

TITLE: Balance Function Testing

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Physiology of Balance

Humans use three basic mechanisms to obtain a sense of balance in daily life. The three mechanisms (visual, vestibular, and proprioceptive) interact to maintain posture and impart a conscious sense of orientation. There are measurable reflexes associated with these stimulus modalities. Reflexes generally serve to maintain stability in posture (e.g. by extending muscle groups in the direction of an anticipated fall), or in maintaining stability of the visual field. A defect in one of these systems, or incongruous inputs amongst the systems can be compensated for by reliance on the other two systems. However, such a defect decreases the patient's overall ability to adjust to incongruous stimuli between the other two fields. Also, a defect can result in a serious subjective feeling of disequilibrium in the affected patient until compensation for the deficit occurs.

Visual system:

Visual inputs aid in the maintenance of an upright posture and in orientation. Conscious and unconscious correction of posture is possible through processing of visual inputs. The adjustment of posture and sensation of movement in response to visual stimuli can be seen by observing individuals' responses to optokinetic stimuli (repeated movement of large objects in the subject's visual field). Such stimuli (e.g. a train moving on the adjacent platform) impart a sense of acceleration to the individual and lead to reflexive postural adjustments (e.g. leaning in the direction of the moving train) to maintain balance. Visual reflex arcs also aid in maintaining the stability of the visual field. The saccade system focuses a visual target of interest onto the fovea through a fast movement of the eyes. The smooth pursuit system allows fixation of gaze onto a moving object with a frequency of less than 1.2 Hz. The optokinetic reflex is a result of

multiple objects moving through a patient's visual field (with the moving objects occupying about 80% of the patient's visual field). The optokinetic reflex imparts a sense of motion to the patient. It presents as a jerk nystagmus with the slow component in the direction of the moving objects and the fast component back to the midline.

Proprioceptive system:

Proprioceptive inputs aid in static and dynamic postural control primarily through two reflex arcs. The first is the myotonic reflex (deep tendon reflex), in which stretch on a muscle causes contraction of the muscle. The myotonic reflex serves to maintain stability across a joint. The second proprioceptive reflex arc that aids in posture control is the functional stretch response, which utilizes multiple somatosensory inputs to provide for coordinated limb and trunk movements across joints, for instance to maintain the center of gravity over the support base in an individual who is bumped from behind. This reflex pathway has a higher latency than the myotonic reflex, although both are mediated through spinal pathways. Both of these reflex arcs have lower latencies than visual-postural reflexes and vestibular-postural reflexes.

Vestibular system:

The vestibular system consists of two groups of specialized sensory receptors: the semicircular canal and otolithic organs. The semicircular canal detects angular acceleration of the head. The semicircular canal consists of a membranous semicircle with a widened area, the ampulla, at one end. The ampulla contains the *crista ampullaris*: specialized ciliated cells (several small cilia and one large eccentric kinocilium on each cell) jutting into the lumen of the ampulla. The cilia are embedded in a gelatinous structure called the cupula. The membranous semicircular canal contains endolymph (extracellular fluid with a high potassium concentration) that is the same specific gravity as the cupula. When angular acceleration of the head occurs in the plane of the semicircular canal, the endolymph's momentum causes it to stay relatively stationary, displacing the cupula slightly. The cupula then displaces the cilia, causing a decrease in the firing rate of the associated vestibular nerve if the cilia bend away from their kinocilium and an increase in vestibular nerve firing if the cilia bend toward their kinocilium. There are 6 total semicircular canals (three in each inner ear): paired lateral, superior and posterior. The lateral semicircular canals have a plane that is elevated 30 degrees from the coronal. The posterior and superior semicircular canals are in planes that are approximately 90 degrees from each other and both are laterally askew from the sagittal plane. The superior (a.k.a. anterior) canal on one side is on the same plane as the posterior canal on the other side, and so detects angular acceleration in the same plane. The ampulla and kinocilia in the *crista ampullaris* of the lateral semicircular canal are arranged in such a way that ampullopetal flow of endolymph causes increased firing of the vestibular nerve and ampulofugal flow decreases the firing of the vestibular nerve. The arrangement is opposite to this in the posterior and superior canal, with ampullopetal flow leading to inhibition of the associated vestibular nerve branch and ampulofugal flow leading to excitation. The end result is the same though: when head is turned towards the side of the semicircular canal (in its plane), that side's vestibular nerve is excited and the opposite paired side's nerve is inhibited. The range of response in excitation of a nerve is greater than the range of response in inhibition. Therefore, both vestibular nerves are generally required to sense acceleration without any detectable deficit.

