of the Barbeque Roll technique due to pain or difficulty or inability in assuming any of the positions, an alternate treatment is the Appiani maneuver. In this maneuver, the patient is brought quickly down to sidelying on the unaffected side (affected side up) with the therapist cradling the patient’s head, and remains in that position for 2 minutes to allow otoconia to settle in the gravity-dependent position in the posterior aspect of the horizontal canal. Once 2 minutes have elapsed, the therapist then rotates the head quickly 45° toward the table (nose down), flushing the otoconia out of the canal. That position is maintained for 2 minutes. The patient can then move to a seated position while maintaining the 45° head-rotated position, and can resume...
midline head orientation once he or she comes to a full upright sitting position (see Figure 12).

For the patient with horizontal canal cupulolithiasis, the Casani maneuver has been found to be an effective treatment. The Casani maneuver is a modification of the Semont liberatory maneuver used in posterior canal cupulolithiasis. It is similar to the Semont position in that the patient is brought quickly into a sidelying position on the affected side to dislodge the otoconia from the cupula, but in this maneuver there is no initial cervical rotation. The head is then rotated quickly 45° toward the floor, and the patient remains in that position for 3 minutes before returning to a seated position. Although this maneuver looks very similar to the Appiani maneuver, there are two distinct differences between them. First, the Appiani is indicated for horizontal canalithiasis, while the Casani is used to treat horizontal cupulolithiasis. Second, the Appiani maneuver starts with the unaffected ear down (affected ear up; Appiani, “up”), while the Casani starts with the affected ear down. Both move the head into 45° of cervical rotation toward the table (see Table 2). Appreciation for the position of the semicircular canals along a 3-dimensional axis of motion will help elucidate the mechanics of movement of debris through each position.

**Vestibular Hypofunction**

As previously discussed, spontaneous rebalancing of tonic inputs occurs within 2 weeks...
for the patient with altered vestibulo-ocular and vestibulospinal responses due to disruption of function in CN VIII or the otoliths. Patients presenting with residual deficits in postural control, gaze stabilization, and symptoms of dizziness due to vestibular hypofunction require a program of vestibular rehabilitation. A wealth of studies demonstrates that recovery of the disturbances due to loss of peripheral vestibular function is dependent on activities that incorporate visual inputs and head and body movement (Gill-Body, Krebs, Parker, & Riley, 1994; Herdman, 1998; Herdman, Blatt, & Schubert, 2000; Herdman, Clendaniel, Mattox, Holliday, & Niparko, 1995; Herdman, Schubert, Das, & Tusa, 2003; Whitney & Rossi, 2000), which is the basis for vestibular rehabilitation. Vestibular rehabilitation programs should include habituation exercises, adaptation exercises as warranted for patients with impaired gaze stabilization, balance retraining, and exercises to restore or maintain physical conditioning.

### Table 2: Canal Repositioning Maneuvers for BPPV

<table>
<thead>
<tr>
<th>Maneuvers</th>
<th>Canal</th>
<th>Initial Position</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epley</td>
<td>Anterior or Posterior Canalithiasis</td>
<td>Patient in supine with 20° neck extension and 45° head rotation to affected side</td>
<td>2 subsequent positions: head rotation to opposite side, then continue with trunk and head rotation, then to sit</td>
</tr>
<tr>
<td>Semont</td>
<td>Posterior Cupulolithiasis</td>
<td>Affected side down, 45° head rotation away from affected side (nose up)</td>
<td>Move quickly to lying on unaffected side (through returning to sit), without rotating head (nose down)</td>
</tr>
<tr>
<td>Semont</td>
<td>Anterior Cupulolithiasis</td>
<td>Affected side down, 45° head rotation toward affected side (nose down)</td>
<td>Move quickly to lying on unaffected side (through returning to sit), without rotating head (nose down)</td>
</tr>
<tr>
<td>Appiani</td>
<td>Horizontal Canalithiasis</td>
<td>Sidelying on unaffected side for 2 minutes</td>
<td>Quick 45° cervical rotation toward unaffected side (nose down)</td>
</tr>
<tr>
<td>Casani (aka Modified Semont)</td>
<td>Horizontal Cupulolithiasis</td>
<td>Sidelying on affected side without cervical rotation</td>
<td>Immediate quick 45° cervical rotation toward affected side (nose down). Maintain 3 minutes.</td>
</tr>
<tr>
<td>Barbeque Roll (270° roll)</td>
<td>Horizontal Canalithiasis</td>
<td>Supine, head turned toward affected side</td>
<td>3 subsequent steps: head roll to opposite side, patient rolls to side, patient rolls to prone</td>
</tr>
</tbody>
</table>

