Vestibulo-ocular Physiology
Underlying Vestibular Hypofunction

The vestibular system detects motion of the head and maintains stability of images on the fovea of the retina as well as postural control during head motion. Signals representing angular and translational motion of the head as well as the tilt of the head relative to gravity are transduced by the vestibular end organs in the inner ear. This sensory information is then used to control reflexes responsible for maintaining the stability of images on the fovea (the central area of the retina where visual acuity is best) during head movements. Information from the vestibular receptors also is important for posture and gait. When vestibular function is normal, these reflexes operate with exquisite accuracy and, in the case of eye movements, at very short latencies. Knowledge of vestibular anatomy and physiology is important for physical therapists to effectively diagnose and manage people with vestibular dysfunction. The purposes of this article are to review the anatomy and physiology of the vestibular system and to describe the neurophysiological mechanisms responsible for the vestibulo-ocular abnormalities in patients with vestibular hypofunction. [Schubert MC, Minor LB. Vestibulo-ocular physiology underlying vestibular hypofunction. Phys Ther. 2004;84:373–385.]

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The vestibular system is responsible for sensing motion of the head and maintains stability of images on the fovea of the retina and postural control during that motion. When functioning normally, the vestibular receptors in the inner ear provide an exquisitely accurate representation of the motion of the head in 3 dimensions. This information is then used by the central vestibular pathways to control reflexes and perceptions that are mediated by the vestibular system. Disorders of vestibular function result in abnormalities in these reflexes and lead to sensations that reflect abnormal information about motion from the vestibular receptors.1

Best visual acuity is obtained when images are projected on the fovea of the retina. The fovea occupies a small area of the visual field, but movements of an image off the fovea by as little as 1 degree can cause substantial decreases in visual acuity.2 Stabilization of a visual target on the fovea can be achieved by various systems, including the vestibular and smooth pursuit oculomotor systems.3 Influences such as target velocity and distance as well as velocity and frequency of head motion are the stimulus variables the brain uses to determine which oculomotor system is recruited for gaze stability. Each of the oculomotor subsystems has a range in which it functions most efficiently.

Normal activities of daily life (such as running) can have head velocities of up to 550°/s, head accelerations of up to 6,000°/s^2, and frequency content of head motion from 0 to 20 Hz.4,5 Only the vestibular system can detect head motion over this range of velocity, acceleration, and frequency.3 Additionally, the latency of the vestibulo-ocular reflex (VOR) has been reported to be as short as 5 to 7 milliseconds.6,7 In contrast, ocular following mechanisms, such as smooth pursuit, generate slower eye velocities (<60°/s) and have relatively long latencies (up to 100 milliseconds).8,9

The purposes of this article are to review the anatomy and physiology of the vestibular system and to describe the neurophysiological mechanisms responsible for the vestibulo-ocular abnormalities of people with vestibular dysfunction.

An understanding of the exquisite and essential vestibular anatomy and physiology is needed to effectively diagnose and manage people with vestibular dysfunction.

Anatomy and Physiology

Peripheral Vestibular Anatomy

Within the petrous portion of each temporal bone lies the membranous vestibular labyrinth. Each labyrinth contains 5 neural structures that detect head acceleration: 3 semicircular canals and 2 otolith organs (Fig. 1). The 3 semicircular canals (SCC) (lateral, posterior, and anterior) respond to angular acceleration and are orthogonal with respect to each other. Alignment of the SCCs in the temporal bone is such that each canal has a contralateral coplanar mate. The lateral canals form a coplanar pair, whereas the posterior and contralateral anterior SCC form coplanar pairs. The anterior aspect of the lateral SCC is inclined 30 degrees upward from a plane connecting the external auditory canal to the lateral canthus. The posterior and anterior SCCs are inclined about 92 and 90 degrees from the plane of the lateral SCC.10 Because the SCCs are not precisely orthogonal with earth vertical or earth horizontal, angular rotation of the head stimulates each canal to varying degrees.11

The SCCs are filled with endolymph that has a density slightly greater than that of water. Endolymph contains a high concentration of potassium, with a lower concentration of sodium, and moves freely within each canal in response to the direction of the angular head rotation.12 The SCCs enlarge at one end to form the ampulla. Within the ampulla lies the cupula, a gelatinous barrier that houses the sensory hair cells (Fig. 2A). The kinocilia and stereocilia of the hair cells are seated in the crista ampullaris (Fig. 2B). Deflection of the stereocilia caused by motion of the endolymph results in an opening (or closing) of the transduction channels of hair cells, which changes the membrane potential of the hair cells. Deflection of the stereocilia toward the single kinocilia in each hair cell leads to excitation (depolarization), and...
deflection of the stereocilia away from the kinocilia leads to inhibition (hyperpolarization).