The otolithic organs consist of a utricle, which is oriented the axial plane, and a saccule, which is oriented in the sagittal plane. These structures contain ciliated cells underneath a gelatinous layer and are bathed in endolymph. They also contain otoconia, which are calcium carbonate crystals of a higher specific gravity than endolymph. The otoconia are displaced in response to changes in head position with relation to the vertical. The otolithic organs also respond to linear acceleration. The ciliated cells can inhibit or excite the vestibular nerve, depending on the direction of their bend in relation to the kinocilium (away=inhibition, towards=excitation).

The vestibular system can affect posture via vestibulospinal pathways. These pathways, in conjunction with visual-postural and proprioceptive-postural pathways, serve to maintain the patient's center of gravity over the base of support. For instance a quick head tilt to the right causes extension of right sided leg extenders to counteract a change in the perceived center of gravity. A perceived forward motion causes a sway forward to maintain the support base.

The vestibulo-ocular reflex is a system that maintains the stability of the visual field in response to acceleration of the head in a particular direction. The pathway is from vestibule to vestibular nuclei to the ocular motor nuclei, with modulation from cerebellar centers. The reflex results in movement of the eye so that the fovea can focus the same image during movement of the head. Thus the eye rotates (including torsional rotation) in an exactly opposing fashion to the head. When the eye's rotational limit is exceeded, a saccade brings the eye back to the midline. For example, rotation of the head (nose) to the left would result in excitation of the left branch of the vestibular nerve that innervates the left semicircular canal and in inhibition of the right semicircular canal branch. This combination of excitation and inhibition is passed through the reflex arc and is translated into excitation of the ocular muscles to rotate the eye to the right in an exactly opposing fashion to the head rotation until no longer possible, at which point a saccade brings the eye back to the midline. The process then repeats itself until the angular acceleration ceases. The eye movement in this example produces a physiologic left beating nystagmus. Similarly, excitation of the vestibular nerve branch innervating the left superior semicircular canal would lead to a down and left torsional beating nystagmus. Alternatively, inhibition of the right posterior canal would result in the same thing. Complete ablation of the left vestibular nerve would result in nystagmus beating to the right because of the tonic input from the right lateral canal. Torsional nystagmus beating to the right would also be noted because of the superior and posterior canal on the right. Up-beating or downbeating nystagmus cannot be explained by a lesion in the periphery, and so, is almost always due to central etiologies. The otolithic sensory systems can influence eye tilt, for example, elevating the right eye, and depressing the left when the head is tilted toward the right shoulder.

The vestibular system is a very important system in the conscious sensation of acceleration. Peripheral or central damage to the vestibular system would lead to a severe sense of imbalance until compensation occurs. Also, they would result in measurable alterations of the vestibulo-spinal and vestibulo-ocular reflexes until compensation occurs. Compensation in peripheral vestibular injury is via adjustment of the gain of vestibular reflexes in the cerebellum and modification of signal delivery to supratentorial centers.

Types of Balance Function Testing:

The Bedside Evaluation:

Most causes of dysequilibrium can be diagnosed by a complete history and several basic bedside evaluations of the three balance systems. A discussion of the causes of dysequilibrium is beyond the scope of this article. Important questions in the history are directed towards obtaining a concrete description of the patient's symptoms. An important symptom suggesting vestibular pathology is the sensation of vertigo. Vertigo should be defined for the patient as a sensation of motion, or feeling that the world is moving when no motion is actually occurring. The duration of this symptom, aggravating factors (e.g. position), associated symptoms of hearing loss, tinnitus, and affect on daily life should all be probed. When vestibular pathology is suspected, an audiogram is important to check for the presence of concomitant auditory pathology. Syncope and the "light-headedness" that precede syncope are generally not associated with vestibular problems.

The bedside examination of balance function includes a thorough neuro-otologic examination. The exam can help to distinguish between peripheral and central causes of vertigo and can help to determine the cause of peripheral lesions. General physical examination should include a vascular exam. Blood pressures from both arms can be checked for the presence of subclavian steal syndrome. Supine and standing blood pressure can test for orthostatic hypotension. Auscultation for cervical bruits can identify possible posterior circulation abnormalities (which uncommonly cause vertigo during TIA's of the posterior circulation).