*Note. From Western Schools, 2018.*

### Habituation Exercises

Habituation exercises are performed to reduce symptoms of motion-provoked dizziness or imbalance. The underlying principle of habituation exercises is to reduce the CNS response to normal movement stimulus, or “habituating,” driving compensation in the CNS. Any head motion that the patient finds symptomatic, such as bending forward or walking with head turns, can be used as a habituation exercise. However, a habituation program should also incorporate the items in the MSQ, as the MSQ is composed of the most common activities that provoke dizziness in patients with peripheral vestibular loss, and is a valuable tool to document recovery (Shepard et al., 1990; Smith-Wheelock et al., 1991). The patient starts with 4 to 5 of the activities on the MSQ that moderately provoke their symptoms, repeating three times and performing them two to three times daily, resting between each exercise to allow the symptoms to return to baseline. Habituation is characterized by decreased intensity and duration of symptoms in response to
performing the exercise. Once patients can tolerate an activity, with minimal or no symptoms, they can be progressed to other items on the MSQ that they found more provocative. The clinician should be careful not to overprescribe habituation exercises at the risk of overstimulating the patient. In order to drive CNS neuroplasticity and habituation, exercises must provoke symptoms, but symptoms should resolve within a few minutes. Symptoms lasting an hour or more necessitate a review of how the patient is performing the exercises and program modification. The patient may need to perform exercises for a few months before being able to modify the program, and may take as long as 6 months for habituation to take place (Herdman & Clendaniel, 2014; Smith-Wheelock et al., 1991).

**Gaze Stabilization (Adaptation) Exercises**

Gaze stabilization exercises, also called “adaptation exercises,” have been found to be effective in restoring the VOR gain (1:1 ratio of head-eye movement) in patients with vestibular dysfunction who are experiencing blurred vision or dizziness when performing activities that require visual tracking during head movement (Hillier & McDonnell, 2011). Not all patients with vestibular loss will experience difficulties with gaze stabilization, but positive findings on DVA testing or symptoms of blurred vision while walking or scrolling text on their computer screen are indicative of diminished gaze stabilization. Gaze stabilization exercises require the patient to maintain visual fixation on a target while moving the head. Visual fixation is mediated by the CNS. During visual fixation on a stationary object, any slip of the image on the retina due to drifts in ocular position will cause the brain to generate a responsive eye movement to hold the image steady on the retina. The retinal “slip” of the image on the fovea during head motion due to the altered VOR gain induces centrally mediated changes in interpretation of vestibular signals from the residual vestibular function. The retinal “slip” produces an error signal, and the CNS compensates to decrease the error signal by increasing the VOR gain. Retinal slip can be induced by both horizontal and vertical head movements applied at various amplitudes and frequencies to allow for adaptive changes to occur in the VOR gain. These changes, or adaptation, are the basis for restoring adequate VOR function.

The first level of the exercise is termed X1 (“times one”) viewing. It has the patient fix gaze on a discreet target, such as a card with a single letter on it, which is held at arm’s length. The patient moves his or her head in a small trajectory (20° to 30°) from side to side as fast as possible while maintaining a clear focus on the target (see Figure 13). He or she should continue the motion for 1 minute without stopping, modulating the speed of the head movement to perform the activity for the full minute. The tempo should also be reduced if the patient experiences any extreme nausea or dizziness. This should be performed 3 times with a 1-minute rest in between to allow symptoms to resolve to baseline, 3 to 5 times per day. Care should be taken not to increase the patient’s dizziness so that it becomes persistent for greater than a few minutes. The goal is to work up to 2-minute increments. This activity can start in a sitting position, and when able, progressed to a standing position. The level of challenge can also be increased by having the patient perform the activity with the target taped to a blank wall 6 feet away, and then adding a visually complex field, such as a checkerboard, on the wall while the patient holds the card at arm’s length.