Hair cells are oriented in the lateral SCC so that endolymph motion toward the ampulla causes excitation. In contrast, hair cells of the vertical SCCs (posterior and anterior) are oriented so that depolarization occurs when endolymph moves away from the ampulla. Each of the SCCs responds best to motion in its own plane, with coplanar pairs exhibiting a push-pull dynamic. For example, as the head is turned to the right, the hair cells in the right lateral SCC are excited, whereas the hair cells in the left lateral SCC are inhibited.\textsuperscript{13} The brain detects the direction of head movement by comparing input from the coplanar labyrinthine mates.

The saccule and utricle make up the otolith organs of the membranous labyrinth. Sensory hair cells project into a gelatinous material that has calcium carbonate crystals (otoconia) embedded in it, which provide the otolith organs with an inertial mass (Fig. 3). The utricle and the saccule have central regions known as the striola, dividing the otolith organs into 2 parts. The kinocilia of the utricular hair cells are oriented toward their striola, whereas the kinocilia of the saccular hair cells are oriented away from their striola. Motion toward the kinocilia causes excitation. Utricular excitation occurs during horizontal linear acceleration or static head tilt, and saccular excitation occurs during vertical linear acceleration.

**Vestibular Afferent Physiology**

In primates, primary vestibular afferents of the healthy vestibular system have a resting firing rate that is typically 70 to 100 spikes per second.\textsuperscript{13,14} The discharge regularity (determined by the spacing of the interspike intervals between action potentials [Fig. 4]) of vestibular nerve afferents provides a useful marker for the information carried by these afferents. The coefficient of variation (standard deviation/mean discharge) of the interspike interval provides a useful measurement for classifying afferents into irregularly and regularly discharging groups. The information carried by irregular and regular afferents varies over the spectral range of frequency and acceleration that encompasses natural head movements. Generally, irregular afferents are more sensitive to rotations during large head accelerations than regular afferents are.\textsuperscript{14} The increased sensitivity of the irregular afferents may be more critical for the rapid detection of head movements as well as initiation of the VOR.\textsuperscript{6,11} The regular afferents, in contrast, provide a signal that is proportional to head velocity over a wide spectral range.\textsuperscript{14} In addition, the regular afferents may be the primary source of input to the VOR for steady-state responses to sinusoidal rotations because temporarily silencing the irregular afferents has no affect on the VOR during low-frequency and small head accelerations.\textsuperscript{15}

The cells bodies of vestibular nerve afferents are located in the superior or inferior divisions of Scarpas ganglion (Gangl. Scarpae), which lie within the internal auditory canal near the emergence of the vestibular nerve into the cerebello-pontine angle.\textsuperscript{16} From the vestibular labyrinth, the afferent information travels ipsilateral in 1 of 2 branches of the vestibular nerve. The superior vestibular nerve innervates the lateral and anterior SCC as well as the utricle. The inferior vestibular nerve innervates the posterior SCC and the saccule.\textsuperscript{17} It is estimated that between 15,000 to around 25,000 vestibular nerve fibers exist in humans.\textsuperscript{18–20} Variation of nerve fiber counts among studies appears to be a function of age, although rate of
decline of the number of afferent fibers also appears to be variable. The branches of the vestibular nerve travel together into the pontomedullary junction where they bifurcate. Primary vestibular afferents in the superior division of the vestibular nerve include axons that synapse in the superior and medial vestibular nuclei or the uvula, nodulus, flocculus, or fastigial nucleus of the cerebellum. Primary vestibular afferents from the inferior branch synapse with neurons in either the medial, lateral, or inferior vestibular nuclei, which, along with the superior vestibular nuclei and other subnuclei, comprise the vestibular nuclear complex.