Proprioceptive and vestibulospinal function can be tested with a group of gross balance tests. The Romberg test involves having the patient stand feet together with the eyes closed to see if the patient can maintain balance. It tests vestibular (primarily otolith organs) and proprioceptive balance pathways. The vestibulospinal pathway can be isolated by having the patient stand on a foam surface to minimize proprioceptive input. The past-pointing test is primarily (though not completely) a test of proprioception, and it involves having the patient repeatedly bring his finger to a remembered position with his eyes closed. Patients with vestibular pathology may point more to the side with the lesion. The Fukuda stepping test involves having the patient step in place with the eyes closed about 20 times. The patient may turn towards the side of the lesion. It is important to note though that right handed people often drift to the left somewhat with this test.

Motor and sensory examination further tests proprioception and cerebellar function. Cerebellar function is tested with rapid alternating movements and finger-to-nose tests. Proprioception can be tested directly by flexing or extending body parts (e.g. toes) and asking patients to describe their positions. Proprioception is also tested by testing deep tendon reflexes.

Vision should be tested by testing visual acuity. Acute change in visual acuity in one eye may lead to loss of depth perception and to a sense of disequilibrium. Ocular nerve palsy can also affect binocular vision. Saccades and smooth pursuit should be evaluated. The optokinetic reflex can be tested by rotating a drum with vertical stripes that covers 80% of the patient's visual field and observing the nystagmus pattern.

Static vestibular balance can be evaluated by an assessment of nystagmus. Nystagmus is

a pattern of back-and-forth eye movement with a slow and fast phase. The directionality of nystagmus refers to its fast phase. Spontaneous nystagmus is the hallmark of static vestibular imbalance. Characteristics that distinguish central from peripheral imbalance include the effect of visual fixation (which suppresses nystagmus from peripheral, but not central lesions), the wave-form of the slow phase (peripheral lesions are steady and beat in one direction, while central lesions may not), and the axis of eye rotation (which usually involves directional and torsional components in peripheral nystagmus). Visual fixation induced suppression of nystagmus may make detection of nystagmus difficult. The Frenzel lenses remove this obstacle. Peripheral nystagmus is a jerk nystagmus with unidirectional constant-velocity slow phases; but velocity of the slow phase increases when looking the direction of the quick phase. Nystagmus of a central etiology may have a decaying-velocity slow phase or a pendular nystagmus. Otolith imbalance may lead to a skew deviation (vertical misalignment of the eyes) and a head tilt toward the side of the ablative lesion. Skew deviation may also be seen with lesions of the medial longitudinal fasciculus (MLF) or a trochlear nerve paresis.

Dynamic vestibular function can be tested in several ways. The patient's head can be turned to one side, then the other while asking the patient to fixate on a stationary object. This tests the gain of vestibulo-ocular reflex. Patients with an ablative lesion on one side exhibit saccadic eye movements during a turn to that side to keep focused on the target because the gain of the vestibulo-ocular reflex for that motion is less than 1. This is a result of decreased stimulus from that side. Head shaking nystagmus occurs after shaking a the patient's head with a frequency of 2 Hz for at least 10 seconds, with a brief post-shaking nystagmus beating away from the ablative lesion.

Provocative measures can be used at the bedside to evoke nystagmus in patients. Hyperventilation may induce nystagmus in patients by increasing the excitability of depressed vestibular nerves (e.g. those compressed by a vestibular schwannoma), thus negating the central compensation that has occurred over time. Positioning is useful in the diagnosis of benign paroxysmal positional vertigo, a disorder involving posterior canal irritation secondary to otoliths slipping into the posterior canal (canalolithiasis). The Dix-Hallpike maneuver involves moving a patient from a seated position with the head turned 45 degrees to one side to the head-hanging supine position. This is repeated for the other side. The maneuver moves the otoconial debris into an irritating position, causing, after a latency of 1 to 10 seconds, an upbeat and torsional nystagmus with the fast phase of the torsional component towards the affected ear in reference to the upper pole of the eye. The nystagmus fatigues after 20 to 45 seconds. The valsalva maneuver, either with a closed glottis, or an open glottis and closed nose and mouth increases intracranial pressure, or middle ear pressure, respectively. This may cause vertigo in patients with superior canal dehiscence, perilymph fistulas, or in people with cranio-cervical abnormalities (Arnold-Chiari malformation).