Once patients tolerate the X1 viewing paradigm, they can then progress to the X2 viewing paradigm, where the patient’s head and the target are moving together in opposite directions. In this exercise, the patient holds the card with the letter target at arm’s length and moves the
card and the head from side to side in a small trajectory in opposite directions, maintaining a clear focus on the image (see Figure 13). As with X1 viewing, the patient performs this exercise 3 to 5 times per day for 1 minute, as fast as possible while still maintaining a clear focus, eventually working up to 2 minutes. This exercise can be progressed to standing, and then standing with a complex visual background as in the X1 viewing exercise.

**Balance Training**

A problem-oriented approach to balance retraining should be directed at the particular
areas of postural control that were found to
be impaired on examination and through the
patient subjective report. Postural control is
achieved through the interplay of effective input
and interpretation of sensory afferent informa-
tion from visual, somatosensory, and vestibular
systems, with effective motor output. Balance
retraining should take into account
• addressing static, anticipatory, and reaction-
ary postural control;
• increasing the excursion of the center of
mass in all directions in sitting and standing;
• walking in various environmental contexts
such as busy environments, over different
surface conditions such as compliant and
uneven surfaces, and in multiple directions;
• adding a cognitive task to increase the chal-
lenge (split attention) and train mobility in a
functional context; and
• altering the reliance on visual and somato-
sensory inputs.

The aim of static postural control is to main-
tain the center of mass (COM) within the base
of support (BOS). The larger the BOS, the more
easily static stabilization is achieved. Static pos-
tural control training should incorporate tech-
niques, such as manual resistance in alternating
agonist-antagonist muscle groups, that facilitate
smooth coordination of agonists and antago-
nists. The aim is to create muscle co-contraction
around a joint or body segment in sitting and
standing, leading to stable holding of positions.

Dynamic postural control includes both antici-
patory and reactionary strategies. Successful
dynamic postural control requires that sensory
inputs provide accurate information about move-
ment through space to allow the CNS to create
an effective motor plan to maintain balance.
Training anticipatory postural control requires
maintenance of upright orientation during move-
ment of the COM within and to the edges of the
BOS. Activities that displace the COM, such
as reaching, lifting, raising the arms overhead,
shifting weight acceptance from leg to leg, and
walking all utilize anticipatory postural con-
trol. The patient’s balance must be challenged
in order for physiological changes to take place.
Increasing the direction, degree, and smooth
control of COM excursion and reducing reliance
on external support are important components of
anticipatory postural control training.

Reactionary postural control refers to pos-
tural adjustments that are made in response to
unexpected perturbations. These postural adjust-
ments are stereotypical movement strategies
known as ankle, hip, and stepping strategies,
mediated by synergistic muscle activation pat-
terns. The CNS uses information from sen-
sory inputs and muscle stretch receptors about
quick unplanned movement to elicit a reflexive
response. The particular strategy employed is
based on the surface conditions, direction of
movement, and the degree of loss of balance.
An ankle strategy will be employed during small
amplitude movements while standing on a firm
surface, creating a net inverted pendulum move-
ment over the ankles to maintain vertical ori-
entation of the body with respect to gravity. An
example of this would be standing on a slowly
moving train. A hip strategy will be elicited
when an ankle strategy is not sufficient, such as
when there is a large anterio-posterior displace-
ment of COM, or when the surface conditions
are not conducive to utilizing an ankle strategy
(standing on a narrow or compliant surface). In
this case, the person will exhibit a forward and
backward sway of the trunk over the hips. A
stepping strategy is elicited when a large ampli-
tude or velocity of movement brings the COM
outside of the BOS, necessitating a step to restore
the BOS under the COM. This is what happens
when the train makes an abrupt stop or acceler-
ation (Shumway-Cook & Woollacott, 2011).
Although the mCTSIB is not a diagnostic test, the conditions under which the patient exhibits difficulty maintaining postural control can guide treatment by providing insight into which sensory cues the patient relies upon and may not be utilizing effectively. The patient with vestibular dysfunction will tend to rely on other sensory inputs for postural control, with reliance on vision the most predominant. Altering sensory cues by having patients close their eyes or stand on a compliant foam surface will systematically reduce reliance on vision and somatosensation and facilitate more effective interpretation and utilization of vestibular inputs. However, the clinician must take care not to obliterate sensory inputs on which the patient is reliant due to damage to other sensory systems caused by injury or disease. For example, the patient with impaired somatosensation due to peripheral neuropathy will require both visual and vestibular information for postural control. Patients with profound vestibular loss will have difficulty with conditions 3 and 4 on the mCTSIB (eyes closed, standing on floor; eyes closed, standing on foam, respectively) as they will rely on somatosensory and visual inputs to compensate for loss of vestibular inputs. Under these circumstances, the treatment program takes on an accommodative approach, rather than a restorative approach, training increased use of visual cues for postural control in the absence of effective vestibular inputs. In these cases, the patient should have full use of visual inputs, performing activities with eyes open and in well-lit environments. The clinician gains an understanding of the integrity of the visual, somatosensory, and vestibular systems through comprehensive impairment-level examination strategies.