Central Vestibular Anatomy
Secondary vestibular afferents have been identified as relaying signals from the vestibular nuclei to the extraocular motor nuclei, the spinal cord, or the flocculus of the cerebellum. Central vestibular neurons differ in terms of the inputs they receive from regular and irregular afferents. Those central vestibular neurons that project to the extraocular motor nuclei receive a majority of their monosynaptic inputs from regular afferents, whereas those that project to the spinal cord receive a majority of their inputs from irregular afferents. Those central vestibular neurons projecting to the flocculus of the cerebellum receive relatively equal contributions from regular and irregular afferents.

Many vestibular reflexes are controlled by processes that exist primarily within the brain stem. Tracing techniques, however, have identified extensive connections between the vestibular nuclei and the reticular formation, thalamus, and cerebellum. Vestibular pathways appear to terminate in a unique cortical area. In studies of primates, fibers terminating in the junction of the parietal and insular lobes have been identified and considered the location for a vestibular cortex. Recent evidence in studies of humans using functional magnetic resonance imaging appears to confirm the parietal and insular regions as the cortical location for processing vestibular information. Connections with
the vestibular cortex, thalamus, and reticular formation enable the vestibular system to contribute to the integration of arousal and conscious awareness of the body and to discriminate between movement of self and the environment. The cerebellar connections help maintain calibration of the VOR, contribute to posture during static and dynamic activities, and influence the coordination of limb movements.

Vestibulo-ocular Physiology

The ability of the VOR to elicit rapid compensatory eye movements that maintain stability of images on the fovea depends on relatively simple patterns of connectivity in the central vestibular pathways. In its most basic form, the pathways controlling the VOR can be described as a 3-neuron arc. In the case of the lateral SCC, primary vestibular afferents from the lateral SCC synapse in the ipsilateral medial and ventrolateral vestibular nuclei. Some of the secondary vestibular neurons receiving innervation from the ipsilateral labyrinth have axons that decussate and synapse in the contralateral abducens nucleus, whereas others ascend ipsilaterally to the oculomotor nucleus. Motoneurons from the abducens nucleus and the medial rectus subdivision of the oculomotor nucleus then synapse at the neuromuscular junction of the lateral rectus and medial rectus muscles, respectively. Similar patterns of connectivity exist for the anterior and posterior SCC and the eye muscles that receive innervations from them (Tab. 1).

Figure 4 illustrates the insertions of the ocular muscles.

The VOR has been tested across multiple frequencies and velocities and shows velocity-dependent nonlinearities, which may correlate with unique afferent physiology. The gain of the VOR remains constant (linear) across multiple frequencies of sinusoidal rotations, with peak velocities of $\pm 20^\circ/s$. For rotations at higher frequencies and velocities, the VOR gain rises with increases in stimulus velocity (nonlinear). Similar effects of stimulus frequency and velocity are seen in responses to steps of acceleration. Therefore, it may be that the output of the VOR is the combined result of linear and nonlinear components. Adaptation experiments in which spectacles were used to modify the gain of the VOR support the notion that a linear component and a nonlinear component may be responsible for mediating the VOR. Using different frequency and velocity profiles for the adaptation stimulus, the nonlinear component has been shown to be adaptable only with high-frequency and high-velocity stimuli.

Incidence and Prevalence of Dizziness in the United States

The incidence of dizziness in the United States is approximately 5.5%, which means that more than 15 million people develop the symptom each year. The reported prevalence of dizziness as a medical complaint in community-dwelling adults varies based on their age, sex, and definition of the complaint (1%–35%). Researchers using specific definitions such as vertigo (an illusion of motion) have reported a prevalence of up to 6.7%, which increases with age. When researchers used a broader definition that included light-headedness and disequilibrium, they reported a greater prevalence of dizziness (25%–35%). Many of these patients most likely had nonvestibular causes of their dizziness. Dizziness is one of the most common complaints reported in physicians’ offices, with the prevalence increasing with age. For patients over 75 years of age, dizziness is the
most common reason they see a physician. Regardless of age, patients who experience dizziness report a significant disability that reduces their quality of life.45–48 Furthermore, it has been reported that more than 70% of patients with initial reports of dizziness will not have a resolution of symptoms at a 2-week follow-up. Of those patients with persistent dizziness, 63% reported recurrent symptoms continuing beyond 3 months.49

**Distinguishing Between Vestibular and Nonvestibular Causes of Dizziness**

Clinicians who work with people who report dizziness and imbalance have the difficult task of sorting through potential causes. Capturing a thorough history is a critical component of the assessment. Many patients and clinicians use the imprecise term “dizziness” to describe a vague sensation of light-headedness or a feeling that they have a tendency to fall. The imprecision of the term can make clinical management decisions complicated. Generally, most complaints of being “dizzy” can be categorized as light-headedness, disequilibrium, vertigo, or oscillopsia.

