Dizziness, Vertigo, and Imbalance

Last Updated: February 21, 2007

Synonyms and related keywords: Ménière disease, benign paroxysmal positional vertigo, BPPV, labyrinthitis, motion sickness, vestibular neuronitis, brainstem ischemia, immune-mediated inner ear disease, inner-ear disease, brainstem syndrome, migraine headache, vertebrobasilar ischemia, tinnitus, hearing loss, central dizziness, peripheral dizziness, non-vertigo, nystagmus, non vertigo, non-vertigo, nonvertigo, disequilibrium, positional dizziness, motion intolerance, visual motion sensitivity, dizzy

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Dizziness and vertigo are among the most common symptoms causing patients to visit a physician (as common as back pain and headaches). The overall incidence of dizziness, vertigo, and imbalance is 5-10%, and it reaches 40% in patients older than 40 years. The incidence of falling is 25% in subjects older than 65 years. Falling can be a direct consequence of dizziness in this population, and the risk is compounded in

those with other neurologic deficits.

Mild hearing loss is the most common disability in the United States. The incidence of hearing loss is 25% in people younger than 25 years, and it reaches 40% in persons older than 40 years. About 25% of the population report tinnitus. Tinnitus and hearing loss are commonly associated with inner-ear diseases, leading to vertigo and dizziness.

Migraine is more prevalent (10%) than Ménière disease (<1%). About 40% of patients with migraine have vertigo, motion sickness, and mild hearing loss. Therefore, differentiating migraine from primary inner-ear disorders is sometimes difficult.

The role of the primary care physician and the neurologist in treating patients with dizziness or verity has increased over the last decade. This article outlines the clinical approach to the patient with dizziness from a neurologic perspective. Emphasis is on differentiating peripheral from central dizziness and on office management of the most common diseases. In addition, indications for referral to an otolaryngologist and/or neuro-otologist and for specialized vestibular testing are discussed.

For excellent patient education resources, visit eMedicine's Brain and Nervous System Center and Ear, Nose, and Throat Center. Also, see eMedicine's patient education articles Benign Positional Vertigo, Dizziness, Ménière Disease, and Tinnitus.

HISTORY

The patient's history is critical in the evaluation of the patient with dizziness. Ask the patient to describe their symptoms by using words other than "dizzy." The rationale for using other words is that patients may use dizzy nonspecifically to describe vertigo, unsteadiness, generalized weakness, syncope, presyncope, or falling.

A critical distinction is differentiating vertigo from nonvertigo. Vertigo is the true rotational movement of self or the surroundings. Nonvertigo includes light-headedness, unsteadiness, motion intolerance, imbalance, floating, or a tilting sensation. This dichotomy is helpful because true vertigo is often due to inner-ear disease, whereas symptoms of nonvertigo may be due to CNS, cardiovascular, or systemic diseases.

Sudden onset and vivid memory of vertiginous episodes are often due to inner-ear disease, especially if hearing loss, ear pressure, or tinnitus is also present. Gradual and ill-defined symptoms are most common in CNS, cardiac, and systemic diseases. The time course of vertigo is also important. Episodic true vertigo that lasts for seconds and is associated with head or body position changes is probably due to benign paroxysmal positional vertigo (BPPV). Vertigo that lasts for hours or days is probably caused by Ménière disease or vestibular neuronitis. Vertigo of sudden onset that lasts

for minutes can be due to brain or vascular disease, especially if cerebrovascular risk factors are present.

Central vertigo secondary to brainstem or cerebellar ischemia is often associated with other brainstem characteristics, including diplopia, autonomic symptoms, nausea, dysarthria, dysphagia, or focal weakness. Patients with cerebellar disease are frequently unable to ambulate during acute episodes of vertigo. Patients with peripheral vertigo can usually ambulate during episodes and are consciously aware of their environment.

A history of headaches, especially migraine headaches, can be associated with migraine-related dizziness. Previous viral illness, cold sores, or sensory changes in the cervical C2-C3 or trigeminal distributions usually indicate vestibular neuronitis or recurrent episodes of Ménière disease.