The conceptual framework for balance training is simply summarized here, intended as a review for the practicing clinician. However, a detailed approach to balance training lies outside of the scope of this course, but can be found in textbooks dedicated to that aspect of rehabilitation care for those who desire further edification (Bronstein, Brandt, Woollacott, & Nutt, 2004; Shumway-Cook & Woollacott, 2011).

**Aerobic Conditioning**

Rehabilitation programs for patients with vestibular dysfunction should include some component of aerobic conditioning because many patients will reduce their movement and activity level to manage their vestibular symptoms. Incorporating a physical conditioning program will not only manage or prevent deconditioning, but it will provide functionally relevant balance and vestibular system challenges as the patient moves through various environments, over different terrains, and utilizing different speeds of movement to conform to different environmental demands, such as crossing a street or navigating a crowded sidewalk. Prescribing a walking program four or more times per week is a good place to start. The duration is determined by the patient’s baseline ability. The program should begin with less challenging environments (flat surfaces, quiet areas, etc.) and gradually progress to more challenging environments (outside, busy malls, etc.), eventually incorporating recreational activities such as golf or tennis on a gradual basis. As with any rehabilitation program, safety is of utmost importance. Patients who wish to return to swimming must not swim alone initially, and must be advised that ocean swimming poses a challenge of not having an adequate sense of orientation due to lack of visual and somatosensory inputs to determine where one is in relation to gravity, or a sense of the direction of “up.” Biking may not be advisable until the patient can demonstrate higher-level balance skills due to the limitations of somatosensory and visual cues to orient the patient to upright and the
advanced postural control required to maintain balance while on the bike.

**SUMMARY**

The adoption of direct access legislation across the nation and the transition to a doctoring profession places greater responsibility on the physical therapist toward autonomy in practice. Physical therapists must be able to utilize an in-depth knowledge of human systems anatomy and physiology along with scientific evidence to be able to make decisions effectively about when to treat and when to refer. Most importantly, physical therapists need to be able to determine when physical therapy care is the most efficacious management approach. For patients with vestibular disorders, physical therapy care is central to the practice of vestibular rehabilitation. This course provided a foundation for the practicing physical therapist to better understand effective management of common peripheral vestibular disorders.

Effective management of vestibular disease starts with a solid understanding of the functional anatomy of the vestibular system as a basis of determining the source of the vestibular dysfunction. Use of the evidence to foster accurate interpretation of examination findings further facilitates differential diagnosis. Physical therapists must also provide evidence that the patient is improving to support our recommendations and to establish medical necessity for skilled care. Much of what was presented in this course emphasized these main areas to guide the clinician’s diagnostic process and use of outcome measures. Treatment of vestibular dysfunction is a targeted approach, based on the specific vestibular pathology and the patient’s individual limitations and functional needs. Exercises specific to vestibular system rehabilitation to promote vestibular adaptation and habituation were presented, with a review of the principles of gait and balance retraining as a basis to facilitate immediate application to clinical practice.