**Light-headedness** is often defined as a feeling that fainting is about to occur and can be caused by nonvestibular factors such as hypotension, hypoglycemia, or anxiety.50

**Disequilibrium** is defined as the sensation of being off balance. Often, disequilibrium is associated with nonvestibular problems such as decreased somatosensation or weakness in the lower extremities. **Vertigo** is defined as an illusion of movement. Vertigo tends to be episodic and tends to indicate pathology at one or more places along the vestibular pathways. Vertigo is common during the acute stage of a unilateral vestibular lesion, but also may manifest itself through displaced otoconia (benign paroxysmal positional vertigo [BPPV]) or acute brain stem lesions affecting the root entry zone of the peripheral vestibular neurons or the vestibular nuclei.50 **Oscillopsia** is the experience that objects in the visual surround that are known to be stationary are in motion. Oscillopsia can occur in association with head movements in patients with vestibular hypofunction because the vestibular system is not generating an adequate compensatory eye velocity during a head rotation.51 A deficit such as this in the VOR results in motion of images on the fovea and in a decline in visual acuity. The severity of gaze instability, however, varies among people with vestibular hypofunction.51–54

Table 2 lists some of the more common causes associated with symptoms due to vestibular and nonvestibular dizziness and imbalance. Baloh50 provided a thorough review that distinguishes vestibular causes of dizziness from nonvestibular causes.
### Table 2.
Possible Causes of Vestibular and Nonvestibular Symptoms

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Symptoms</th>
<th>Possible Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vestibular</td>
<td>Oscillopsia with head movement</td>
<td>Unilateral vestibular hypofunction, bilateral vestibular hypofunction, benign paroxysmal positional vertigo, unilateral central lesion affecting the vestibular nuclei</td>
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<tr>
<td></td>
<td>Vertigo</td>
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<td></td>
<td>Imbalance</td>
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<tr>
<td>Nonvestibular</td>
<td>Light-headedness</td>
<td>Orthostatic hypotension, hypoglycemia, anxiety, panic disorder, lower-extremity somatosensory deficit, upper brain stem and motor pathway lesions</td>
</tr>
<tr>
<td></td>
<td>Disequilibrium</td>
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**Clinical Measures of Vestibular Function**

To clinically assess vestibular dysfunction, first a careful history is taken. The clinical examination then encompasses assessment of eye movements, posture, and gait. Because of the direct relationship between vestibular receptors in the inner ear and eye movements produced by VORs, the bedside examination of eye movements can be of primary importance in defining and localizing vestibular pathology.

Clinical evaluation of the vestibulo-ocular system takes advantage of 2 physiological principles: the high resting firing rate and the inequality in firing rates within the central vestibular neurons for excitation and inhibition. The presence of a high resting firing rate means each vestibular system can detect head motion through excitation or inhibition. During angular head rotations, ipsilateral vestibular afferents can be excited up to 400 spikes per second. Such head movements also result in inhibition of peripheral afferents and of many central vestibular neurons receiving innervation from the labyrinth opposite the rotation. Because the resting discharge rate of these afferents and central vestibular neurons averages 70 to 100 spikes per second, inhibitory cutoff is more likely to occur than is excitation saturation.

**Head Thrust Test**

The head thrust test is a widely accepted clinical tool that is used to assess semicircular canal function. The head is flexed 30 degrees (to ensure cupular stimulation primarily in the tested lateral SCC). Patients are asked to keep their eyes focused on a target while their head is manually rotated in an unpredictable direction using a small-amplitude (5°–15°), high-acceleration (3,000–4,000°/s²) angular thrust. When the VOR is functioning normally, the eyes move in the direction opposite to the head movement and through the exact angle required to keep images stable on the fovea. In the case of vestibular hypofunction, the eyes move less than the required amount. At the end of the head movement, the eyes are not looking at the intended target and images have shifted on the fovea. A rapid, corrective saccade is made to bring the target back on the fovea. The appearance of these corrective saccades indicates vestibular hypofunction as evaluated by the head thrust test. During a horizontal rotation toward the ear with vestibular hypofunction, corrective saccades occur because inhibition of vestibular afferents and central vestibular neurons on the intact side (inhibitory cutoff) is less effective in encoding the amplitude of a head movement than excitation is.

