

VESTIBULAR 101

Your guide to understanding the basics of the human balance system and what happens when something goes wrong.

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ABOUT THE HUMAN BALANCE SYSTEM

Maintaining balance depends on information received by the brain from the eyes, muscles and joints, and vestibular organs in the inner ear. When this system is disrupted by damage to one or more components through injury, disease, or the aging process you may experience impaired balance accompanied by other symptoms such as dizziness, vertigo, vision problems, nausea, fatigue, and concentration difficulties.

The Balance System

By the Vestibular Disorders Association, with Mary Ann Watson, MA, F. Owen Black, MD, FACS, and Matthew Crowson, MD

ARTICLE 036

Good balance is often taken for granted. Most people don't find it difficult to walk across a gravel driveway, transition from walking on a sidewalk to grass, or get out of bed in the middle of the night without stumbling. However, with impaired balance such activities can be extremely fatiguing and sometimes dangerous. Symptoms that accompany the unsteadiness can include dizziness, vertigo, hearing and vision problems, and difficulty with concentration and memory.

WHAT IS BALANCE?

Balance is the ability to maintain the body's center of mass over its base of support. **1** A properly functioning balance system allows humans to see clearly while moving, identify orientation with respect to gravity, determine direction and speed of movement, and make automatic postural adjustments to maintain posture and stability in various conditions and activities.

Balance is achieved and maintained by a complex set of sensorimotor control systems that include sensory input from vision (sight), proprioception (touch), and the vestibular system (motion, equilibrium, spatial orientation); integration of that sensory input; and motor output to the eye and body muscles. Injury, disease, certain drugs, or the aging process can affect one or more of these components. In addition to the contribution of sensory information, there may also be psychological factors that impair our sense of balance.

SENSORY INPUT

Maintaining balance depends on information received by the brain from three peripheral sources: eyes, muscles and joints, and vestibular organs (**Figure 1**). All three of these information sources send signals to the brain in the form of nerve impulses from special nerve endings called sensory receptors.



INPUT FROM THE EYES

Sensory receptors in the retina are called rods and cones. Rods are believed to be tuned better for vision in low light situations (e.g. at night time). Cones help with color vision, and the finer details of our world. When light strikes the rods and cones, they send impulses to the brain that provide visual cues identifying how a person is oriented relative to other objects. For example, as a pedestrian takes a walk along a city street, the surrounding buildings appear vertically aligned, and each storefront passed first moves into and then beyond the range of peripheral vision.

INPUT FROM MUSCLES AND JOINTS

Proprioceptive information from the skin, muscles, and joints involves sensory receptors that are sensitive to stretch or pressure in the surrounding tissues. For example, increased pressure is felt in the front part of the soles of the feet when a standing person leans forward. With any movement of the legs, arms, and other body parts, sensory receptors respond by sending impulses to the brain. Along with other information, these stretch and pressure cues help our brain determine where our body is in space.

The sensory impulses originating in the neck and ankles are especially important. Proprioceptive cues from the neck indicate the direction in which the head is turned. Cues from the ankles indicate the body's movement or sway relative to both the standing surface (floor or ground) and the quality of that surface (for example, hard, soft, slippery, or uneven).

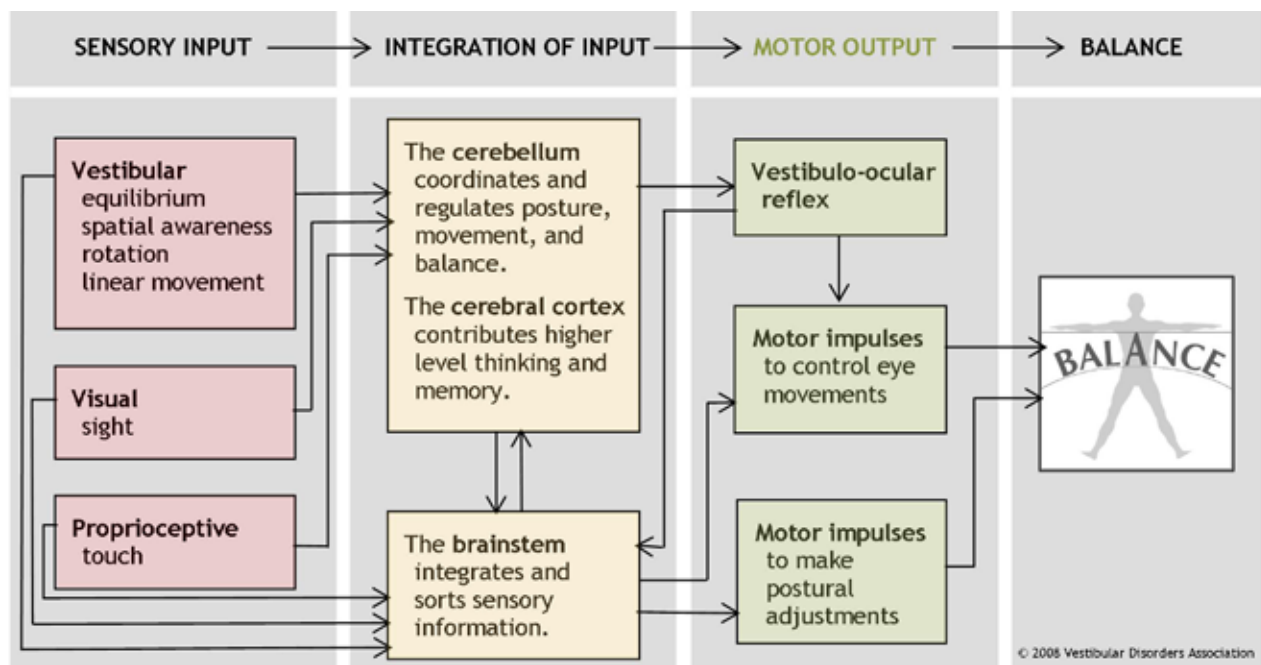


FIGURE 1. Balance is achieved and maintained by a complex set of sensorimotor control systems.



INPUT FROM THE VESTIBULAR SYSTEM

Sensory information about motion, equilibrium, and spatial orientation is provided by the vestibular apparatus, which in each ear includes the utricle, saccule, and three semicircular canals. The utricle and saccule detect gravity (information in a vertical orientation) and linear movement. The semicircular canals, which detect rotational movement, are located at right angles to each other and are filled with a fluid called endolymph. When the head rotates in the direction sensed by a particular canal, the endolymphatic fluid within it lags behind because of inertia, and exerts pressure against the canal's sensory receptor. The receptor then sends impulses to the brain about movement from the specific canal that is stimulated. When the vestibular organs on both sides of the head are functioning properly, they send symmetrical impulses to the brain. (Impulses originating from the right side are consistent with impulses originating from the left side.)

INTEGRATION OF SENSORY INPUT

Balance information provided by the peripheral sensory organs—eyes, muscles and joints, and the two sides of the vestibular system—is sent to the brain stem. There, it is sorted out and integrated with learned information contributed by the cerebellum (the coordination center of the brain) and the cerebral cortex (the thinking and memory center). The cerebellum provides information about automatic movements that have been learned through repeated exposure to certain motions. For example, by repeatedly practicing serving a ball, a tennis player learns to optimize balance control during that movement. Contributions from the cerebral cortex include previously learned information; for example, because icy sidewalks are slippery, one is required to use a different pattern of movement in order to safely navigate them.

PROCESSING OF CONFLICTING SENSORY INPUT

A person can become disoriented if the sensory input received from his or her eyes, muscles and joints, or vestibular organs sources conflicts with one another. For example, this may occur when a person is standing next to a bus that is pulling away from the curb. The visual image of the large rolling bus may create an illusion for the pedestrian that he or she—rather than the bus—is moving. However, at the same time the proprioceptive information from his muscles and joints indicates that he is not actually moving. Sensory information provided by the vestibular organs may help override this sensory conflict. In addition, higher level thinking and memory might compel the person to glance away from the moving bus to look down in order to seek visual confirmation that his body is not moving relative to the pavement.

MOTOR OUTPUT

As sensory integration takes place, the brain stem transmits impulses to the muscles that control movements of the eyes, head and neck, trunk, and legs, thus allowing a person to both maintain balance and have clear vision



while moving.

MOTOR OUTPUT TO MUSCLES AND JOINTS

A baby learns to balance through practice and repetition as impulses sent from the sensory receptors to the brain stem and then out to the muscles form a new pathway. With repetition, it becomes easier for these impulses to travel along that nerve pathway—a process called facilitation—and the baby is able to maintain balance during any activity. Strong evidence exists suggesting that such synaptic reorganization occurs throughout a person's lifetime of adjusting to changing motion environs.

This pathway facilitation is the reason dancers and athletes practice so arduously. Even very complex movements become nearly automatic over a period of time. This also means that if a problem with one sensory information input were to develop, the process of facilitation can help the balance system reset and adapt to achieve a sense of balance again.

For example, when a person is turning cartwheels in a park, impulses transmitted from the brain stem inform the cerebral cortex that this particular activity is appropriately accompanied by the sight of the park whirling in circles. With more practice, the brain learns to interpret a whirling visual field as normal during this type of body rotation. Alternatively, dancers learn that in order to maintain balance while performing a series of pirouettes, they must keep their eyes fixed on one spot in the distance as long as possible while rotating their body.

MOTOR OUTPUT TO THE EYES

The vestibular system sends motor control signals via the nervous system to the muscles of the eyes with an automatic function called the vestibulo-ocular reflex (VOR). When the head is not moving, the number of impulses from the vestibular organs on the right side is equal to the number of impulses coming from the left side. When the head turns toward the right, the number of impulses from the right ear increases and the number from the left ear decreases. The difference in impulses sent from each side controls eye movements and stabilizes the gaze during active head movements (e.g., while running or watching a hockey game) and passive head movements (e.g., while sitting in a car that is accelerating or decelerating).



THE COORDINATED BALANCE SYSTEM

The human balance system involves a complex set of sensorimotor-control systems. Its interlacing feedback mechanisms can be disrupted by damage to one or more components through injury, disease, or the aging process. Impaired balance can be accompanied by other symptoms such as dizziness, vertigo, vision problems, nausea, fatigue, and concentration difficulties.

The complexity of the human balance system creates challenges in diagnosing and treating the underlying cause of imbalance. The crucial integration of information obtained through the vestibular, visual, and proprioceptive systems

means that disorders affecting an individual system

can markedly disrupt a person's normal sense of balance. Vestibular dysfunction as a cause of imbalance offers a particularly intricate challenge because of the vestibular system's interaction with cognitive functioning, [2](#) and the degree of influence it has on the control of eye movements and posture.