Patients with dizziness seek care across multiple disciplines and across the continuum of care settings. The source of their symptoms of dizziness, and difficulties with ambulation and balance can be due to peripheral vestibular dysfunction, CNS lesions, or other nonvestibular etiologies. As movement specialists, physical therapists play a pivotal role in the rehabilitation of all of these conditions. The focus of this course was to provide physical therapists with advanced knowledge of this highly specialized system, to expand their practice and to further augment their management of patients with balance dysfunction.

**CASE STUDIES**

**Case Study 1**

The patient is a 42-year-old male who presents with a history of persistent vertigo that lasted for a few days without any eventful onset. Now that the persistent symptoms have subsided, the patient is noting transient vertigo with bending forward. His past medical history (PMH) is unremarkable.

Examination: Normal sensorimotor and coordination testing, normal oculomotor testing. HIT is positive to the right. Right Hallpike-Dix elicits upward and right torsional nystagmus for 25 seconds. Once that fatigues, a left beating nystagmus is seen. Left Hallpike-Dix demonstrates persistent left beating nystagmus without a torsional component.

**Questions**

1. What is your interpretation of these findings?
2. What is an appropriate plan of care for this patient?
Responses

1. Interpretation: The patient’s initial history of vertigo is suspect for vestibular neuritis. The positive HIT to right confirms right unilateral vestibular hypofunction. Given that vestibular neuritis can result in posterior canal BPPV due to otolith degeneration, the positive right Hallpike-Dix test is not a surprising finding. The persistent left lateral nystagmus may present as a confounding finding. However, recall that vestibular neuritis typically affects the superior portion of the vestibular nerve, which can result in horizontal canal paresis. The Hallpike-Dix position can stimulate horizontal canals, generating directional nystagmus from asymmetry in horizontal canal inputs.

2. The BPPV should be treated first with an Epley maneuver because it is the source of the patient’s primary complaint, and easily treatable. If movement-provoked vertigo does not subside, reassess vestibular function by the HIT and initiate habituation activities as warranted.

Case Study 2

The patient is a 32-year-old female stockbroker who presents to physical therapy as referred by her primary care provider. She has had persistent vertigo and imbalance for the past few days and is finding it difficult to perform her job; she becomes severely dizzy and nauseated when trying to read the stock ticker boards on the floor of the stock exchange. She denies prior illness or injury. Her PMH is unremarkable.

Examination: Normal sensorimotor examination noted. Oculomotor examination with Frenzel lenses reveals right beating nystagmus with central gaze, which increases with gaze to the right. Vestibular testing reveals a positive HIT on the left. Gait assessment is normal, with mild loss of balance with head turns to the left. mCTSIB reveals increased sway on conditions 3 and 4 without loss of balance. DVA testing reveals a 4-line degradation. DGI score is 23/24 with difficulty walking with head turns.

Questions

1. What is your interpretation of these findings?
2. What is an appropriate plan of care for this patient?

Responses

1. The HIT and DVA are the most revealing findings for this patient, indicating left unilateral vestibular hypofunction. Her oculomotor exam reveals second-degree nystagmus that follows Alexander’s law, and is suppressed by visual fixation – all indicative of a peripheral vestibular deficit. Further, the finding of second-degree nystagmus is consistent with the timing of onset of her symptoms. The fact that her balance tests (DGI and mCTSIB) did not strongly identify balance deficits can be explained by the fact that her age and lack of sensory or motor deficits provide her with redundant resources to compensate adequately for her vestibular deficits to maintain postural control. Her deficit in gaze stabilization is her chief limitation at this point, making it difficult for her to perform her job.

2. Focus on adaptation exercises, starting with X1 viewing, and progressing to X2 viewing. Habituation exercises should also be prescribed, focusing on walking with head turns because this activity was found to be most challenging. The patient should perform these exercises daily, and be followed once weekly for reassessment and progression.
Case Study 3:

The patient is a 70-year-old community-dwelling male with a chief complaint of difficulties with balance and occasional dizziness on getting out of bed. His balance has been slowly worsening over the past 2 years, and he has started using a cane for his balance, especially when walking to the bathroom at night. PMH is remarkable for cervical spinal stenosis, changes in hearing with recent hearing aids, and hypertension for which he takes Lisinopril. The patient wears bifocal lenses. He denies a history of falls, aural fullness, and tinnitus. He has no recent medical events, swims regularly at the local YMCA, and lives in a private house with his wife.