The head thrust test provides a sensitive indication of vestibular hypofunction in patients with complete loss of function in the affected labyrinth that occurs following ablative surgical procedures, such as labyrinthectomy. The test is less sensitive in detecting hypofunction in patients with incomplete loss of function. The test is less sensitive in detecting hypofunction in patients with incomplete loss of function.

**Head-Shaking–Induced Nystagmus**

Nystagmus is an involuntary back-and-forth motion of both eyes. Any nystagmus due to vestibular stimulation or pathology is composed of slow and fast eye movements. The slow component (slow eye velocity) is produced by the intact ear, which generates a normal VOR as a result of the asymmetry between the discharge rates of central vestibular neurons on each side. The fast component is a resetting eye movement that brings the eyes close to the center of the oculomotor range.

The head-shaking–induced nystagmus (HSN) test is a useful aid in the diagnosis of people with asymmetry of peripheral vestibular input to central vestibular regions. Patients undergoing the HSN test must have their vision blocked because fixation on a visual target can suppress nystagmus. Similar to the head thrust test, the head should initially be flexed 30 degrees. Next, the head is oscillated horizontally for 20 cycles at a frequency of 2 repetitions per second (2 Hz). Upon stopping the oscillation, people with symmetric peripheral vestibular input will not have HSN. Typically, a person with a unilateral loss of peripheral vestibular function will manifest a horizontal HSN, with the quick phases of the nystagmus directed toward the healthy ear and the slow phases directed toward the lesioned ear. Not all patients with a unilateral vestibular loss will have HSN. Patients with a complete loss of vestibular function bilaterally will not have HSN because neither system is
functioning and there is no asymmetry between the tonic firing rates.

**Positional Testing**

Positional testing is commonly used to identify whether otocinia have been displaced into the SCC, causing benign paroxysmal positional vertigo (BPPV). The addition of the otocinia into the endolymph makes the semicircular canals sensitive to changes in head position. The abnormal signal results in nystagmus and vertigo, nausea with or without vomiting, and disequilibrium. Once the patients are in the provoking position, the resultant nystagmus indicates which semicircular canal is involved. Honrubia et al.\(^67\) and Herdman\(^68\) have reviewed the oculomotor signs and intervention associated with BPPV pathology.

**Dynamic Visual Acuity**

Dynamic visual acuity (DVA) is the measurement of visual acuity during self-generated horizontal motion of the head. A "bedside" and computerized form of the test can be used to identify the functional significance of the vestibular hypofunction.\(^69,70\) Head velocities need to be greater than 100°/s at the time DVA is measured in order to ensure that the vestibular afferents from the semicircular canals on the contralateral side are driven into inhibition and the letters are not identified with a smooth pursuit eye movement.

In people without vestibular problems, head movement results in little or no change of visual acuity compared with the head still. For patients with vestibular hypofunction, the VOR will not keep the eyes stable in space during the rapid head movements. This results in a decrease in visual acuity during head motion compared with the head still. Dynamic visual acuity has been found to correctly identify the side of lesion in patients with unilateral hypofunction for self-generated and unpredictable head motion.\(^70,71\)

**Laboratory Measures of Vestibular Function**

The VOR is typically measured by monitoring eye motion during stimulation of the peripheral vestibular system. The VOR gain is expressed as the ratio of eye velocity to head velocity (eye velocity/head velocity). Under ideal conditions, when the eyes are not verged (adducting), the VOR gain is –1, implying a compensatory eye velocity equal to the head velocity and in the opposite direction. The VOR phase is a second useful measure of the vestibular system and represents the timing relationship for the eye and head position. Ideally, eye position should arrive at a point in time that is equal with the oppositely directed head position. By convention, this is described as zero phase shift (Fig. 6).