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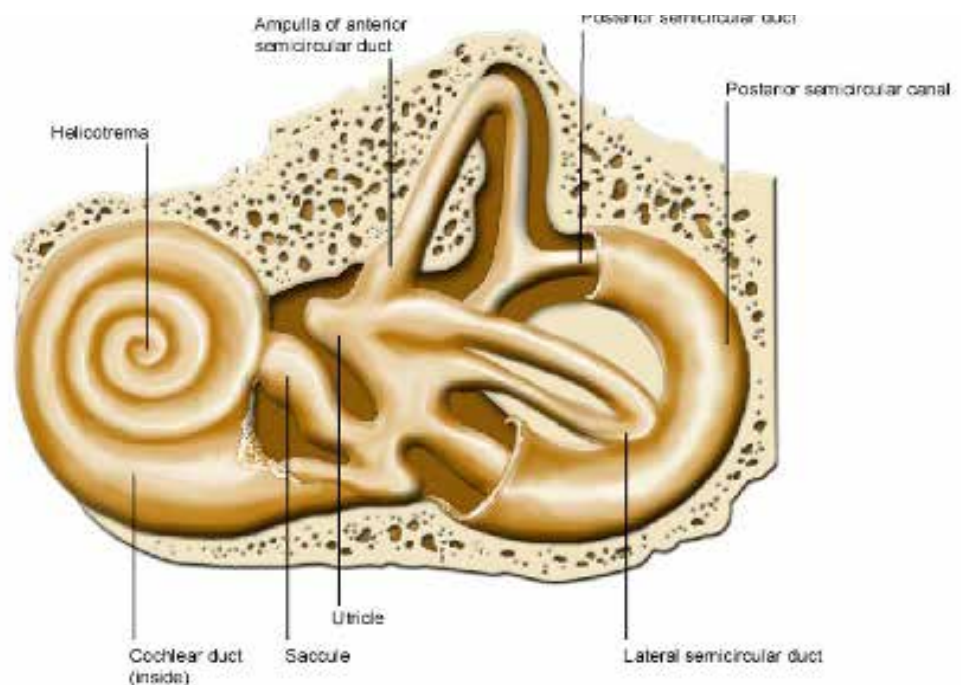
Ear Anatomy

By Brian Keith, B.S. & Habib Rizk, MD

ARTICLE 04

OVERVIEW

There are three anatomical components to the human ear: the external, middle, and inner ear. The inner ear is a complex system found within a hollow cavity of the temporal bone of the human skull known as the bony labyrinth. In this bony labyrinth, there are several structures responsible for both our sense of hearing as well as our sense of balance and position collectively forming the membranous labyrinth. The bony labyrinth can be thought of as a shell surrounding and protecting the membranous labyrinth. The auditory (hearing) component of the inner ear is driven by the cochlea, a spiral-shaped, fluid-filled organ. The vestibular (balance and position) component of the inner ear is directed by the semicircular ducts, the utricle, and the saccule.



"RCSI - Drawing Inner ear showing labyrinth - English labels" at AnatomyTOOL.org by Royal College of Surgeons of Ireland, license: Creative Commons Attribution-NonCommercial-ShareAlike



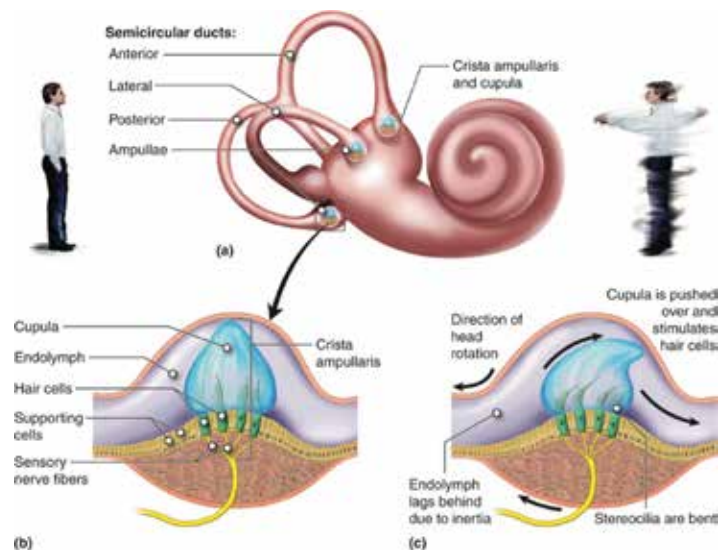
The fluid within the inner ear's membranous labyrinth is called endolymph (endo-: inside or within). The fluid contained between the bony labyrinth and the membranous labyrinth is perilymph (peri-: around or about). These two fluids contain similar chemicals such as potassium, calcium, sodium, and magnesium, but the concentration of chemicals varies substantially between the two. When the composition of these fluids is disturbed, normal function of hearing and balance may be affected.

SEMICIRCULAR DUCTS

With head movement in the plane or angle in which a canal is positioned, the semicircular ducts are fluid-filled tubes deep within the bony semicircular canals that detect the rotational movement of the head about the axes of motion (X, Y, and Z). There are three semicircular ducts: superior, lateral, and posterior. Each semicircular duct detects the motion of the head around a different axis. The superior semicircular duct detects rotational movement around the frontal, or X, axis (e.g. nodding your head to indicate "yes"). The lateral semicircular duct detects movement around the vertical, or Y, axis (e.g. shaking of the head to indicate "no"). The posterior semicircular duct detects movement around the anterior-posterior, or Z, axis (e.g. touching your ear to your shoulder).

Located at the base of each semicircular duct is a widened area referred to as the ampulla. The ampulla

contains the cupula and hair cells. The cupula is a gelatinous structure that spans the entire duct, and its motion is coupled to the hair cells. The hair cells are receptor cells that have hairlike projections called hair bundles that extend into the cupula. When the head moves, the endolymph lags behind due to inertia, which creates a fluid wave that collides with the cupula. The deflection of the cupula, and the hair bundles, activates the hair cells which sends a signal to the brain via the vestibular nerve, a branch of the vestibulocochlear nerve (cranial nerve eight).



"Cenveo - Drawing Structure and Function of the Semicircular Canals - English labels" at AnatomyTOOL.org by Cenveo, license: Creative Commons Attribution

This is how the semicircular ducts detect and transmit rotational movement information to the central nervous system to maintain balance



and position. In addition, this information is used by our brain to carry out corresponding eye movements as our head turns to maintain forward vision. Each ear has a matching set of semicircular ducts and canals, and they work in tandem. Vestibular symptoms (e.g. dizziness, vertigo, imbalance, etc.) may be seen in semicircular duct disturbances such as benign paroxysmal positional vertigo (BPPV), superior canal dehiscence syndrome (SCDS), and alcohol positional nystagmus.

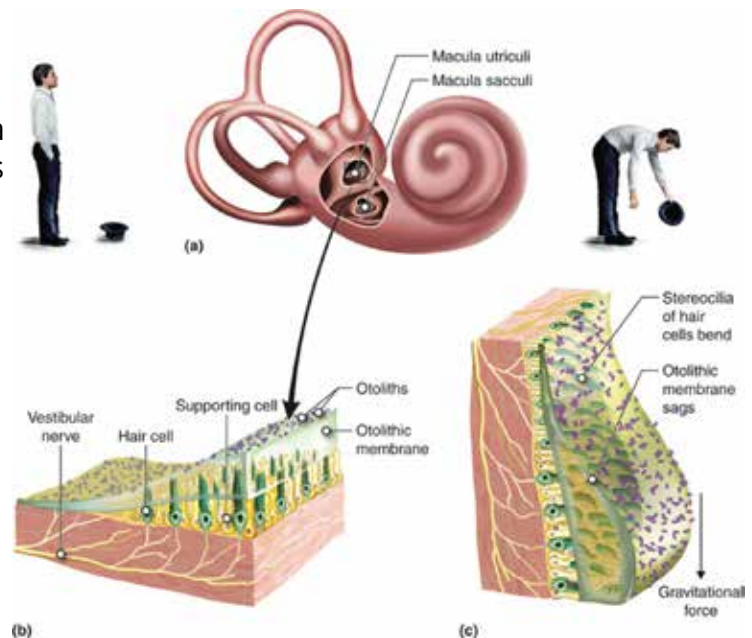
UTRICLE & SACCULE

The utricle and the saccule are involved with linear motion of the head. These two membranous structures, referred to as the otolithic organs, are the sole residents of the vestibule, a hollow cavity located between the cochlea and the semicircular canals. The utricle detects linear motion in the horizontal plane (e.g. accelerating/decelerating in a car) and the saccule detects linear motion in the vertical plane (e.g. movement in an elevator).

The otolithic organs each contain a single curved structure called the macula that contains receptor cells known as hair cells and additional supporting cells. Similar to the mechanism of the semicircular ducts, these hair cells contain tiny hairlike projections. These projections extend into a gelatinous membrane called the otolithic membrane. Resting on top of this membrane are microscopic crystals made of calcium carbonate called otoconia. These are commonly referred to as “ear rocks”

or “ear stones.” Upon linear motions of the head, these tiny crystals will drag along the otolithic membrane and deflect the hairlike projections of the hair cell. This deflection allows the hair cells of the otolithic membranes to fire off a signal to the vestibular nerve and communicate with the brain that the head is moving in the horizontal and/or vertical plane.

Dysfunctions of the otolithic organs may lead to vestibular disorders such as BPPV or persistent postural-perceptual dizziness (PPPD).