Dysdiadochokinesis and gait ataxia during episodes are more likely due to cerebellar diseases, especially in the elderly population. Sensory and motor symptoms and signs are usually associated with CNS diseases. The history should include a review of systems (especially head trauma and/or ear diseases) and screening for anxiety and/or depression. History of prescription medicines, over-the-counter medications, herbal medicines, and recreational drugs (including smoking and alcohol) can help to identify pharmacologically induced syndromes.

The most common causes of peripheral vertigo include BPPV, vestibular neuronitis, Ménière disease, and immune-mediated inner-ear disease. The most common cause of central dizziness is migraine, frequently referred to as vestibular migraine or migraine-associated dizziness. Other central causes include demyelination, acoustic tumors, or cerebellar lesions.

Physical examination

In patients with dizziness, general examination should emphasize vital signs, supine and standing blood-pressure measurement, and evaluation of the cardiovascular and neurologic systems. Examine the ears for visible external- and/or middle-ear infection and/or inflammation. Test hearing by using a tuning fork or by whispering. Examine the neck for range of motion. However, specific examination of the vestibular system, beyond the ears, nose, throat and neurologic examination, is fundamental to the evaluation of the patient with dizziness.

Differentiating peripheral and central nystagmus

Examine eye movements for spontaneous nystagmus, gaze-evoked nystagmus, and ocular motor abnormalities. Differentiating peripheral and central nystagmus is a key step. Central nystagmus is a purely horizontal or vertical gaze and not suppressed by visual fixation. Peripheral nystagmus is usually rotatory and most evident with removing visual fixation (eg, by using Frenzel goggles or infrared video nystagmography). It also obeys the Alexander law; that is, the intensity of nystagmus increases with gaze in the direction of the fast phase.

A robust oculocephalic reflex and intact visual acuity with active head movements (dynamic visual acuity) reflect good vestibular function. Absence of the oculocephalic reflex or a decrease in visual acuity with head movements reflect decreased vestibular function. Nystagmus after rapid head shaking reflects asymmetric vestibular input. Evaluating for failure of fixation suppression (FFS) is an important test of the cerebellar modulation of vestibular reflexes. FFS is evident if nystagmus is observed during en bloc head and trunk rotation while the patient fixates on outstretched arms with his or her hands clasped together.

Positioning tests

The positioning test (Dix-Hallpike test) is an important component of the vestibular examination to identify BPPV commonly caused by otolith debris (canalith) floating in the semicircular canals (canalithiasis) or adhering to the cupula (cupulolithiasis). The Dix-Hallpike maneuver is performed by guiding the patient rapidly from a sitting position with the head turned 45° to 1 side to a lying position**. For torsional nystagmus, observation or video recording is more sensitive than electronystagmography (ENG). BPPV is due to posterior semicircular canal canalithiasis approximately 90% of the time.**

Typical nystagmus related to posterior semicircular canal benign positioning and its symptoms are delayed by several seconds (latency). They peak in 20-30 seconds and then decay (paroxysmal), with complete resolution of symptoms while the patient maintains the same head position (habituation).

The vertical component of benign positioning nystagmus is best observed by asking the patient to move the eyes away from the downmost ear to detect if the vertical component of nystagmus is due to downmost posterior canal or uppermost anterior canal (rare).

Symptoms and reversed nystagmus may occur when the patient is brought back to a sitting position. Therefore, benign positioning nystagmus is latent, paroxysmal, geotropic, reversible, and fatigable. Nystagmus of the less common horizontal semicircular canal canalithiasis form of BPPV is purely horizontal, geotropic (beating toward the down ear), and asymmetric. The direction reverses with the change in head

position from 1 side to the other in the supine position. The intensity of nystagmus is strongest when the head is rotated to the involved side.