**Semicircular Canal Function**

The caloric test is the “gold standard” for identifying peripheral unilateral vestibular hypofunction (UVH).\(^72,73\) By introducing a cold or warm stimulus in the external auditory canal, a temperature gradient is created with the temporal bone. The change in temperature is the greatest for the lateral aspect of the temporal bone and the least for the medial aspect. In the presence of gravity, this temperature gradient results in the convective flow of endolymph that deflects the cupula and generates nystagmus. Direct hair cell stimulation as well as changes in pressure across the middle ear also cause cupular deflection, contributing to the resulting nystagmus.\(^74–76\) The caloric test is particularly useful for determining the side of a deficit because each labyrinth is stimulated separately. Slow components of the nystagmus resulting from irrigations of the right ear are compared with slow components of the nystagmus resulting from irrigations of the left ear. The caloric test provides limited information, however, because only the lateral SCCs are stimulated and that stimulation corresponds to a frequency (0.025 Hz) that is much lower than the natural frequencies of head movement (1–20 Hz).\(^4,5\) The rotary chair test is the “gold standard” for identifying bilateral vestibular hypofunction (BVH) and the extent of central nervous system compensation due to vestibular hypofunction.\(^73\) The rotary chair test provides a physiological stimulus because rotating the patient causes endolymphatic flow in both lateral SCCs. Nystagmus should be generated for rotations in subjects without known pathology or impairments. Depending on the extent of the lesion, people with vestibular hypofunction will demonstrate varied compensatory slow eye velocities. The extent of pathology can be determined by comparing VOR gain and phase from rotations toward one ear with rotations toward the opposite ear. In addition, VOR gain and phase of people without vestibular problems can be compared with that of people with suspected vestibular hypofunction. Rotary chair testing is limited because only the lateral SCCs are routinely assessed to determine extent of pathology.

**Otolith Function**

Recent advances in vestibular diagnostic testing have extended the region of identifiable pathology to include the otolith organs.\(^77–79\) The vestibular-evoked myogenic potentials (VEMP) test has gained broad clinical use in recent years.\(^77\) The VEMP test exposes patients to a series of loud (95 dB) clicks. During the sound application, the ipsilateral sternocleidomastoid (SCM) muscle is assessed for myogenic potentials. In people with healthy vestibular function, an initial inhibitory potential (occurring at a latency of 13 milliseconds after the click) is followed by an excitatory potential (occurring at a latency of 21 milliseconds after the click). For patients with vestibular hypofunction, the VEMPs are absent on the side of the
lesion. The pathway of the VEMP is believed to be associated with the head-neck reflex that maintains verticality of the head in relation to gravity (the vestibulocollic reflex). The saccule has been implicated as the site of afferent stimulation during VEMP testing because saccular afferents provide ipsilateral inhibitory disynaptic input to the SCM muscle,80 are responsive to click noise,81–83 and are positioned close to the footplate of the stapes and, therefore, are subject to mechanical stimulation.78,81

The subjective visual vertical (SVV) and subjective visual horizontal (SVH) tests are used to assess otolith function, though they cannot be used to uniquely detect saccular or utricular pathology. With the SVV test, patients are asked to align a dimly lit luminous bar (in an otherwise darkened room) with what they perceive as being vertical. With the SVH test, patients are asked to align a bar with what they perceive as being horizontal. Subjects without vestibular problems can align the bar within 1.5 degrees of true vertical or horizontal, whereas patients with UVH generally align the bar more than 2 degrees of true vertical or horizontal with the bar tilted toward the lesioned side.79,84,85 Whether the SVV test or the SVH test can detect chronic UVH is the subject of debate.85–87

Causes of Vestibular Hypofunction

Unilateral

The most frequent cause88 of UVH is vestibular neuroitis, which is commonly caused by the herpes simplex virus. The superior vestibular nerve is more likely to be affected than the inferior vestibular nerve.89–91 Less common causes include Ménière disease and vestibular schwannoma on the eighth cranial nerve. The incidence