“Cenveo - Drawing Structure of the Maculae - English labels” at AnatomyTOOL.org by Cenveo, license: Creative Commons Attribution

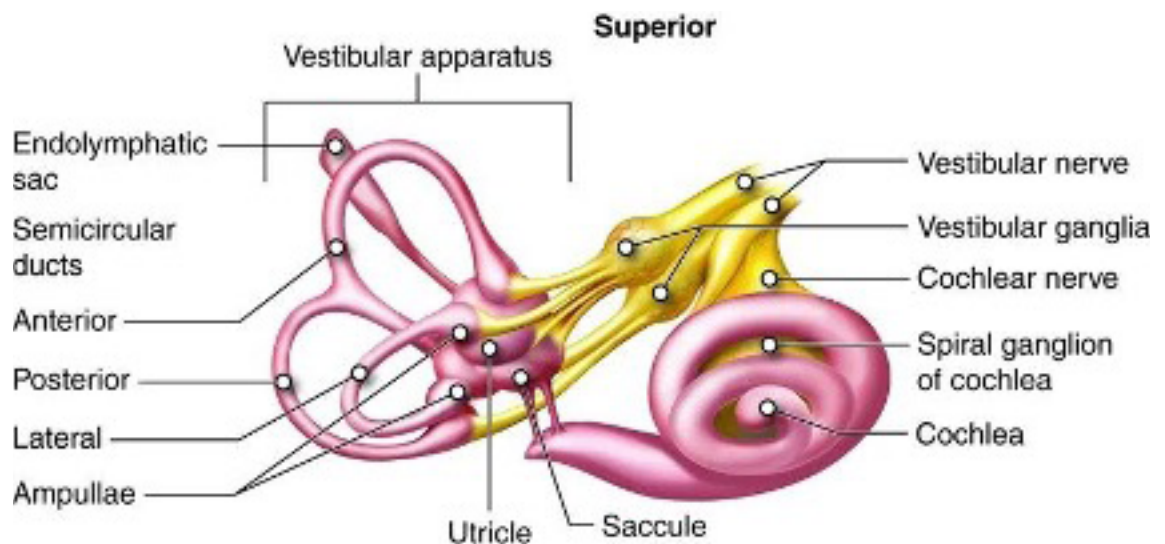
OTHER STRUCTURES

The endolymphatic sac is a pouch that connects to the vestibule via



the endolymphatic duct. The endolymphatic sac is believed to maintain the volume and composition of endolymph as it circulates through the inner ear. If this regulatory system is disrupted, endolymph may accumulate within the inner ear resulting in a condition known as endolymphatic hydrops. In this condition, the excessive volumes of endolymph cause the structures containing endolymph to swell up and may result in vestibular and/or auditory symptoms. Disruption of the endolymphatic sac is only one potential cause of endolymphatic hydrops. Commonly, this condition is associated with Ménière's disease.

The oval window is a kidney-shaped opening connecting the middle ear to the inner ear and this opening is covered with connective tissue. The footplate of the stapes, one of the middle ear bones, sits on top of the connective tissue. The function of the oval window is to transmit vibrations from the stapes to the inner ear fluid, sending a fluid wave through the cochlea and generating sound through specialized cells. Although its role is primarily related to the auditory system, compromise of the oval window's seal may result in vestibular problems. A perilymphatic fistula is an abnormal connection between the perilymph-filled inner ear and the middle ear. This undesirable communication between the inner and middle ear may change the perilymph composition and volume which can lead to vestibular and auditory symptoms.



"Cenveo - Drawing Structures of the Vestibular Apparatus - English labels" at Anatomy-TOOL.org by Cenveo, license: Creative Commons Attribution

The round window is another connective tissue-covered opening between the middle and inner ear. This membrane is on the receiving end of the fluid wave generated by the oval window. When the oval window bulges inward towards the inner ear, the round window bulges outwards towards the middle ear. Like the oval window, disruption of the round window's connective tissue membrane can result in a perilymphatic fistula.



GLOSSARY

auditory: related to the sense of hearing.

canalithiasis: the theory of BPPV (*Benign Paroxysmal Positional Vertigo*), where free-floating debris can migrate into a semicircular canal and cause short episodes of vertigo when it moves within the canal.

central vestibular system: parts of the central nervous system (brain and brainstem) that process information from the peripheral vestibular system about balance and spatial orientation.

cochlea: portion of the inner ear concerned with hearing.

cochlear implant: a prosthetic device that, unlike hearing aids which amplify sound, bypass the outer, middle, and inner ear and directly stimulate auditory nerve fibers.

conductive hearing loss: hearing loss produced by abnormalities of the outer ear or middle ear. These *abnormalities create a hearing loss by interfering with the transmission of sound from the outer ear to the inner ear.*

cupulolithiasis: a variant of BPPV in which the debris is stuck to the cupula of a semicircular canal rather than being loose within the canal.

disequilibrium: unsteadiness, imbalance, or loss of equilibrium; often accompanied by spatial disorientation (a sensation of not knowing where one's body is in relation to the vertical and horizontal planes).

dizziness: lightheadedness; does not involve a rotational component (see vertigo).

endolymph: the fluid within the semicircular canals and vestibule (utricle and saccule).

Eustachian tube: connects the middle ear space with the throat; maintains equal air pressure on both sides of the tympanic membrane (eardrum).

labyrinth: complex system of chambers and passageways of the inner ear; includes both the hearing and balance portions of the inner ear.

labyrinthitis: an inflammation of the labyrinth.

middle ear: air-filled cavity containing the ossicles and tympanic membrane, the function of which is to transfer sound energy from the outer ear to the cochlea of the inner ear.

mixed hearing loss: hearing loss produced by abnormalities in both the



conductive and sensorineural mechanisms of hearing.

nystagmus: involuntary, alternating, rapid and slow movements of the eyeballs.

ossicles (incus, malleus, stapes): tiny bones of the middle ear that conduct sound from the tympanic membrane to the oval window of the inner ear.

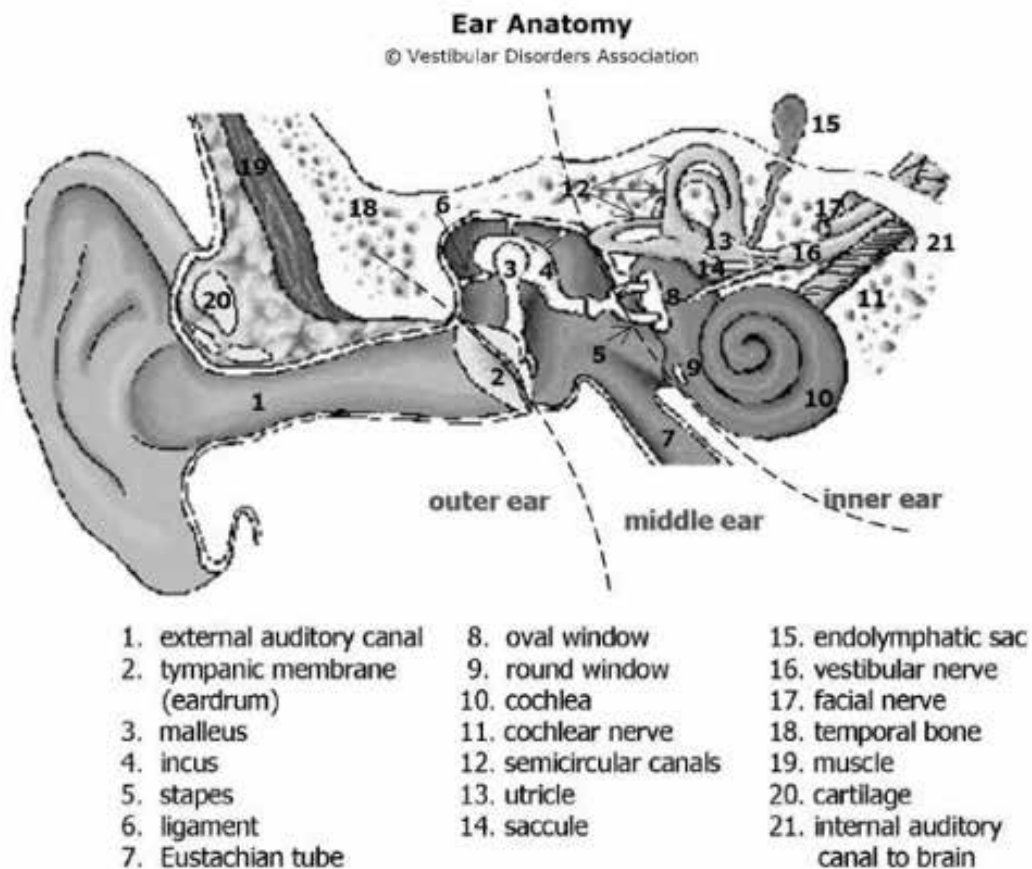
otoliths: calcium carbonate crystals found in the utricle and saccule of the inner ear. Damage to the otoliths may lead to BPPV.

oval window: oval-shaped opening from the middle ear into the inner ear. The footplate of the stapes fits into the oval window.

perilymph: the fluid that fills the space between the semicircular canals and vestibule (utricle and saccule) and the surrounding bone.

peripheral vestibular system: parts of the inner ear concerned with balance and body orientation; consists of the semicircular canals, utricle, and saccule. Peripheral in this context means outside the central nervous system (brain and brainstem), to which the peripheral system sends information.

perilymph fistula: abnormal opening that permits perilymph from the inner



ear to leak into the middle ear.

pinna: external, visible portion of the ear.

Its primary function is to carry sounds to the middle ear. Also called the auricle.

round window: membrane-covered opening between the inner ear and the middle ear.

saccul: sac-like inner ear organ containing otoliths; senses vertical motion of the head.

sensorineural hearing loss: hearing loss produced by abnormalities of the cochlea or the auditory nerve or of the nerve pathways that lead beyond the cochlea to the brain.

temporal bone: part of the skull in which the inner ear is located.

tinnitus: noise or ringing in the ears.

tympanic membrane: eardrum; separates the external ear canal from the middle-ear air cavity.

utricle: sac-like inner ear organ containing otoliths; senses forward, backward, and side-to-side motion of the head.

vertigo: perception of movement (either of the self or surrounding objects) that is not occurring or is occurring differently from how it is perceived.

vestibulo-cochlear nerve: nerve that carries information from the inner ear to the brain. Also called the *eighth cranial nerve, auditory nerve, or acoustic nerve.*

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Causes of Dizziness

By Brian Keith, B.S. & Habib Rizk, MD

ARTICLE 11

Dizziness is a very common complaint, and it is estimated that this symptom accounts for 5% of all primary care visits. "Dizziness" can mean many things to patients but generally it refers to a sense of disorientation without a false sense of motion. On the other hand, "vertigo" is a spinning sensation or false sense of motion in the absence of head movement. The two terms are often used interchangeably. "Dizziness" may also be used by patients to describe a feeling of being off-balance (disequilibrium), a feeling of losing consciousness (pre-syncope), or lightheadedness.

To clarify "dizziness" the Barany Society defined the term and others in 2009:

Dizziness: The sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion.

Vertigo: The sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement.

Unsteadiness: The feeling of being unstable while seated, standing, or walking without a particular directional preference.

Source: Bisdorff A, Von Brevern M, Lempert T, Newman-Toker DE. Classification of vestibular symptoms: Towards an international classification of vestibular disorders. *Journal of Vestibular Research*. 2009;19:1-13.

Causes (etiologies) of dizziness can be most easily divided into two categories: vestibular and nonvestibular. Vestibular causes of dizziness arise from the peripheral or the central vestibular system. Nonvestibular causes of dizziness originate outside the vestibular system, including the peripheral nervous and cardiovascular systems. This article explores the most common causes of vestibular and nonvestibular dizziness to help patients better understand the cause of their symptoms.