Anterior-canal BPPV nystagmus, which is rare, is rotary, with its vertical component beating downward. BPPV due to cupulolithiasis (otoconia adherent to the cupula) is relatively uncommon and has different features of nystagmus. With posterior-canal cupulolithiasis, nystagmus is usually geotropic, nonlatent, intense, long lasting, and nonfatigable. With horizontal-canal cupulolithiasis, nystagmus is ageotropic (beating away from the down ear) and intense.

Characterization of nystagmus

Nystagmus, whether spontaneous, gaze induced, or positional, must be completely characterized to be correctly interpreted. This characterization should include provocative factors, latency, directions, effects of gaze, temporal profiles, habituation, fatigability, suppression by visual fixation, and accompanying sensation of dizziness. Failure to fully characterize nystagmus can lead to misdiagnosis.

Caloric testing

Caloric testing can be done as part of the bedside examination. After checking both ear canals for tympanic perforation and wax, instill 1 ml of water at 30°C. Observe the nystagmus response by using Frenzel goggles or an infrared video system. In this way, dizziness, duration and intensity of the nystagmus, and visual fixation suppression can be evaluated.

Test of vestibulospinal reflexes

Vestibulospinal reflexes (VSRs) can be evaluated with tandem gait, Romberg, and Fukuda stepping tests. These tests provide information about the patient's postural stability when his or her visual and proprioceptive inputs are removed. The experienced physician can observe the patient's postural stability, limits of stability, and strategy of movement at the limits of stability. Clinical testing of postural stability is qualitative and requires both experience on the part of the examiner and cooperation by the patient.

Hamid vestibular stress test

The Hamid vestibular stress test is composed of a sensory and a motor component and is performed using a high-compliance foam pad (HCFP). The examination is simple, easy to administer, and applicable to most patients with dizziness and disequilibrium.

In the sensory component, the patient stands on the HCFP with his or her eyes open and the arms stretched out while the examiner observes the degree of sway. The patient then tilts his or her head backward and moves it right and left with the eyes

open and then with the eyes closed. The examiner must be prepared to catch patients if they fall. Experience with this examination has shown that patients cannot stand on the HCFP with eyes closed and head tilted backward unless they have an intact vestibular and balance system.

The motor component is more challenging than the sensory component and is referred to as the body-impulse test. The examiner places his or her hands on the upper part of the patient's chest, and patient is asked to push forward against examiner's hands for a count of 10. The examiner then releases his or her hands, watches the patient's response, and catches the patient if necessary. Most patients can correct for the sudden perturbation by performing 3 corrective responses: forward bending (hip-sway strategy), stepping forward, and stepping back to their original position. This response pattern is repeatable and physiologic. It demonstrates the physiologic postural reaction and the switch between ankle and hip-sway strategies expected at the limits of stability.

Patients with peripheral and central dysfunction have patterns that do not include quick and corrective movements, performing a hip-sway, or taking a step. Of course, these tests are qualitative and subject to the examiner's experience and the patient's musculoskeletal condition and ability to cooperate.

Hyperventilation test

If the results of vestibular examination normal, hyperventilation for 2 minutes is helpful in identifying patients with hyperventilation syndrome. This should be done in the sitting position. Hyperventilation must be done while the examiner monitors for nystagmus by using Frenzel goggles or an infrared video system. Hyperventilation can accentuate both central and peripheral vestibular dysfunction and reproduce dizziness and neurologic symptoms due to hyperventilation syndrome.

Differential diagnosis

On the basis of the patient's history and physical findings, the examining physician should be able to formulate a differential diagnosis and if the symptoms are probably peripheral or central. The table below the most common physical findings in patients with peripheral or central vestibular disorders.