Figure 6.
Simulated eye movements during low frequency sinusoidal head rotation. Positive numbers along ordinate indicate rightward velocity rotation, whereas negative numbers indicate leftward velocity rotation. Dashed line placed at zero velocity is for reference. Arrow line styles match simulated eye velocities. For people with healthy vestibular function, as the head rotates to the right at 10°/s, the eyes move to the left at 10°/s, and the eye and head velocity reach zero at the same time (gain=1, zero phase shift). For people with bilateral reduced vestibular function, eye velocity may be one half or less with respect to head velocity (5°/s in this example, gain=0.5) and the eyes cross zero velocity in advance of the head crossing zero velocity (eye position leads the head position – phase lead). VOR gain=eye velocity/head velocity.
rates for these disorders are: 1,710 cases of vestibular neuronitis per million per year, \(^{88}\) 500 cases of Ménière disease per million per year, \(^{92}\) and 11.5 cases of vestibular schwannoma per million per year. \(^{93}\) Other pathological events such as vascular lesions affecting the vestibular nerve or traumatic brain injury also may damage the vestibular system unilaterally. Patients who sustain unilateral vestibular damage may experience vertigo, spontaneous nystagmus, oscillopsia, postural instability, and disequilibrium.

When the peripheral vestibular system is damaged unilaterally, neuronal activity reaching the ipsilesional vestibular nuclei is reduced compared with that reaching the contralateral vestibular nuclei. The brain interprets the asymmetry between resting firing rates as a head rotation toward the contralesional ear. This results in spontaneous nystagmus, with slow components directed toward the lesioned ear and fast components directed toward the intact ear. Resolution of spontaneous nystagmus in the light typically occurs within 3 to 7 days but may vary, and it can be a process as long as 2 months. \(^{94,95}\) Spontaneous nystagmus may always be present in the dark after a unilateral loss of vestibular function. Regardless, resolution of spontaneous nystagmus in the light or dark occurs when symmetry between the resting firing rates of both vestibular systems is reestablished. \(^{96}\) A number of authors \(^{97–100}\) have provided more detail on the complex processes involved in vestibular compensation.

**Bilateral**

The most common cause of vestibular hypofunction on both sides (BVH) is ototoxicity due to certain aminoglycoside antibiotics (gentamicin, streptomycin). The antibiotics selectively damage the vestibular hair cells, often preserving auditory function. It is estimated that 3% to 4% of the population who receive gentamicin will sustain damage to both vestibular systems. \(^{101}\) For people who receive gentamicin and renal dialysis concurrently, it is estimated that the likelihood of sustaining BVH is from 12.5% to 30%. \(^{102,103}\) Unfortunately, it appears that people who are susceptible to ototoxicity have little protection from monitoring serum levels of these antibodies. \(^{104}\) Less common causes of BVH include meningitis, head trauma, tumors on each eighth cranial nerve (including bilateral vestibular schwannoma), transient ischemic episodes of vessels supplying the vestibular system, and sequential unilateral vestibular neuronitis. \(^{105–107}\) Patients with BVH typically experience gait ataxia, postural instability, and oscillopsia. \(^{104}\)

**Vestibular Rehabilitation**

Vestibular rehabilitation refers to interventions such as adaptation exercises, habituation exercises, repositioning techniques, and exercise to improve muscle force, gait, or balance. The beneficial effect of much of the rehabilitation for people with vestibulospinal impairments as a result of vestibular hypofunction is well documented. \(^{108–110}\) Controlled studies have been used to demonstrate improvements in dynamic visual acuity and to reduce complaints of oscillopsia as well as to reduce VOR gain asymmetry in people receiving vestibular adaptation exercises. \(^{110,111}\)

Basic research may identify additional roles for programs of vestibular rehabilitation. The angular VOR has components that can be selectively modified based on the frequency and velocity of head movements. \(^{30}\) Future studies may reveal unique head movement strategies that optimize performance and promote recovery of the VOR. These strategies might then be used in the design of interventions. Existing principles of vestibular neurophysiology warrant vestibular rehabilitation that exposes the damaged vestibular system to multiple head frequencies and velocities, thereby ensuring a broad range of stimuli to which the system can adapt.

**Conclusions**

When receptors in the inner ear and central pathways are functioning normally, the vestibular system provides exquisitely accurate mechanisms for stabilizing gaze and posture. Disorders affecting the end organs in the labyrinth or the central pathways cause decreases in the performance of the system, including asymmetries in reflex responses. An understanding of vestibular anatomy and physiology can reveal the reasons that these deficits occur. Further advances in research may lead to design of more effective rehabilitation strategies.

**References**