VESTIBULAR CAUSES OF DIZZINESS

The vestibular system is responsible for our sense of balance and position. This system is controlled by the semicircular canals, the utricle, and the saccule. The semicircular canals detect rotational (circular) movement of the head while the utricle and the saccule detect linear (forward/backward) motion of the head. For more detailed information about the vestibular system, refer to this article on ear anatomy.

Disorders of the vestibular system could relay inaccurate movement information to the brain, and this information may not be compatible with sensory information received elsewhere from the body. For example, the semicircular canals could be activated in the absence of head movement, resulting in a false sense of rotational movement where the environment would appear to be spinning around us.

It is important to note that vestibular causes of dizziness can be central or peripheral. The central vestibular system refers to the vestibular components of the brain and brainstem. The peripheral vestibular system refers to the components of the inner ear and the nerves and pathways connecting the inner ear to the central vestibular system. A common misconception may be that all dizziness relates to the peripheral component. See the articles on central vestibular disorders, and the peripheral vestibular system to further explore these systems.

What follows is a rundown of the more common disorders originating from the vestibular system that may cause patients to present with the complaint of “dizziness.” This list is extensive but not exhaustive.

RECURRENT EPISODIC VERTIGO & DIZZINESS

Patients experiencing recurrent episodes of vertigo and dizziness often suffer from one of these four disorders, listed from most common to less common: benign paroxysmal positional vertigo (BPPV), Ménière’s disease, vestibular migraine, and persistent postural perceptual dizziness (PPPD).

DEFINITIONS
Dizziness A sensation of lightheadedness, faintness, or unsteadiness. Dizziness does not involve a rotational component.
Vertigo The perception of movement or whirling, either of the self or surrounding objects.
Disequilibrium Unsteadiness, imbalance, or loss of equilibrium, often accompanied by disorientation.
Spatial disorientation A sensation of not knowing where one’s body is in relation to the vertical and horizontal planes.



Benign Paroxysmal Positional Vertigo (BPPV)

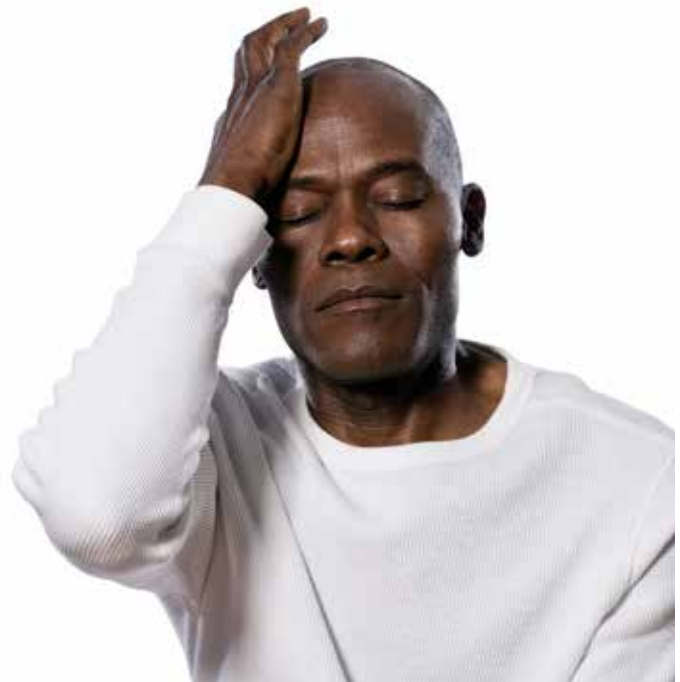
BPPV is the most common peripheral vestibular disorder. BPPV involves displaced free-floating crystals (otoconia) within the semicircular canals which lead to vertigo. Vertigo can be triggered by changes in head position or certain body movements such as getting out of bed. BPPV is typically specific to one side of the head. Which side may be determined by the Dix-Hallpike maneuver, in which the patient's head is turned 45 degrees towards the ear in a seated position on the exam table/chair. The patient is then quickly laid back with the head extended below the head of the table if possible. The patient's eyes are observed for repetitive, uncontrolled movements called nystagmus. The direction of the head when nystagmus is observed indicates the side of BPPV. So, if the patient has nystagmus when their head is turned towards the right ear during the Dix-Hallpike maneuver, this indicates the problem is within the right semicircular canals. Treatment involves maneuvers (e.g. the Epley maneuver) that attempt to return the otoconia to their normal position.

Ménière's Disease & Ménière's Syndrome

The classic triad of symptoms for Ménière's disease and Ménière's syndrome consists of episodes of vertigo, hearing loss, and ear ringing/humming (tinnitus). The root cause of these disorders is an increased volume of inner ear fluid (endolymph) referred to as endolymphatic hydrops. When the cause of the endolymphatic hydrops is unknown (idiopathic), the disorder is referred to as Ménière's disease. When endolymphatic hydrops can be linked to a specific cause it is termed Ménière's syndrome. Ménière's disease is progressive, and no cure exists. Treatment for Ménière's disease is focused on symptom relief. Treatment options include lifestyle changes, physical therapy, medications, and surgery. Treatment for Ménière's syndrome also focuses on symptomatic relief as well as treating the underlying cause.

Vestibular Migraine

Vestibular migraine can cause episodic vertigo accompanied by migraine features such as headache, photophobia and phonophobia, and visual auras. Patients may also complain of dizziness with nausea, with dizziness in this case referring to "a sensation of disturbed spatial orientation." There is significant overlap between vestibular migraine and Ménière's disease, which complicates making an accurate diagnosis. The mechanism behind vestibular migraine is not well understood. Treatment may



be conservative with efforts to avoid triggers, optimize diet and exercise, and improve sleep. Patients may also benefit from vestibular rehabilitation therapy and a variety of medications, including but not limited to selective serotonin reuptake inhibitors (SSRIs), beta-blockers, calcium channel blockers, triptans, and non-steroidal anti-inflammatory drugs (NSAIDs). Episodes of vertigo may require anti-nausea and anti-vertigo medications. The prognosis for vestibular migraine is not well described, however, studies suggest that most patients will continue to suffer from vestibular migraine long-term with varying reductions in the frequency of migraines and severity of symptoms.

Persistent Postural Perceptual Dizziness (PPPD)

PPPD involves waxing and waning periods of dizziness, unsteadiness, or non-spinning vertigo that is typically made worse by upright posture, active or passive motion, self-motion, and complex visual stimuli (e.g. a wavy carpet pattern). The mechanism driving PPPD is not well understood but it appears that psychological factors play a major role in both its onset and persistence. Additionally, research suggests that patients with PPPD rely strongly on visual information, which may explain why busy patterns worsen symptoms. There is no specific test to diagnose PPPD, but clinicians may utilize vestibular testing to eliminate other potential causes of episodic vertigo. Treatment options include medication (e.g. SSRIs or SNRIs), vestibular rehabilitation therapy, and psychotherapy (talk therapy).

NON-EPISODIC VERTIGO & DIZZINESS

Vestibular Neuritis

Vestibular neuritis is the third most common cause of vertigo in the peripheral vestibular system. This disorder is thought to be caused by a viral or bacterial infection of the vestibular nerve, and the inflamed nerve sends atypical signals to the brain. This causes patients to experience sudden, severe vertigo that lasts more than 24 hours up to a week. Patients may also experience nausea, vomiting, and issues with balance. In contrast to labyrinthitis, patients with vestibular neuritis do not experience hearing loss, as the cochlea is not involved. Treatment options include medications (e.g. steroids, antivirals, antibiotics, anti-nausea medications, antiemetics) and vestibular rehabilitation therapy. It may take several months for balance and position to recover and sometimes over a year.

Labyrinthitis

Labyrinthitis, like vestibular neuritis, may be caused by a viral or bacterial infection. However, this infection involves both the vestibular and cochlear organs, resulting in vertigo, nausea, vomiting, hearing loss, tinnitus, and balance issues. The vertigo, or “dizziness,” component of this disorder occurs due to inflammation in the vestibular system. Treatment options are like those used to treat vestibular neuritis. The vertigo in labyrinthitis typically resolves within a few days, but other mild symptoms may last several weeks.

Vestibular Schwannoma (Acoustic Neuroma)

A vestibular schwannoma, also known as an acoustic neuroma, is a slow-



growing, benign mass that originates from the Schwann cells supporting the vestibulocochlear nerve (cranial nerve VIII). Schwann cells are cells in the peripheral nervous system that support the nerve cells in our nervous system. Their uncontrolled growth can form a mass that can compress the vestibulocochlear nerve leading to vertigo, tinnitus, hearing loss, and instability. Hearing loss is the most common symptom of patients with a vestibular schwannoma. MRI typically makes the diagnosis. Treatment options include observation, surgery, and radiation. Patients may have permanent symptoms following treatment.

Superior Semicircular Canal Dehiscence Syndrome (SSCDS)

SSCDS is caused by a defect in the base of the skull where the bone has a hole or is too thin. This bony defect functions as an unwelcome third window of the inner ear resulting in changes of pressure and sound. Patients with SSCDS may experience dizziness or vertigo after experiencing loud noises (Tullio phenomenon), tinnitus, and chronic instability. Diagnosis is made based on symptoms, vestibular testing, and CT imaging. The first line of treatment for this disorder is surgery.

Perilymphatic Fistula

A perilymphatic fistula is the result of an abnormal opening between the middle and inner ear. The fistula could be due to a congenital defect, trauma to the head or inner ear, barotrauma, or a complication of inner ear surgery. Patients may experience sudden single-sided hearing loss, vertigo, tinnitus, ear fullness, and imbalance. Perilymphatic fistulas may be diagnosed by the detection of inner ear fluid on laboratory testing or during surgical exploration. CT and MRI could also assist with diagnosing a fistula. Treatment depends on the cause and extent of the fistula. Options include conservative management if no cause can be identified or surgical if the cause is known.

Cholesteatoma

A cholesteatoma is a benign mass of keratinized skin cells that collects within the middle ear or mastoid. Middle ear cholesteatomas can occur without any inciting event (congenital, i.e. exists at birth) or may be acquired through tympanic membrane perforations (holes), recurrent middle ear infections, eustachian tube dysfunction, etc. Cholesteatomas can invade inner ear structures leading to dizziness and other vestibulocochlear symptoms. Methods to diagnose cholesteatoma include ear examination, audiometric testing, CT, and MRI. The primary treatment is surgery. Unfortunately, patients may experience cholesteatoma recurrence even after surgery.