Features Differentiating Peripheral from Central Nystagmus

System or Reflex	Peripheral Lesions	Central Lesions
Oculomotor	Spontaneous nystagmus with eyes closed	Saccades (velocity, accuracy), internuclear ophthalmoplegia, saccadic pursuit, gaze-evoked nystagmus
Vestibulo-ocular reflex (VOR)	Nystagmus without fixation, nystagmus after head shaking, eye-head mismatch, bilateral vestibular loss	Hyperactive VOR, FFS, positional nystagmus, bilateral vestibular loss
VSR	Cautious gait; normal spontaneous movement; normal, spontaneous, and correct movement	Wide-based gait, minimal spontaneous movement

Evaluation of the patient with dizziness begins with careful history taking and complete physical examination, including vestibular examination. In the course of evaluating patients with vestibular and balance disorders, additional tests that are commonly considered include audiometry, vestibular tests, blood tests, CT, and MRI. These tests, especially vestibular tests, must be tailored according to the history and physical findings.

The yield of MRI in patients younger than 50 years is low (<1%). The incidence of an acoustic tumor or other brainstem and posterior-fossa lesions also are low. Clinical judgment, careful neurotologic examination, and audio and vestibular studies are often helpful in obviating MRI.

Of importance, results of these tests are not diagnostic in the medical sense. For example, unilateral vestibular loss can be due to vestibular neuronitis or an acoustic tumor. Therefore, clinicians must avoid the temptation to interpret the results as

indicating pathologic entities. Physicians who are responsible for the medical interpretations of these results should also have the proper training and background in neurophysiology and electrophysiology to be able to use these results effectively. They also must be aware of the limitations and variability inherent in such tests.

The most common vestibular tests are ENG, rotating chair test or sinusoidal harmonic acceleration (SHA), and computerized dynamic posturography (CDP).

ENG testing

The standard ENG test battery is composed of saccadic, gaze, pursuit, optokinetic-eye movement, head-shake nystagmus, positional nystagmus, positioning nystagmus, and bithermal caloric tests.

Saccadic test

The saccadic test is used to evaluate voluntary fast-eye movements. The neural substrate of the saccadic system includes the frontal eye fields, brainstem reticular formation, oculomotor nuclei, and cerebellum. The test should be performed by recording each eye separately, especially if dysconjugate eye movements are suspected. A single-channel saccadic test does not provide meaningful clinical information and should be used only as a calibration signal for horizontal eye movements. Common saccadic abnormalities include dysmetria, slow saccadic velocity, and dysconjugate saccades.

Gaze test

The gaze test is used to evaluate the ability to generate and hold a steady gaze without drift or gaze-evoked nystagmus. The neural substrates of the gaze system are similar to those of the saccadic system. A direct current ENG recording is used to distinguish electronic from pathologic drift. The most common abnormalities detected by the gaze test are gaze-evoked nystagmus and rebound nystagmus due to cerebellar disease.

Pursuit eye-movement test

Pursuit eye movements prevent slipping of an image on the retina while the patient is tracking moving objects. The neural substrate of the pursuit system includes parietal cortex, brainstem reticular formation, cerebellum, vestibular nuclei, and oculomotor nuclei. Pursuit abnormalities occur with brainstem and cerebellar lesions.

Test for optokinetic nystagmus

Optokinetic nystagmus (OKN) is a complex CNS reflex initiated by moving images on the retina. OKN supplements pursuit and vestibular eye movements to stabilize retinal images during constant-velocity head motion. The cortical origin is the parietal lobes. Vestibular nuclei, accessory optic tract, inferior olivary nucleus, cerebellum, and

oculomotor nuclei participate. OKN abnormalities are seen in deep parietal-lobe lesions. OKN testing can also be used to identify subtle ocular motor abnormalities, such as incomplete internuclear ophthalmoplegia.

Test for head-shake nystagmus

Head movements produce vestibular responses with an extremely short latency (<15 ms). Oculomotor responses are slower than this, with latencies approaching 100-200 msec. The compensation for this temporal discrepancy is the ability of the central vestibular system to maintain a memory of head motion, so that eye movements can be accurately matched to head movement.

This capability is referred to as velocity storage, which is usually impaired with unilateral vestibular deficit and uncovered by the head-shake test. The test if performed by 20 cycles of low amplitude, high-velocity active or passive head movements followed by observation for nystagmus. This is done in both the horizontal and vertical direction. Observation must be done with suppression of visual fixation, with Frenzel goggles or an infrared video system. Head-shake nystagmus is seen with uncompensated, unilateral vestibular hypofunction of any cause.