Laboratory Tests of Balance Function:

There is wide range of laboratory tests to evaluate the balance system. Each test has its own advantages and drawbacks and must be considered in light of the patient's history and physical exam findings. Laboratory balance function tests cannot provide a diagnosis, but can give insight into the pathophysiology of the patient suffering from a balance disorder. Vestibular function tests can provide important information confirming the side involved in a peripheral

vestibular lesion. Balance function tests can provide a quantitative measure of the extent of a vestibular lesion, which is particularly useful in monitoring progression of a lesion (e.g. in ototoxicity). Balance function tests of posture can provide a measure of the patient's ability to integrate sensations from several different modalities. Vestibular function tests may also help to decide which patients would benefit most from vestibular rehabilitation.

Electronystagmography (ENG), and Infrared Cameras:

The ENG is the centerpiece of most formalized balance function testing. The ENG detects quantitative changes in eye position by measuring the change in natural charge difference between the retina (-) and cornea (+) that occurs with eye movement. Electrodes are placed to measure vertical and horizontal movements. The advantages of this recording methodology include the low expense, minimal patient discomfort, and audiologist experience with the technique. The disadvantages include the susceptibility of the signal to changes in skin resistance, eye blink artifacts, and poor signal-to-noise ratio. Newer technologies exist for recording eye movement as well. Infrared imaging systems can be used to quantitatively monitor and record eye movements via the use of goggles that emit infrared light and contain infrared sensitive cameras. Such systems allow visualization of the patient's eyes on a TV monitor and allow recording of torsional eye movements, which the ENG cannot. Visual stimuli can be presented directly via the goggles. In addition to recording of torsional nystagmus, other advantages of the infrared systems include elimination of artifact, elimination of the need for frequent recalibration, and more easy identification of disconjugate eye movements. The disadvantages of these systems include the need for bulky goggles and the expense of the system. Both ENG and infrared detection systems measure eye movements to assess the saccade system, the smooth pursuit system, the optokinetic reflex, and the vestibulo-ocular reflex.

Saccades are rapid eye movements designed to bring a peripheral visual target onto the fovea. Most systems use a computer generated model for the presentation of visual targets to the patient and then characterize saccades in terms of velocity, accuracy, and latency. Abnormalities in the saccade system often result from central pathology. Undershooting or overshooting saccades implicate pathology in the cerebellum. Slow saccades can result from a variety of central lesions (especially the pontine reticular formation) and from the use of certain medications. Long latencies in the initiation of saccades may result from neurodegenerative disorders or from lesions of the brainstem and cerebellum .

Smooth pursuit testing involves tracking a visual target as it moves back and forth in the visual field. Inability to smoothly pursue a target results in frequent corrective saccades, and can result from a broad range of CNS pathology, especially in the cerebellum or brainstem.

Optokinetic nystagmus testing involves the stimulation of the almost the entire retina (and not just the fovea). Rotating stripes that fill 80% of the visual field are frequently used. The eyes follow a stripe and then make a quick saccade to catch the next stripe, resulting in the pattern of optokinetic nystagmus. Lesions responsible for abnormalities in the slow phase of the nystagmus are similar to those responsible for smooth pursuit system defects.

The vestibulo-ocular reflex is the pathway assessed by the rest of the ENG or infrared system. Spontaneous nystagmus is measured by removing visual fixation (dark visual field).

Spontaneous nystagmus is considered abnormal if its peak slow phase exceeds 5 degrees per second. Gaze-evoked nystagmus is tested by having the patient focus on targets 30-40 degrees to the right, left, above, and below the center. Gaze evoked nystagmus that is of peripheral vestibular origin is typically unidirectional and beating toward the side of greater neural activity, has both torsional and horizontal components, and its amplitude increases when gaze is directed toward the direction of nystagmus. Gaze-evoked nystagmus of central etiology may be purely vertical or torsional, it does not suppress with fixation, and it may change direction with gaze. End-point, or eccentric nystagmus may be physiologic when it is not sustained. Symmetric gaze-evoked nystagmus may result from drugs such as phenobarbital, alcohol, diazepam, or phenytoin.

Position testing can be static or paroxysmal. Static testing involves placing the patient in various positions relative to gravity with removal of visual fixation and observing the vestibular response. The significance of abnormalities in this modality are debatable and must be correlated with other findings in the ENG. Paroxysmal testing is embodied by the Dix Hallpike maneuver. Occasionally, it is necessary to perform formal testing to obtain a quantitative measure of the severity of disease in BPPV (for example, if surgery is to be performed, although it is now rare to perform surgical ablation for BPPV).