Mal de Débarquement Syndrome

Mal de Débarquement Syndrome (MdDS), translated to “sickness of disembarkment,” is a rare cause of dizziness that usually occurs within 48 hours of disembarking from a moving vessel (e.g. car, boat, plane). The dizziness in this disorder is persistent and may be described as “rocking,” “swaying,” or “bobbing.” The cause of MdDS remains a mystery, however, imaging studies have uncovered metabolic differences in certain areas of the brain in patients with MdDS. The diagnosis is almost entirely based on



the patient's history but the elimination of other diseases through audiometric testing and imaging may prove useful. Treatment options include medications, vestibular rehabilitation therapy, and neuromodulation techniques.

Vertebral Artery Compression Syndrome (VACS)

This disorder is caused by the vertebral artery compressing the vestibulocochlear nerve. Patients may experience vestibular symptoms as well as hearing loss and tinnitus. Surgery may be required to relieve pressure on the nerve. Symptoms may be temporary or permanent depending on how long the nerve was compressed.

Otosclerosis

Otosclerosis is a disorder marked by abnormal bone growth in the middle ear. Patients primarily present with conductive hearing loss due to the involvement of the middle ear bones (ossicles), particularly the stapes. Otosclerosis is associated with vertigo as well. However, the cause of vestibular symptoms in this disorder is unclear. The primary treatment of otosclerosis is surgery (e.g. stapedectomy) but medications may be required to treat the vestibular component associated with this disorder.

Medication Toxicity (Ototoxicity)

There are several medication classes associated with damage to the ear (ototoxicity): aminoglycosides (e.g. gentamicin), loop diuretics (e.g. Lasix), NSAIDs (e.g. ibuprofen), and acetylsalicylic acid (aspirin). Patients may experience hearing loss, tinnitus, vertigo, dizziness, and imbalance. These medications should be discontinued if determined to be the cause of audiovestibular symptoms.

Autoimmune Disease

Autoimmune disease is the result of the body's immune system attacking healthy body tissues. The inner ear may be the specific target of the attack or could be a casualty of a systemic autoimmune process. Either cause can produce vestibular symptoms. Systemic autoimmune diseases implicated in the damage of the vestibular system include but are not limited to, multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, Sjögren syndrome, and Cogan's syndrome. These disorders are typically treated by primary care physicians and rheumatologists. Medications capable of dampening the immune response are the mainstay treatment for autoimmune disease (e.g. corticosteroids, disease-modifying antirheumatic drugs, tumor necrosis factor inhibitors).

Stroke

A stroke is a condition marked by neurological deficits such as sudden numbness or weakness of the face or limbs, sudden confusion or difficulty speaking, sudden changes in vision, sudden severe headache, or sudden dizziness, imbalance, or vertigo. Strokes are caused by a lack of oxygen being delivered to the central nervous system, and there are two types of strokes: ischemic and hemorrhagic. Ischemic stroke is the most common and occurs when the blood vessels supplying the central nervous system



Strokes can damage the vestibular system temporarily or permanently, and patients may experience dizziness, vertigo, imbalance, unsteadiness, and more. The treatment of stroke depends on the type, location, duration, and extent of the injury. Recovery is also highly dependent on the characteristics of the stroke. Most importantly, patients can take steps to reduce their risk of stroke by controlling certain risk factors such as diet, exercise, high blood pressure, diabetes, and high cholesterol.

Trauma

NON-VESTIBULAR CAUSES OF DIZZINESS

The origins of dizziness complaints are not confined to the central and peripheral vestibular systems. Nearly all body systems can be associated with dizziness in some way. For example, dysfunction of the urinary system (kidneys, ureters, bladder, and urethra) could result in excessive loss of fluids, thus leading to dehydration, a very common cause of dizziness. Given the never-ending causes of nonvestibular dizziness, this section will discuss only a handful of etiologies.

Cervicogenic Dizziness

The name of this disorder, cervicogenic dizziness, means dizziness produced from the cervical spine or neck region. The neck and cervical spine relay information about the movement and position of the head relative to the body (proprioception). In combination with the vestibular system, this information is used to maintain balance and equilibrium. Neck muscle spasms or fatigue, neck pain, cervical degenerative disease, and neck trauma could alter signals from the neck region and cervical spine, and when it does not match up with the signals from the inner ear (vestibular system) patients may experience vertigo, dizziness, and unsteadiness. Diagnosis requires a combination of patient history, physical testing, laboratory testing, and imaging. Treatment of cervicogenic dizziness is similar to the treatment of neck pain, which may include medications, physical therapy, or surgery.

Visual Disorders

The influence of the visual system plays a significant role in our balance, as seen in PPPD, where complex visual information can induce episodes of vertigo. Sensory information from the eyes is combined with vestibular and proprioceptive (muscles and joints) information to maintain balance and a sense of equilibrium. Therefore, disorders of the eyes or visual pathways can produce dizziness and disequilibrium. Perhaps the simplest example is the loss of close-up vision as we age, referred to as presbyopia. In presbyopia, close objects may be out of focus and the blurred vision may induce a sense of dizziness or disequilibrium. Visual changes should be discussed with a primary care physician, ophthalmologist, or optometrist.

Autonomic Nervous System (ANS) Dysfunction

The ANS is a component of our nervous system that controls involuntary functions such as breathing, blood pressure, heart rate, digestion, salivation, and many more. Orthostatic hypotension is a common consequence of ANS dysfunction where the blood pressure drops after going from sitting to standing. Normally, upon standing the blood vessels constrict in the lower body to maintain blood flow to the upper portion. When the vessels do not constrict, gravity allows blood to pool, resulting in inadequate blood flow from the heart to the brain. Low blood flow to the brain can cause dizziness or lightheadedness. Causes of orthostatic hypotension include old age, dehydration, blood loss, medications, and neurodegenerative disorders such as Parkinson's disease.



Medication-induced Dizziness

Dizziness is a common side effect of medications, and the mechanism for dizziness varies. Classes of medications that can cause dizziness include but are not limited to, antihypertensives, antibiotics, SSRIs, tricyclic antidepressants, and diuretics. Medications should be reviewed with a physician to determine if there is a potential for a side effect of dizziness.



Hyper- & Hypoglycemia

Both high blood sugar (hyperglycemia) and low blood sugar (hypoglycemia) can lead to dizziness. Hyperglycemia may be caused by endocrine disorders (e.g. diabetes), medications, and stress.

Longstanding hyperglycemia can damage the blood vessels and the ANS, which can cause dizziness. Interestingly, there is also an association between diabetes and damage to the peripheral vestibular system, so it is important to realize that hyperglycemia can generate both vestibular and nonvestibular dizziness. The brain requires sugar to function properly, and therefore, hypoglycemia can cause patients to feel dizzy, off balance, or lightheaded. Control of blood sugar should be discussed with a physician to prevent complications.

Cardiovascular Disorders

The flow of blood throughout the body is controlled primarily by the heart and the vessels that make up the cardiovascular system. There are an impressive number of disorders that affect the cardiovascular system, including heart attack, pulmonary embolism, cardiomyopathy, atrial fibrillation, and aortic stenosis. When proper blood flow to the brain is disrupted, patients may experience dizziness, lightheadedness, and loss of consciousness (syncope). Patients experiencing dizziness potentially related to the cardiovascular system should consult their primary care physician or cardiologist.

Respiratory Disorders

The system responsible for gas exchange (e.g. oxygen and carbon dioxide) is referred to as the respiratory system, and this system has two parts: an upper respiratory tract and a lower respiratory tract. The former is made up of the nose, nasal cavity, and throat (pharynx) and the latter is made up of the windpipe (trachea), airway branches (bronchi), and lungs. Respiratory diseases such as asthma, COPD, pneumonia, and pulmonary fibrosis, can disrupt gas exchange causing dizziness, lightheadedness, and presyncope (i.e. the feeling before you lose consciousness) or syncope (fainting). Respiratory disorders may be evaluated and treated by primary care physicians or respiratory specialists (pulmonologists).

Disuse Equilibrium/Deconditioning

This cause of dizziness primarily affects the elderly population. With periods of inactivity, the body can go through a detrimental functional



change known as “deconditioning.” Patients who have been immobilized for a significant period (e.g. hospitalization, bed bound after surgery) may experience dizziness, lightheadedness, and presyncope because of deconditioning. Physical rehabilitation may be necessary to return patients to a healthy level of functioning.

Autoimmune Thyroid Disease

Thyroid disease is a widespread endocrine disorder and the most common autoimmune disease in the US. Both an overactive thyroid (hyperthyroidism) and an underactive thyroid (hypothyroidism) can lead to a sense of dizziness and lightheadedness. Surprisingly, thyroid disease has been associated with an increased risk of Ménière’s disease. So, what may start as a nonvestibular cause of dizziness could potentially lead to a vestibular cause of dizziness. Thyroid disease is managed by a primary care physician or endocrinologist.

Psychogenic Dizziness

There is a close relationship between psychiatric disorders and dizziness or vertigo. Patients with mental disorders such as anxiety or depression are more likely to experience dizziness or vertigo and vice versa. This is thought to be due to the overlap of pathways involved in psychiatric disorders and the central vestibular system. As a result, patients with vestibular disorders and a coexisting psychiatric disorder may experience more debilitating vestibular symptoms. Treatment plans for psychogenic dizziness may include medications, talk therapy, vestibular rehabilitation, and patient education.

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Vestibular Symptoms

By The Vestibular Disorders Association

ARTICLE 120

OVERVIEW

The vestibular system includes the parts of the inner ear and brain that help control balance and eye movements. If the system is damaged by disease, aging, or injury, vestibular disorders can result, and are often associated with one or more of these symptoms, among others:

Dizziness

A sensation of lightheadedness, faintness, or unsteadiness.

Imbalance: Unsteadiness or loss of equilibrium that is often accompanied by spatial disorientation.

Vertigo

A rotational, spinning component, and is the perception of movement, either of the self or surrounding objects.

Brain fog

When the brain is dedicating a great deal of energy to maintain equilibrium and stay steady, activities such as recalling details or short-term memory may become more difficult, and thinking might seem “slow”.

Tinnitus

Abnormal noise perceived in one or both ears or in the head. May be intermittent or continuous and can be experienced as a ringing, hissing, whistling, buzzing, or



clicking sound and can vary in pitch from a low roar to a high squeal.

Hearing loss

Reduction in the ability to hear sounds is a common symptom of many vestibular disorders. When VeDA conducted a patient poll, over two thirds reported that they had hearing loss in one or both ears.

Vision impairment

The link between the vestibular system and vision, vestibulo-ocular reflex (VOR), is described in detail with information on evaluation, treatment, coping strategies, and potential solutions for vision correction, including glasses and contact lenses.

Nausea

The feeling of being nauseated.

Cognitive changes

Difficulty thinking, paying attention/ concentrating, recalling basic facts (such as your own phone number), short-term memory loss, etc.

Psychological changes

Due to the unpredictable nature of symptoms and the chronic nature of most disorders, vestibular patients tend to suffer from anxiety and/or depression.