Positional test

Positional testing is performed by recording eye movements without visual fixation in 3 cardinal positions: supine, head right, and head left. Direction-fixed or changing positional nystagmus is usually peripheral and an objective sign of vestibular asymmetry, even if it is present in only 1 head position.

Dix-Hallpike positioning test

Positioning nystagmus is a classic finding in patients with BPPV. It is elicited by positioning the patient rapidly from sitting to the head right, left, and center supine positions; by recording the induced nystagmus; and by noting the patient's symptoms. Hyperextension of the neck is not necessary and should be avoided. Two ENG channels are required to determine the direction of the torsional component of the nystagmus. ENG is less sensitive than clinical observation of benign positioning nystagmus because ENG is insensitive to record torsional BPPV components. In the authors' opinion, ENG should not be used to evaluate patients for BPPV nystagmus.

Bithermal caloric test

Barany introduced the caloric test in 1903. Since then, it has been the time-honored vestibular test in clinical neurotology. The caloric test remains the standard for evaluating unilateral vestibular deficit. However, it is a limited and nonphysiologic test of the vestibular system. Literature about the caloric test is extensive; therefore, only a brief description of the test and its interpretation are provided here.

The traditional caloric test is performed with the patient lying with the head elevated 30°. Cold (30°C) and warm (44°C) water are used to irrigate each ear, 1 at a time. Cold irrigation is an inhibitory stimulus, and warm irrigation is excitatory. The direction of postcaloric nystagmus is determined by the quick phase direction and is easily remembered by using the mnemonic COWS: cold opposite and warm same, (ie, quick phase away from or toward the irrigated ear).

The 3 most important findings from the caloric test are unilateral weakness, bilateral weakness, and FFS of caloric-induced nystagmus. The first 2 abnormalities are due to peripheral vestibular disease, and the third is due to central cerebellar disease.

Rotating chair test, or SHA

Barany introduced rotational testing in 1907. In clinical practice, the rotation test lagged behind the caloric test. However, with the advancement of computer technology, rotational chair-test systems were developed in the late 1970s and continue to evolve. They are now used in several vestibular testing laboratories.

The test is used to evaluate the integrity of the VOR in the low- (0.1-0.32 Hz) or high-frequency (1-4 Hz) ranges. The measured parameters are VOR gain, phase (latency), and symmetry. The test is most useful in determining residual vestibular function and the degree of central vestibular compensation.

An alternative to the rotating chair test is the active head-rotation test, which is used to evaluate VOR gain in the high-frequency range. This test is substantially less expensive and more practical than the chair test. Active head rotation involves recording head and eye position while the patient actively turns his or her head from side to side at progressively faster frequencies.

CDP test

Dynamic posturography has become an integral part of vestibular testing in many vestibular test centers. The clinical application of posturography in neurotology was introduced in the 1970s. CDP system consists of a computer-controlled platform and visual booth used to evaluate both sensory and motor components of balance. The sensory test is most clinically useful, especially in peripheral lesions, vestibular rehabilitation, and medicolegal cases. Posturography is not a substitute for a careful gait examination and probably is of more value in rehabilitation than in diagnosis.

Clinical yield of vestibular tests

Several observations discussed below are drawn from a database of 10,000 patients who underwent the 3 tests (ENG, SHA, CDP) in 1985-1995 under direct supervision of 1 of the authors.

First, the raw data tracings should be viewed and evaluated, particularly those acquired

by using computerized systems, and clinicians should not relying on computerized analysis generated by the system software, even if the raw data are merely noise.