Examination: Sensorimotor and musculoskeletal examination is unremarkable except for cervical extension limited to 15° by pain and stiffness. Position changes were without orthostatic hypotension (stable BP). Gait was normal on level surfaces. Oculomotor examination is within normal limits. Vestibular testing reveals a positive HIT bilaterally and a negative Head-Shaking Nystagmus test. Due to cervical ROM limitations, the Hallpike-Dix test position required modification, and the patient was tested on an exam table with the foot of the table elevated slightly so that the patient’s head was extended at 20° from the horizon. Hallpike-Dix was positive on the left with upward beating and left torsional nystagmus. The BBS score was 46/56, DGI was 21/24 with difficulty on walking with head turns and obstacle negotiation, and mCTSIB exhibited loss of balance on conditions 2 and 4.

Questions
1. What is your interpretation of these findings?
2. What is an appropriate plan of care?

Responses
1. The presentation of slow onset of loss of hearing and balance needs to be further investigated to rule out possible acoustic neuroma. The lack of classic symptoms associated with acoustic neuroma of aural fullness and tinnitus helps to lessen the likelihood that that is the etiology of this patient’s symptoms. The finding of fatiguing nystagmus with Hallpike-Dix testing further confirms a peripheral versus central lesion. This is an important finding given the fact that acoustic neuroma can present with a similar profile of symptoms as a peripheral lesion, but will present with nonfatiguing vertigo and nystagmus with positional testing. Positional testing was also indicated given the prevalence of BPPV in the older adult, along with the patient’s complaints of symptoms provoked with position changes. Modify the Hallpike-Dix position to ensure the posterior canal is in the proper plane relative to gravity to reduce the likelihood of a false negative test result. Coming back to the findings of slow progression of loss of hearing and balance coupled with the finding of a positive HIT bilaterally points to age-related degeneration of CN VIII. The mCTSIB findings reveal that the patient is reliant on vision, which is consistent with progressive bilateral vestibular hypofunction.

2. Treat the BPPV first with an Epley maneuver in a modified position so that the patient achieves head extension of 20° relative to the horizon in the absence of being able to achieve 20° of cervical extension. The treatment plan should also include habituation exercises to maximize any residual vestibular function and balance exercises to maximize postural control. The items that were most challenging on the BBS and
DGI could guide balance training, and a program of balance exercises tailored to this patient’s needs should incorporate encountering obstacles, stairs, and uneven terrains to foster higher-level community-based mobility. Care should be taken when interpreting the mCTSIB to guide progression of exercises, avoiding training postural control with vision obliterated. With significant vestibular loss there may not be enough residual vestibular function for CNS compensation to occur and the patient will be reliant on vision as a compensation for loss of vestibular function. Taking away vision as a strategy to challenge the patient will leave him with inadequate sensory inputs to maintain balance. Encouraging the patient to continue to use the cane will help provide additional somatosensory inputs through the upper extremity, contributing to enhanced postural control.
EXAM QUESTIONS

EXAMINATION AND TREATMENT OF PERIPHERAL VESTIBULAR DISORDERS
Questions 1–23

Note: Choose the one option that BEST answers each question.

1. The semicircular canals of the vestibular system detect what type of motion?
   a. Angular velocity
   b. Linear velocity
   c. Linear acceleration
   d. Rotational acceleration

2. In a normally functioning vestibular system, how will the neural firing rate change with head rotation to the right at 100° per second?
   a. Increase on the left side by 100° per second
   b. Decrease on the left side by 50° per second
   c. Decrease on the right side by 50° per second
   d. Increase on the right side by 100° per second

3. The phenomenon of inhibitory cutoff occurs with head velocities greater than
   a. 90° per second.
   b. 180° per second.
   c. 220° per second.
   d. 550° per second.