Motion sickness

Symptoms appear when the central nervous system receives conflicting messages from the visual system and the vestibular system in the inner ears.

Derealization

Includes feelings of being alienated from or unfamiliar with your surroundings, emotionally disconnected from people you care about, and/

POSSIBLE VESTIBULAR SYMPTOMS

Dizziness

Vertigo

Brain Fog

Tinnitus

Hearing Loss

Visual Impairment

Nausea

Cognitive Changes

Psychological Changes

Motion Sickness

Derealization

Depersonalization



or distortions in time or size and shape of objects.

Depersonalization

Feelings that you're an outside observer of your thoughts, feelings, your body or parts of your body; emotional numbness.

TYPES OF VESTIBULAR DISORDERS

There are many conditions that affect the vestibular system and cause vertigo, dizziness, imbalance, and the other symptoms listed in this article. Many people find it helpful to learn about the clusters of symptoms that go along with each diagnosis, as well as the treatment options that can help you learn to live a life rebalanced.

Learn more at vestibular.org/disorders

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Peripheral System

By Brian Keith, B.S. & Habib Rizk, MD

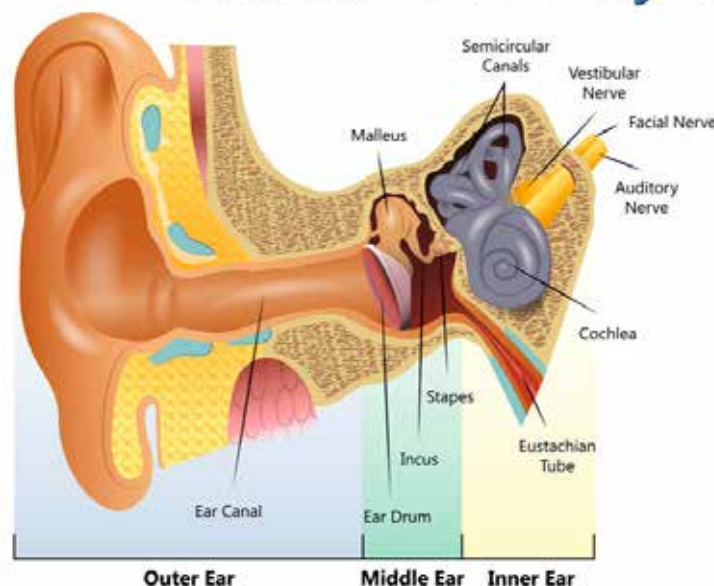
ARTICLE 048

There are two components of the vestibular system: the central system (brain and brainstem) and the peripheral system (inner ear and nerves to brainstem). The inner ear has two primary components: the cochlea and the vestibular apparatus (semicircular canals, utricle, and saccule).

The cochlea is responsible for hearing and the vestibular apparatus is responsible for balance and position.

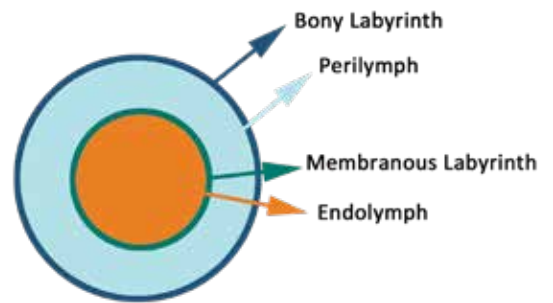
These structures are housed within a hollowed cavity of the human skull known as the bony labyrinth. The bony labyrinth contains a fluid called perilymph (peri-: around or about). Suspended in perilymph is another set of ducts and chambers called the membranous labyrinth. The membranous labyrinth is filled with endolymph (endo-: inside or within).

Human Ear Diagram



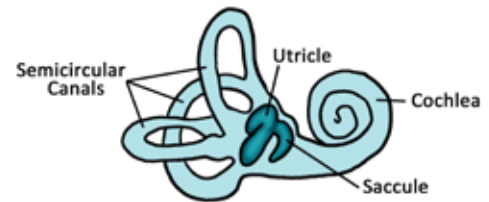
This system can be thought of as a tube within a tube.

The two fluids contain similar chemicals including sodium and potassium, but their compositions are very different. If the composition of either fluid is disturbed, as in the case of Ménière disease, patients may experience vestibular symptoms (dizziness, vertigo, imbalance) or other ear symptoms (ringing/humming, fullness, hearing loss).



THE ANATOMY OF THE VESTIBULAR APPARATUS

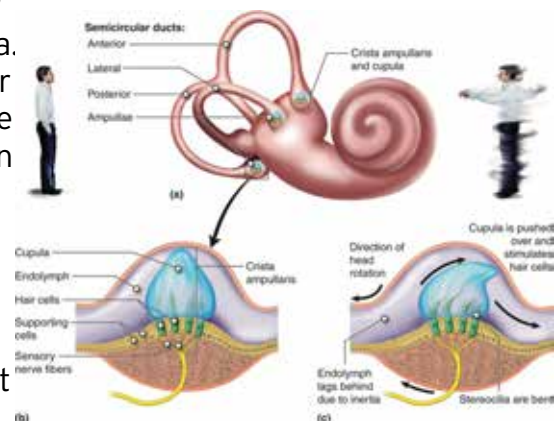
The vestibular apparatus is the most outer part of the peripheral vestibular system. It consists of three semicircular ducts responsible for detecting rotational movement of the head and the otolithic organs (utricle and saccule) that detect linear motion of the head.



THE SEMICIRCULAR DUCTS

The semicircular ducts are a part of the membranous labyrinth within the bony semicircular canals that detect the rotational movement of the head about the axes of motion (X, Y, and Z). There are three semicircular ducts: superior, lateral, and posterior. Each semicircular duct detects the motion of the head around a different axis. The superior semicircular duct detects rotational movement around the frontal, or X, axis (e.g. nodding your head to indicate "yes"). The lateral semicircular duct detects movement around the vertical, or Y, axis (e.g. shaking of the head to indicate "no"). The posterior semicircular duct detects movement around the anterior-posterior, or Z, axis (e.g. touching your ear to your shoulder).

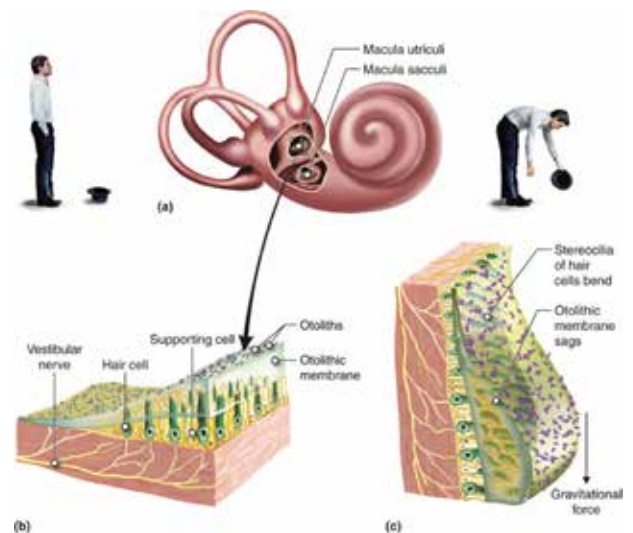
Located at the base of each semicircular duct is a widened area called the ampulla. The ampulla contains the cupula and hair cells. The cupula is a gelatinous structure that spans the entire duct, and its motion is coupled to the hair cells. The hair cells are receptor cells that have hairlike projections called hair bundles that extend into the cupula. When the head moves, the endolymph lags behind due to inertia, which creates a fluid wave that collides with the cupula. The deflection of the cupula, and the hair bundles,



activates the hair cells, sending a signal to the brainstem via the vestibular nerve, a branch of the vestibulocochlear nerve. This is how the semicircular ducts detect and transmit rotational movement information to the central nervous system to maintain balance and position. In addition, this information is used by our brain to carry out corresponding eye movements as our head turns to maintain forward vision. Each ear has a matching set of semicircular ducts and canals, and they work in tandem. Vestibular symptoms may be seen in semicircular duct disturbances such as benign paroxysmal positional vertigo (BPPV), superior canal dehiscence syndrome (SCDS), and alcohol positional nystagmus.

THE UTRICLE & THE SACCULE

The utricle and the saccule are involved with the linear motion of the head. These two membranous structures, referred to as the otolithic organs, are the sole residents of the vestibule, a hollow cavity located between the cochlea and the semicircular canals. The utricle detects linear motion in the horizontal plane (e.g. accelerating/decelerating in a car) and the saccule detects linear motion in the vertical plane (e.g. movement in an elevator).



The otolithic organs each contain a single curved structure called the macula that contains receptor cells known as hair cells and additional supporting cells. Like the mechanism of the semicircular ducts, these hair cells contain tiny hairlike projections. These projections extend into a gelatinous membrane called the otolith membrane. Resting on top of this membrane are microscopic crystals made of calcium carbonate called otoconia. These are commonly referred to as “ear rocks” or “ear stones.” Upon linear motions of the head, these tiny crystals will drag along the otolith membrane and deflect the hairlike projections of the hair cell. This deflection allows the hair cells of the otolith membranes to fire off a signal to the vestibular nerve and communicate with the brain that the head is moving in the horizontal and/or vertical plane.

Dysfunctions of the otolithic organs may lead to vestibular disorders such as BPPV. It can also cause symptoms of brain fog and imbalance. It is not uncommon to have a dysfunction of those organs (or of their central pathways) in vestibular migraine patients.



THE VESTIBULAR NERVE

The information of the inner ear is relayed to the brainstem via the vestibulocochlear nerve which is the eighth cranial nerve (CN VIII). This nerve is considered part of the peripheral vestibular system until it enters the brainstem. The vestibulocochlear nerve is formed by the vestibular nerve and the cochlear nerve after they leave the inner ear. The vestibular nerve is divided into a superior and inferior portion. The superior vestibular nerve relays information from the anterior semicircular canal, horizontal semicircular canal, saccule, and part of the utricle. The inferior vestibular nerve serves the posterior semicircular canal and most of the utricle. Vestibular disorders may involve one portion of the vestibular nerve or both. Vestibular neuritis most commonly affects both portions but is more likely to affect the superior vestibular nerve when only one is involved. When the superior branch is affected, it is not uncommon for patients to develop BPPV a few weeks after their vestibular neuronitis presentation.



Each ear has a vestibular nerve, and they work together. Disruption of the vestibular nerve can lead to vestibular symptoms such as vertigo, dizziness, and imbalance. These symptoms can last from hours to days until the brain adapts or the nerve heals. Sometimes vestibular rehabilitation may be needed to restore the function of the peripheral vestibular system or adapt the way the brain interprets the information.