Second, oculomotor findings are frequently overinterpreted, and unnecessary neurologic investigations and MRI studies result. In the database describe above, the yield for abnormalities of central eye movements, saccadic dysmetria, saccadic pursuit, asymmetric optokinetic response, and gaze-evoked nystagmus was less than 5%. Therefore, ENG readers are advised to cautiously interpret eye movements. Novice ENG users sometimes read oculomotor results as being normal for several years while they store their pattern for more-accurate interpretation as their experience increases.

Third, ENG system prints outs only horizontal and vertical eye movements and is therefore insensitive to record pure torsional eye movements often seen with BPPV. Video-based ENG (VNG) has the advantage of depicting and digitally recording pure torsional nystagmus for storing and reediting of the captured video signals.

Fourth, findings on chair and dynamic posturography are infrequently abnormal, and their routine use is probably not cost-effective.

Finally, most abnormalities on vestibular testing can be gleaned from vestibular examination carefully conducted in the office setting.

Acute dizziness and vertigo is usually managed with vestibular suppressants, antiviral medication, and antiemetic medications. Steroids can be used in selected patients. Vestibular suppressants should be used for a few days at most because they delay the brain's natural compensatory mechanism for peripheral vertigo.

Peripheral dizziness

Vestibular neuronitis

Vestibular neuronitis is a common cause of acute vertigo with an incidence of 170 cases per 100,000 people. It is believed to be of viral etiology. A prodromal upper respiratory tract illness may or may not be present. Vertigo without auditory symptoms develops and last for several days.

Vestibular compensation proceeds in the usual fashion, with the most severe vertigo resolving in 1 week. The predilection for the superior division of the vestibular nerve leaves the function of the posterior canal intact in most cases. This effect predisposes to posterior canal benign paroxysmal positioning vertigo as a sequela.

A brief course of an antiemetic and vestibular suppressants is usually needed in the acute phase. Corticosteroids may improve long-term outcomes. Early vestibular rehabilitation is important. Antiviral medications have not proven helpful, possibly because a large spectrum of viruses can cause vestibular neuronitis. One third of patients have chronic vestibular symptoms.

Benign paroxysmal positioning vertigo

BPPV is a common cause of vertigo. The typical symptom is brief vertigo on changing position. Patients may have a residual sensation of disequilibrium between episodes. One half have a symptomatic etiology, such as vestibular neuronitis, Ménière disease, delayed endolymphatic hydrops, sudden sensorineural hearing-loss syndrome, head trauma, or migraine. The remaining patients are usually idiopathic.

Treatment involves dispersing otolithic debris in the semicircular canals (Brandt-Daroff exercises) or repositioning the particles to the utricle (Epley, Semont, Lempert, and Hamid maneuvers, among others). Medications are not effective in the treatment of BPPV.

BPPV treatment should be administered if the patient has the typical history and benign paroxysmal positioning nystagmus (BPPN) on examination. Treatments based on only the history or those applied to atypical nystagmus are not effective and can lead to unwarranted complications.

The most common complication of the Semont or the Epley maneuver is the conversion of the posterior canal-horizontal canal BBPV, which is treated with the Lempert or Hamid maneuvers. Less common is undue cervical strain, especially with the Semont maneuver or with neck hyperextension during the Epley maneuver.

Ménière disease

Ménière disease entails the triad of episodic vertigo, tinnitus, and hearing loss. Untreated, severe hearing loss and unilateral vestibular paresis are inevitable. Bilateral involvement occurs in one third of patients. The mechanism can be hereditary, autoimmune, infectious, or idiopathic. The common pathophysiology is disordered fluid homeostasis in the inner ear, with endolymphatic hydrops representing a histologic footprint rather than an etiology.

More than 80% of patients respond to conservative therapy with salt restriction and diuretics. Corticosteroids, given orally or intratympanically, can be used to stabilize active disease. Intratympanic gentamicin can be used to reduce vestibular symptoms, but it should be used only in an ear with no serviceable hearing. The role of surgical therapy, such as shunting the endolymphatic sac, is controversial. The literature demonstrates wide variation in the effectiveness, or lack thereof, of surgery.