4. Which of the following are the gravity-sensitive structures of the vestibular system?
   a. Labyrinths
   b. Otoliths
   c. Semicircular canals
   d. Ampullae

5. With head rotation to the right, the MLF and ascending tract of Deiters mediates gaze stabilization through
   a. excitation of bilateral medial rectus muscles and inhibition of bilateral lateral rectus muscles.
   b. excitation of right medial and left lateral rectus muscles and inhibition of right lateral and left medial rectus muscles.
   c. excitation of medial and lateral rectus muscles on the right and inhibition of lateral and medial rectus muscles on the left.
   d. excitation of left medial and right lateral rectus and inhibition of left lateral and right medial rectus muscles.

6. The primary symptom of benign paroxysmal positional vertigo is
   a. nonfatiguing nystagmus.
   b. vertigo provoked with changes in head position.
   c. vertigo induced by changes in pressure.
   d. persistent vertigo at rest.

continued on next page
7. What is the mechanism that underlies spontaneous nystagmus in unilateral vestibular hypofunction?
   a. Reduced neural input to the ipsilateral ocular muscles, resulting in slow deviation eye movement toward the affected side
   b. Reduced neural input to the contralateral ocular muscles, resulting in slow deviation eye movement toward the affected side
   c. Increased neural input to the ipsilateral side, resulting in fast corrective movement toward the affected side
   d. Increased neural input to the contralateral side, resulting in fast phase corrective eye movement toward the affected side

8. Which of the following collective exam findings differentiates central vestibular pathology from peripheral pathology?
   a. Direction-changing nystagmus, suppressed with visual fixation
   b. Direction-changing nystagmus, enhanced with visual fixation
   c. Nystagmus that follows Alexander’s law, enhanced with visual fixation
   d. Central-gaze nystagmus, suppressed with visual fixation

9. In the acute stage of vestibular dysfunction, spontaneous nystagmus that is found with gaze center, decreased with gaze toward the lesioned side and increased with gaze toward the unaffected side, is considered to be
   a. third-degree nystagmus.
   b. indicative of a central lesion.
   c. consistent with Alexander’s law.
   d. gaze-holding nystagmus.

10. A patient reports recent history of episodic vertigo while on vacation when coming up from the bottom of the pool or flying. What would be the most appropriate test to include in the clinical exam?
   a. Application of pressure to external ear canal
   b. Hallpike-Dix test
   c. Caloric test
   d. DVA test

11. Upon oculomotor testing of a patient with dizziness, you find corrective saccades on testing smooth pursuits and saccades. How would you manage this patient’s findings?
   a. Prescribe gaze stabilization exercises
   b. Refer the patient for further diagnostic workup
   c. Prescribe habituation exercises
   d. Perform an in-depth balance examination

12. A corrective saccadic eye movement with a HIT to the right is interpreted as positive for
   a. left lateral canal BPPV.
   b. right lateral canal BPPV.
   c. left vestibular hypofunction.
   d. right vestibular hypofunction.

13. A patient with a recent inner ear infection describes dizziness and blurred vision with walking and head turns. Which of the following tests is most appropriate to include in the clinical examination?
   a. Hallpike-Dix test
   b. The MSQ
   c. The DVA
   d. The BBS
14. A 3-line degradation on a DVA test is a clinical finding for which of the following conditions?
   a. BPPV
   b. Ménière’s disease
   c. Perilymph fistula
   d. Vestibular hypofunction

15. On examining a 79-year-old patient with complaints of significant difficulties with balance walking in low-lit environments, but no true vertigo, the HIT is positive to both right and left sides. This finding is indicative of
   a. acoustic neuroma.
   b. bilateral horizontal canal BPPV.
   c. poor gaze stabilization.
   d. bilateral vestibular hypofunction/loss.

16. Your patient describes transient vertigo when getting out of bed in the morning. Which of the following tests would be most important to include in his or her clinical examination?
   a. The HIT
   b. The MSQ
   c. Hallpike-Dix test
   d. Berg Balance Scale

17. When performing a Roll Test, a patient exhibits ageotropic nystagmus, which is increased in the right sidelying position. What is the interpretation of this test?
   a. Left horizontal canal cupulolithiasis
   b. Left horizontal canalithiasis
   c. Right horizontal canal cupulolithiasis
   d. Right horizontal canalithiasis

18. A patient exhibits persistent nonfatiguing upbeat torsional nystagmus for >2 minutes, with symptoms of vertigo in a right Hallpike-Dix position. Based on these findings, the most likely diagnosis would be
   a. severe right posterior canal BPPV.
   b. right posterior canal cupulolithiasis.
   c. perilymph fistula.
   d. a centrally mediated vestibular lesion.