Bithermal Caloric tests are used to evaluate the lateral semicircular canal. The patient is placed at an angle 30 degrees to the horizontal to place the lateral canal in a vertical position. The external auditory canals are irrigated with water 7 degrees Celsius above and below the body temperature. Convective effects on the endolymph of the lateral canal produce very slow fluid flow (.002 to .004 Hz). The induced nystagmus for the cold stimulus beats to the opposite side and that for the warm stimulus beats to the same side. A directional preponderance can be calculated with ENG, with a difference of greater than 30% between ears being considered abnormal. Unilateral weakness, or bilateral weakness with history of labyrinthine disease or after use of ototoxic drugs is indicative of peripheral lesions. Bilateral reduced or absent responses without history of labyrinthine disease may be suggestive of central disease.

Testing for the presence of a labyrinthine fistula or superior canal dehiscence can be accomplished with the use of a tympanometer and an ENG setup. In this test, the patient is first placed in an upright position and visual fixation is removed. Next, a probe from an immittance bridge is placed in the ear canal and a seal is obtained. Pressure is then varied from 0-200 mm H₂O and held for approximately 15 seconds. Pressure is then decreased to - 400 mm H₂O and held for 15 seconds. Patient is questioned for subjective symptoms. The presence of nystagmus or subjective symptoms is suggestive of a fistula.

The rotatory chair apparatus uses a rotating chair to test the semicircular canals at a higher (and more physiologic) frequency than with caloric testing. The test is an integration of responses from both vestibuli, unlike caloric testing. The patient is rotated in a chair at a velocity of from .01 to 1.28 Hz. The slow component of the physiologically induced nystagmus is analyzed in terms of phase, gain, and symmetry of eye movement. Symmetry is measured by comparing the peak slow-wave velocities between left and right rotations of the patient. In uncomplicated acute ablative vestibular lesions, the symmetry measure shows weakness on the affected side, though confounding factors such as compensation, labyrinthine irritation, and cerebellar lesions may render the symmetry test unreliable. Rotatory chair testing is generally

more palatable to patients than caloric testing (especially pediatric patients). It is useful in monitoring changes in vestibular function over time, in monitoring compensation after acute injury, and in monitoring residual labyrinthine function in patients with no response during caloric testing.

Dynamic Posturography:

Dynamic posturography quantitatively measures the patient's ability to maintain an upright posture in the face of varying somatosensory, visual, and vestibular inputs. Six conditions are tested. In condition 1, the patient maintains a stable position with eyes open. In condition 2, a stable position with eyes closed is maintained. In condition 3, the visual surround is shifted so as to give visual perception of motion. In condition 4, the floor is tilted in relation to the visual surround to provide vestibular, proprioceptive, and visual sensation of motion. In condition 5, the floor is tilted with the patient's eyes closed, thus providing only vestibular and proprioceptive sensation of motion. In condition six, the visual surround is moved equivalently with the floor, again depriving visual sensation of motion. The patient's sway from upright in response to these situations is graded on the scale of 0 (fall) to 100 (no sway). Force plates on which he or she stands detect the patient's sway. Posturography is useful in determining the patient's particular strategy in balancing and to see whether he or she prefers one or more of the balance modalities. It is also very useful in following patients in vestibular rehabilitation programs. Movement coordination tests involve a disruptive movement of the patient to see if he or she can regain balance. Like posturography, this is a multi-modality test.

The following chart adapted from Bailey's Textbook of Otolaryngology is an excellent review of how patients with particular disorders perform on formal balance function testing:

Disorder:	Electronystagmography	Rotational Tests:	Posturography
Meniere's Disease	1. Early: normal or reduced caloric responses Nystagmus may be in either direction 2. Late reduced or absent caloric responses	Normal or decreased gain, or increased phase, or both	1. Typically normal, may be profoundly abnormal in bilateral disease 2. Movement coordination test results are usually normal
BPPV	Positive Dix-Hallpike	Normal	Normal
Cerebellar Disease	1. Normal caloric responses 2. Abnormal fixation suppression, saccades, or pursuit.	1. Symmetric responses with increased gain and abnormal phase lead 2. Abnormal fixation suppression	1. Abnormal postural latency 2. Excessive sway
Bilateral Ototoxicity:	1. Absent caloric responses 2. No spontaneous or positional nystagmus 3. Normal results of oculomotor testing	Markedly reduced gain	1. Vestibular deficit pattern (falls on conditions 5,6) 2. May be unable to stand for test because of poor balance 3. Normal latency on movement coordination tests.

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