Autoimmune inner-ear disease

Patients with autoimmune inner-ear disease typically present with rapidly progressive, bilateral hearing loss and vestibular hypofunction. The initial onset may be unilateral. However, the rapid progression and early bilateral involvement distinguishes this disorder from Ménière syndrome. This disease can occur without clinical or laboratory evidence of a systemic inflammatory disorder. Specific laboratory markers for inner-ear antigenicity are of little clinical utility because of their low sensitivity. Corticosteroids are effective. Patients with recurrent symptoms may benefit from methotrexate.

Central dizziness

Migraine

Migraine is a common disorder, affecting 10% of men and 30% of women. About 25% of migraineurs have dizziness. All forms of dizziness can occur with migraine: vertigo, positional dizziness, disequilibrium, motion intolerance, and visual motion sensitivity. Dizziness can occur as an aura or as part of a headache. However, one third of patients consistently have dizziness in the interval between headaches.

The treatment of migraine related dizziness is the same as the treatment of migraine. Headache hygiene and trigger factors should be reviewed. Prophylactic medications are prescribed, if indicated. Abortive medications, such as triptans, are effective for migraine related dizziness, whether accompanied by headache or not.

Cerebrovascular disease

Stroke is the third most common cause of death and the most common cause of disability in adults. The vertebrobasilar circulation supplies the brainstem, cerebellum, and the inner-ear auditory and vestibular structures. Infarction of the cerebellar midline can cause acute vertigo without auditory or other neurologic features (eg, isolated vertigo). This potentially life-threatening occurrence must be differentiated from vestibular neuronitis. About one half of patients have other features of bulbar or long tract involvement, which make the diagnosis of stroke clear.

Evaluation of the patient with stroke is directed at identifying correctable vascular risk factors (hypertension, diabetes, hyperlipidemia, smoking) and at determining the mechanism of stroke (small vessel, large vessel, cardioembolic, dissection, hypercoagulability, vasculitis). Secondary prophylactic therapy and rehabilitation are individualized. Both hearing loss and vertigo can occur in the setting of stroke due to either central and/or peripheral injury.

Multiple sclerosis

Multiple sclerosis is a disorder of recurrent, inflammatory CNS demyelination due to underlying autoimmune disorder. The onset is usually at 20-40 years of age. Episodes begin over hours to a few days and last weeks to months. Typical symptoms include optic neuritis, ocular motor dysfunction, trigeminal neuralgia, sensorimotor deficits,

myelopathy, ataxia, and bladder dysfunction. Vertigo, at times mimicking vestibular neuronitis, is a presenting symptom in less than 10% of patients. Dizziness or vertigo occurs at some point in the course in a third of patients. Few patients present with hearing loss due to brainstem involvement.

The diagnosis of multiple sclerosis requires the presence of dissemination in time and space, ie, different neurologic symptoms at different times. Careful history taking, examination, and serial follow-up combined with MRI and spinal-fluid analysis helps in establishing the diagnosis. The diagnosis should not be based on MRI abnormalities alone. Disease-modifying therapy is available, but it is only modestly effective. The search for improved treatment is ongoing.

Masses and malformations of the posterior fossa

Vestibular schwannoma (acoustic neuroma) is an uncommon lesion with an incidence of 1.1 per 100,000. It typically manifests with slowly progressive, unilateral hearing loss, and tinnitus. Dizziness is not a common symptom, as the vestibular system can compensate for such gradual unilateral hypofunction. Dizziness can occur as the tumor expands in the cerebellopontine angle and effaces the brainstem and cerebellum. Arachnoid cysts can also occur in the posterior fossa and result in subtle and non-specific dizziness and auditory symptoms.

Chiari malformation occurs in a few adults. It is congenital, but often does not become symptomatic until the age of 20-40 years. Occipital headache precipitated by Valsalva maneuvers, coughing, exertion, or changing position is common. Dizziness may occur with the same precipitants.

Once suspected, the diagnosis can be confirmed with MRI. Surgery should be considered for patients with more-than-mild symptoms.