19. Which of the following is the appropriate treatment for posterior cupulolithiasis?
   a. Epley maneuver
   b. Semont maneuver
   c. Casani maneuver
   d. Appiani maneuver

20. Which of the following is the appropriate treatment for horizontal canalithiasis?
   a. Casani maneuver
   b. Epley maneuver
   c. Semont maneuver
   d. Appiani maneuver

21. Which of the following treatment programs is most appropriate to address dizziness and difficulties with balance for the patient with vestibular hypofunction?
   a. Walking with eyes closed, smooth pursuit eye movements in sit, standing on one leg
   b. Walking with head turns, vertical head turns in sit, standing on foam with eyes closed
   c. Walking with head turns, smooth pursuit eye movements in sit, standing on solid floor with eyes open
   d. Walking with eyes closed, head turns in sit, standing on one leg
22. What is the mechanism that underlies the error signals that drive the process of vestibular adaptation?
   a. Hyperpolarization of the affected side
   b. Inhibitory cutoff
   c. Retinal slip
   d. The push-pull arrangement of the semicircular canals

23. Treatment for diminished gaze stabilization is called
   a. repositioning.
   b. adaptation.
   c. habituation.
   d. compensation.

This concludes the final examination. Please answer the evaluation questions found on page v of this course book.
RESOURCES

EQUIPMENT

Frenzel lenses: GN Otometrics North America
800-289-2150
Email: sales@gnotometrics.com
http://www.otometrics.com

Interacoustics USA
800-947-6334 ext.4437
Email: md@interacoustics-us.com
http://www.interacoustics.com

Micromedical Technologies
800-334.4154
http://www.micromedical.com

ORGANIZATIONS

Neurology Section, American Physical Therapy Association
http://www.neuropt.org

Dizziness-and-balance.com
Timothy Hain, MD, Chicago Dizziness and Hearing.
Website containing a wealth of information about peripheral and central vestibular disorders for patients and healthcare providers. Many videos on oculomotor testing are available.

Vestibular Disorders Association (VEDA)
www.vestibular.org
Professional network that supplies additional resources, information, and support. The website contains informative descriptions of vestibular conditions along with general medical and rehabilitative management.
**GLOSSARY**

**Ageotropic:** Describes direction of the fast phase of nystagmus away from the ground with the patient in a sidelying position.

**Alexander’s law:** Slow phase eye movements of nystagmus are faster when the patient looks toward the direction of the quick phase.

**Ampullae:** Widened portion of the semicircular canal at the juncture with the utricle. Contains the sensory hair cells for the semicircular canals.

**Canalithiasis:** A type of BPPV where the otoconia are free-floating in the semicircular canal.

**Cristae:** Sensory structure of the semicircular canals that contain the hair cells that sense angular motion.

**Cupula:** Bulbous gelatinous mass that surrounds the hair cells of the crista within the semicircular canal.

**Cupulolithiasis:** A type of BPPV where the otoconia are adhered to the cupula.

**Frenzel lenses:** Twenty diopter lenses that block visual fixation by the patient, but still allow the examiner to see eye movement responses.

**Geotropic:** Describes direction of the fast phase of nystagmus toward the ground with the patient in a sidelying position.

**Hennebert’s sign:** Nystagmus indices by pressure change in the external auditory canal. Suggests perilymph fistula or Ménière’s disease.

**Maculae:** Gelatinous structure in the utricle and saccule that include hair cells that detect linear motion. Otoconia are embedded on top of maculae.

**Saccades:** Eye movements used to move quickly or “jump” from one target to another.

**Skew eye deviation:** Vertical misalignment of eyes. Sign of a peripheral or central otolith lesion.

**Smooth pursuit:** Smooth eye movement used to track a moving target.

**Tragus:** Ear flap directly in front of the external auditory canal.
REFERENCES