OTHER STRUCTURES

The oval window and the round window are separate membranous structures that lie between the inner and middle ear. These two structures are involved in the transmission of sound waves from the middle ear to the inner ear. Both the oval window and round window are potential sources of a perilymphatic fistula. In this condition, fluid from the inner ear leaks into the middle ear, and the disturbance of fluid content and volume can lead to vestibular symptoms.

THE FUNCTION OF THE PERIPHERAL VESTIBULAR SYSTEM

As previously mentioned, the vestibular system is responsible for maintaining balance and position. Several involuntary responses (reflexes) maintain these functions by stabilizing the body and vision. These include the vestibular ocular reflex, vestibular colic reflex, and vestibular spinal reflex.



Vestibular Ocular Reflex (VOR): The vestibuloocular reflex (VOR) is a reflex involving the vestibular and visual pathways that stabilizes the eyes during head motions. This is one of the fastest reflexes in the human body as the speed is necessary to maintain clear vision. As the head moves, the eyes move in equal and opposite directions through the VOR. When running and walking, the head is constantly bobbing and swaying yet we can maintain clear and steady vision. Without the VOR, objects may appear to jiggle or vibrate, and this illusion of movement is referred to as oscillopsia.

There are many methods of testing the VOR including the head-impulse test (HIT)/head thrust test. This test may be performed manually or with the assistance of high-speed cameras. When assessed manually, the clinician will thrust the patient's head in one direction and see if the patient can maintain a steady, forward gaze. In a video head impulse test (vHIT), video goggles are placed on the patient to track eye stabilization during head movements. This method provides quantitative, objective data for the VOR.

Vestibular Collic Reflex (VCR): The vestibulocollic reflex (VCR) is the activation of neck musculature in response to vestibular signals which stabilizes the head. The VCR can be evaluated by vestibular evoked myogenic potential (VEMP) testing. VEMP testing involves the stimulation of one ear with a sound click or pulse while simultaneously measuring the activity of neck muscles (cervical VEMP) or eye muscles (ocular VEMP) in response to stimulation. VEMP testing may be used to assist with the diagnosis of superior canal dehiscence syndrome, Ménière's disease, and vestibular migraine.

Vestibular Spinal Reflex (VSR): The vestibulospinal reflex (VSR) is an interaction between vestibular signals from the inner ear and spinal nerves. This reflex produces skeletal muscle contractions opposite to the vestibular information to stabilize the body and maintain posture. For example, if our torso is pushed forward from behind, the vestibular system relays information to the spine, and back muscles are activated to prevent a forward fall. Without the VSR, humans would have a much greater risk for falls. The VSR can be tested through computerized dynamic posturography.

SUMMARY

In this article, we have reviewed the anatomy of the peripheral vestibular system and its function. There are several complex reflexes controlled by the vestibular system, and the status of these reflexes can assist clinicians with diagnosing or ruling out certain vestibular disorders. For more information on vestibular testing please see the VeDA article, Tests for Diagnosing Vestibular Disorders.

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Central vs. Peripheral

By Brian Keith, B.S., Neil Monahan, & Habib Rizk, MD, MSc

ARTICLE 012

OVERVIEW

Determining the source of dizziness can be difficult given the overwhelming number of causes. The source may be vestibular or non-vestibular in origin which is discussed in the article, "Causes of Dizziness." When the vestibular system is suspected, it is important to determine if the dizziness comes from the central vestibular system (brain and brainstem) or the peripheral vestibular system (labyrinth, nerves, and pathways from the inner ear to the brainstem). This allows the clinician to narrow the list of potential causes. In this article, we explore the signs and symptoms of central and peripheral vestibular disorders to emphasize the importance of each patient's presentation when determining the origin of their vestibular symptoms.

REVIEW OF TERMINOLOGY

"Dizziness" or "dizzy" are often catch-all terms used by patients to explain their vestibular symptoms. Patients may also use the term "vertigo" when another term may be more appropriate. To improve this confusion, The Bárány Society, an international society for vestibular research, defined the following terms:

- Dizziness: The sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion.
- Vertigo: The sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement.
- Unsteadiness: The feeling of being unstable while seated, standing, or walking

Other symptoms such as imbalance, tinnitus, and hearing loss are defined in the article, "Vestibular Symptoms."



FEATURES OF CENTRAL & PERIPHERAL VESTIBULAR DISORDERS

Several elements of a patient's history may help a clinician determine if their symptoms stem from a central or peripheral vestibular disorder. This includes the timing and triggers of symptoms, characteristics of symptoms, and ear involvement.

FEATURES OF CENTRAL & PERIPHERAL VESTIBULAR DISORDERS

Several elements of a patient's history may help a clinician determine if their symptoms stem from a central or peripheral vestibular disorder. This includes the timing and triggers of symptoms, characteristics of symptoms, and ear involvement.

TIMING & TRIGGERS OF SYMPTOMS

When taking the history, a clinician may inquire about the timing of vestibular symptoms. Did the symptoms come on suddenly? Do they suddenly recur (paroxysmal)? Do symptoms come and go (episodic)? How long do the symptoms last (duration)? If continuous, does the severity fluctuate? Answers to these questions will help guide the clinician to a diagnosis. For example, episodic symptoms tend to be associated with peripheral disorders, but they could also be due to a central disorder like vestibular migraine or vestibular paroxysmia. Peripheral disorders generally result in sudden vestibular symptoms but central disorders like a stroke could cause sudden symptoms as well. In summary, assessing the timing of symptoms is more useful for differentiating certain disorders rather than differentiating central versus peripheral.

TABLE 1: MODIFIED VERSION OF TiTrATE APPROACH

TIMING-TRIGGER CATEGORY	DIFFERENTIAL DIAGNOSIS (ABBRIEGED)	MOST IMPORTANT DIAGNOSTIC TOOL
Episodic, positional	Orthostatic hypotention, BPPV, Central positional vertigo	Vital signs, Dix Hallpike Test
Episodic, spontaneous	Migraine or Meniere's, Hypoglycemia, TIA, Cardiac arrhythmia	History, Hearing test
Acute/continuous, post-exposure	Drug intoxication, Trauma or post-op, carbon monoxide or Wernicke encephalopathy due to alcohol	History
Acute/continuous, spontaneous	Vestibular neuritis, Labyrinthitis, Herpes Zoster, Stroke or Hemorrhage, Brainstem encephalitis	Thorough vestibular exam, hearing test



The triggers for vestibular symptoms vary for each disorder. Peripheral vestibular disorders may be triggered by events like sitting up suddenly (BPPV), seeing a visually complex pattern (PPPD), or viral upper respiratory infection (vestibular neuritis). Central vestibular disorders are variable as well. For instance, a (posterior circulation) stroke may be sudden and come on without warning, whereas a vestibular migraine could have warning signs (auras). Patients with vestibular migraine may experience visual auras, which can be experienced as lines, floaters, spots, stars, or flashes of light in your field of view before the onset of migraine and vestibular symptoms. However, vestibular migraine can also present without warning signs. Patients who have had Ménière's disease or vestibular migraine for a while sometimes start noticing early signs or symptoms (prodromes) that tell them they are about to be dizzy. In Ménière's disease, hearing loss, fullness, and tinnitus can come before vertigo attacks sometimes by several days.

The timing of vestibular symptoms may be the most important metric when considering the diagnosis followed by triggers. For instance, vestibular migraine can present with episodic vertigo that is not triggered, whereas BPPV presents with sudden (paroxysmal) episodes triggered by changes in head position, and vestibular hypofunction can cause symptoms of dizziness and visual blurriness with head movement (rather than position). Dr. David Newman-Toker and Dr. Jonathan Edlow suggested an approach based on "symptom timing, triggers, and targeted bedside eye examinations (TiTrATE)."

TABLE 2: GENERALIZED SYMPTOM PROFILES OF CENTRAL & PERIPHERAL VESTIBULAR DISORDERS

BENIGN CENTRAL DISORDERS (MdDS, VESTIBULAR MIGRAINE)	URGENT CENTRAL DISORDERS (STROKE, TUMOR, ENCEPHALITIS)	PERIPHERAL VESTIBULAR DISORDERS
Triggers like migraine aura, passive motion exposure such as boating or long car rides leading up to vestibular symptoms.	Signs of stroke (5 "D's"), altered mental status	Viral illness, positional changes, visual stimulations leading up to vestibular symptoms.
Non-spinning vertigo, rocking sensations	Severe, acute spinning vertigo (posterior fossa stroke)	True vertigo, more severe nausea and vomiting
Ear symptoms (hearing loss, tinnitus, fullness) are two-sided > one-sided		Ear symptoms are one-sided > two-sided
Slower progression, more likely to be constant		Episodic, less than 24 hours



CHARACTERISTICS OF SYMPTOMS

Although both peripheral and central vestibular disorders may present with any vestibular symptom, there are certain characteristics of the symptom profiles that may point to one over the other. Vertigo of a central origin tends to be associated with more severe imbalance but less nausea. In central disorders, the imbalance may be so significant that patients cannot walk or stand.

Central causes may also be accompanied by neurologic signs including dysphonia (difficulty speaking), dysphagia (difficulty swallowing), and diplopia (double vision); these three symptoms plus dystaxia (abnormal balance) and dizziness are known as the “Five D’s” which are classic symptoms in a posterior circulation stroke. On the other hand, patients with a peripheral vestibular disorder may present with more severe nausea and vomiting as well as auditory symptoms of tinnitus, hearing loss, ear fullness, or ear pain.

Lastly, patients with peripheral vestibular disorders are more likely to present with spinning external vertigo (seeing objects spinning around them). In contrast, central vestibular disorders are more strongly associated with false vertigo (dizziness, lightheadedness, unsteadiness). Of note, a rocking sensation is rarely related to an inner ear disorder. While it is vertigo in the true sense of the term (illusion of movement) it is sometimes described as a non-spinning vertigo. MdDS, vestibular migraine, and PPPD are hallmark causes of ‘rocking’ dizziness sometimes called motion-modulated oscillatory vertigo.

EAR INVOLVEMENT

Patients with central vestibular disorders such as vestibular migraine are more likely to experience ear symptoms on both sides (bilateral) rather than one side (unilateral). Ear symptoms are also more likely to be constant until their resolution whereas in peripheral disorders they may come and go (episodic). Peripheral vestibular disorders also tend to slowly progress through different stages, as seen in Ménière’s disease.

SIGNS OF CENTRAL & PERIPHERAL VESTIBULAR DISORDERS

In addition to symptoms, clinicians may consider the observable manifestations of the patient’s vestibular disorder (signs) when determining if the origin is central or peripheral. Perhaps the most important sign to discuss is nystagmus. Some tests, like headshaking, rely on nystagmus. Other features of a central vestibular disorder worth discussing include abnormalities in smooth pursuit testing and saccade testing. There is a wide range of vestibular testing available to clinicians and this is discussed in the article, “Tests for Diagnosing Vestibular Disorders.”

NYSTAGMUS

Nystagmus, as defined by the book *The Neurology of Eye Movements*, is “a



repetitive, to-and-fro movement of the eyes that is initiated by slow phases." The movement of the eyes may be from side-to-side (horizontal), up-to-down or down-to-up (vertical), or in a circle (torsional). Nystagmus may occur in one direction (unidirectional) or both directions (bidirectional). Vertical and torsional nystagmus are classically associated with central disorders whereas horizontal nystagmus is more widely associated with peripheral disorders. The direction of nystagmus is typically fixed in peripheral disorders but may change direction in central disorders. Nystagmus could be unprovoked or produced by provocative testing such as head shaking and positional maneuvers.

HEADSHAKE TEST

The test is performed by having the patient sit upright, close their eyes, and then shake their head horizontally or vertically. The patient will then open their eyes and allow the clinician to observe for active-head shaking nystagmus (AHSN).

In peripheral vestibular disorders, the direction of AHSN typically will match up with the direction of the head shake test. So, if the direction of AHSN is horizontal following a horizontal head shake test, this would support a peripheral vestibular disorder. On the other hand, AHSN may occur perpendicular (at a 90 degree angle) to the head shake test in a central vestibular disorder. For example, the examiner shakes the head left and right but observes the eyes rapidly beating up and down, and this may suggest to the clinician that the vestibular symptoms are central rather than peripheral. In addition, in a peripheral vestibular weakness, AHSN typically would show the eye beating in the direction of the better ear. The observations of this test are not always black-and-white and will be used in conjunction with other testing. AHSN can be seen in central disorders as well and is not always localizing. Vestibular migraine patients can have a downbeat after AHSN following a

TABLE 3: GENERALIZED SIGNS OF CENTRAL & PERIPHERAL VESTIBULAR DISORDERS

CENTRAL VESTIBULAR DISORDERS	PERIPHERAL VESTIBULAR DISORDERS
Vertical or torsional nystagmus	Horizontal nystagmus
Direction of nystagmus is variable	Direction of nystagmus is fixed
Headshake nystagmus perpendicular to direction shake	Headshake nystagmus matches direction of shake
Abnormal smooth pursuit test	Normal smooth pursuit test
Abnormal saccade test	Normal saccade test



horizontal head shake test.

SMOOTH PURSUIT TESTING

The smooth pursuit system of the eyes allows us to track objects as we move or the object moves. Clinicians may evaluate this by having a patient follow a dot on a screen that moves slowly from one side of the screen to the other while the patient's eye movements are tracked. Rapid jerking motions of the eyes, or saccades, are more likely to be seen in central vestibular disorders during the smooth pursuit test. Patients with a peripheral vestibular disorder will likely have normal smooth pursuit testing. As with the head shake test, smooth pursuit testing is not a surefire method of diagnosing a central versus peripheral disorder.

SACCADE TESTING

Saccade testing assesses the voluntary, rapid movements of the eye. For saccade testing, a patient may be asked to follow a dot on a screen that will quickly change position without moving their head. This test allows the clinician to calculate the lag time, accuracy, and speed of eye movements. Abnormal saccade testing is more likely to be seen in central disorders.

CONCLUSION

In this article, we have summarized the elements of a vestibular patient's disease that will help determine whether the signs and symptoms are of central or peripheral origin. Although this information may be used to narrow down the origin of your vestibular disorder, it is only one piece of the puzzle. If questions remain, please consult your otolaryngologist for additional information.

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Vestibular Injury

By Thomas Boismer, MPH updated by Kamran Barin, PhD

ARTICLE 078

The vestibular system includes the inner ear balance organs and the parts of the brain that coordinate and process balance information. The balance organs contribute vital sensory information about motion, equilibrium, and spatial orientation. In addition, the vestibular system helps maintain clear vision during movements via an automated function called the vestibulo-ocular reflex (VOR).

The vestibular system can be compromised in many ways. Viral or bacterial infections (labyrinthitis and vestibular neuritis), disorders that affect inner ear fluid levels (Ménière's disease and secondary endolymphatic hydrops), trauma from head injury (mild traumatic brain injury or mTBI), benign tumors (vestibular schwannoma), and age-related degeneration can all cause permanent damage to the vestibular system.

The body has limited ability to repair damage to the vestibular organs. However, the body can compensate for the vestibular injury by recalibrating the part of the brain that controls balance. This compensation process occurs naturally in most people, but when it is not successful, vestibular rehabilitation therapy may be required.

ACUTE (IMMEDIATE) COMPENSATION

When a sudden injury occurs to the balance organs on one side, the patient may feel very sick for hours to a few days with vertigo (a spinning feeling), lightheadedness (dizziness), and unsteadiness, often accompanied by sweating, nausea, and vomiting. This is because the spatial orientation signals sent to the brain from both ears are no longer equal and opposite, and the brain

WHAT IS COMPENSATION?

Compensation from vestibular injury is accomplished by recalibrating the part of the brain that controls balance.



interprets the difference as constant movement.

Researchers theorize that after this initial period, the brain recognizes that the signals being received from the ears are incorrect and turns the signals down through a process called the cerebellar clamp. When the clamp is in place, the vertigo and much of the sick feelings improve. However, the patient feels unsteady while standing, because the vestibular signals normally used to maintain balance have been reduced.

The patient may also report disorientation or blurred vision during head movements because the VOR no longer functions properly. In addition to the vestibular system, proprioception (the sensors in the muscles, joints and at the bottom of the feet) and vision provides information that is necessary for maintaining balance. So without vestibular input, the patient can still walk but will feel unsteady and may fall in the dark or on soft or bumpy surfaces such as a thickly carpeted floor, grass lawn, or gravel path.

At this point, most patients are well enough to get out of bed and visit a doctor. The doctor sees a person who is not experiencing dizziness or vertigo but whose gait is ataxic, meaning awkward and uncoordinated. If the patient is not given an opportunity to clearly describe what has happened, he or she may be immediately referred to a neurologist to rule out stroke because of this ataxic gait.

The cerebellar clamp may persist for days after the initial injury. If balance testing is performed during this phase, test results may incorrectly suggest that the patient has damage to both sides of the balance system, because the cerebellar clamp reduces the eye movements (VOR) that are typically evaluated during balance testing.

CHRONIC (LONG-TERM) COMPENSATION

After the initial compensation phase, the cerebellum slowly releases the clamp, gradually allowing more signals from the inner ear to pass to the brain. The brain can then fine-tune its calculations in order to account for differences between the signals received from damaged versus undamaged vestibular organs. Such compensation can't occur if the cerebellar clamp remains in place.

For most patients, the movements made during normal daily activities are enough to achieve chronic (long-term) compensation, usually in two to four weeks after the injury has occurred.



Once this chronic compensation process is complete, the patient is essentially symptom-free. If unsteadiness and/or motion-provoked dizziness persist after that time, compensation is not complete and the physician may prescribe a program of vestibular rehabilitation therapy (VRT).

VRT is a treatment program administered by a specially-trained physical or occupational therapist. It is designed to (1) desensitize the balance system to problematic movements and (2) enhance the fine-tuning required for long-term compensation. This is accomplished with exercises that involve small, controlled, and repeated “doses” of the movements and activities that provoke dizziness. VRT is most effective when administered by a therapist who has specific training and experience in this specialized form of therapy.

DE-COMPENSATION

It's important to remember that even after symptoms go away, the balance system remains injured, the brain has simply adapted to the injury. For many patients, dizziness and vertigo will return months or years after compensating for a balance system injury. It is critical for the physician to find out what type of dizziness and vertigo the patient has.

DE-COMPENSATION?

When the brain forgets how it adapted to a vestibular injury. This can happen following a cold, flu, surgery, or other interruption in activities that promote compensation.

If the patient describes another severe attack of spinning with unsteadiness and nausea lasting hours to days, this suggests that a second injury has occurred to the balance system, such as another viral infection or an attack of Ménière's disease or secondary endo-lymphatic hydrops. These conditions require diagnosis and medical treatment.

If the patient reports that dizziness occurs after particular head movements and lasts seconds to a few minutes, this suggests one of two things. It is possible that the patient has developed benign paroxysmal positional vertigo (BPPV), which is common after some vestibular injuries. BPPV can be diagnosed and treated effectively using simple procedures. If the patient's symptoms turn out to be unrelated to BPPV, then the most likely explanation is that de-compensation has occurred. De-compensation simply means that the brain has 'forgotten' the fine-tuning it developed during the chronic compensation phase described above.

Events that can provoke de-compensation include a bad cold or the flu, minor surgery, or anything that interrupts normal daily activity for a few days, such as long trips. Recovery after de-compensation is exactly like the recovery that occurs during the chronic compensation phase. Movements and activities are the stimuli the brain needs to fine-tune the system. We recommend that patients keep their VRT exercise program instructions available even after



they recover so that they can begin the exercises immediately if symptoms return. Usually recovery after de-compensation is quicker than the time it takes to recover after the initial injury to the balance system.

FAILURE TO COMPENSATE

Two things are required in order to compensate for an injury. First, the brain must continue to receive signals from the balance organs so that it can learn how to interpret different head movements. Thus, movements must not be avoided, because they create the signals the brain needs to compensate for the injury. Second, the areas of the brain that are responsible for compensation must not be damaged.

During the early stages of dizziness, patients may avoid quick movements and reduce their activities. Most patients will also be prescribed one or more anti-dizziness medications such as Antivert (meclizine), Valium (diazepam), Xanax (alprazolam), Phenergan (promethazine), or Compazine (prochlorperazin). This is fine during the acute stages of dizziness. However, once the acute phase is past, inactivity and medications can interfere with the long-term compensation process.

Any medication that makes the brain sleepy, including all of the anti-dizziness medications, can slow down or stop the process of compensation, so they are often not appropriate for long-term use. Most patients who fail to compensate are found to either be strictly avoiding certain movements, using anti-dizziness medications daily, or both. Treatment includes VRT and gradual reduction and eventual elimination of these medications.

Brain damage caused by stroke, head injury, or other causes can slow down or stop the natural compensation process. It is difficult to predict which patients with brain injury will improve or to what degree, so all patients should be given the chance to improve through a VRT program. There are several different measures of symptoms and functional capabilities used to assess progress repeatedly as treatment goes on. As long as a patient continues to show improvement, even if it is gradual, treatment should be continued.

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