Falls

The most common fall is a simple fall in which the patient trips and has no ominous underlying peripheral or central disorder. Hazards in the environment (eg, rugs, electrical wires, poor lighting), polypharmacy, and orthopedic factors often contribute to fall.

Because of the substantial risk of injury and the resultant decline in independence and/or quality of life after a fall, a well-directed evaluation is indicated. Balance is not a single physiologic function. The sensory inputs are vision, vestibular, and proprioceptive. While a person is walking, the CNS must instantaneously integrate this information and execute appropriate motor plan and output. This function must be supported by an adequate musculoskeletal system. All of these factors change with age. Any further disease related decline in any of these systems further impairs balance. Bilateral vestibular failure is a contributor in one fourth of elderly patients with disequilibrium. Untreated BPPV can be a risk factor for falling.

Orthostatic hypotension due to aging or to medications is also a common contributor. Vestibular rehabilitation is an important consideration is all patients with acute or chronic vestibular dysfunction. Patients with gait and balance problems should undergo physical therapy and a home-safety evaluation.

Drug Category: *Antihistamines* -- These drugs prevent the histamine response in sensory nerve endings and blood vessels and are effective in treating vertigo.

Drug Name	Meclizine (Antivert) -- Decreases excitability of inner-ear labyrinth and blocks conduction in inner-ear vestibular-cerebellar pathways. Effects are associated with therapeutic effects in relief of nausea and vomiting. Most effective if used prn for 2-3 d with episodes of true vertigo.
Adult Dose	25 mg PO q4-6h
Pediatric Dose	<12 years: Not established >12 years: Administer as in adults
Contraindications	Documented hypersensitivity
Interactions	May increase toxicity of CNS depressants, neuroleptics, and anticholinergics
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Caution in angle-closure glaucoma, prostatic hypertrophy, pyloric or duodenal obstruction, and bladder neck obstruction
Drug Name	Dimenhydrinate (Dramamine) -- A 1:1 salt of 8-chlorotheophylline and diphenhydramine believed to be particularly useful in treatment of vertigo. Diminishes vestibular stimulation and depresses labyrinthine function by means of central anticholinergic activity.
Adult Dose	50 mg PO/IM q4-6h or 100-mg supp q8h
Pediatric Dose	2-6 years: Up to 12.5-25 mg PO/IM q6-8h; not to exceed 75 mg/d 6-12 years: 25-50 mg PO q6-8h; not to exceed 150 mg/d >12 years: Administer as in adults
Contraindications	Documented hypersensitivity; administration to neonates (IV products may contain benzyl alcohol, which has been associated with

	fatal gasping syndrome in premature infants and low-birth-weight infants)
Interactions	Alcohol or other CNS depressants may have additive effect; caution with concurrent antibiotics that may cause ototoxicity; may mask ototoxic symptoms caused by certain antibiotics (irreversible damage may result)
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Do not treat severe emesis with antiemetic drugs alone; may contain either sulfites or tartrazine, which may cause allergic-type reactions in susceptible persons; may impede diagnosis of conditions such as brain tumors, intestinal obstruction, and appendicitis; may obscure signs of toxicity from overdosage of other drugs

Drug Category: *Anticholinergics* -- These agents are thought to work centrally by suppressing conduction in the vestibular-cerebellar pathways.

Drug Name	Scopolamine (Isopto) -- Blocks action of acetylcholine at parasympathetic sites in smooth muscle, secretory glands, and CNS. Antagonizes histamine and serotonin action. Transdermal may be most effective agent for motion sickness. Use in treatment of vestibular neuronitis limited by slow onset of action. Severe adverse effects preclude use in elderly. Robinul more effective and has fewer adverse effects, especially in elderly patients.
Adult Dose	0.6 mg PO q4-6h or 0.5 mg TD q3d
<>	
Pediatric Dose	6 mcg/kg/dose IV/IM/SC; not to exceed 0.3 mg/dose; or 0.2 mg/m^2 repeated q6-8h
Contraindications	Documented hypersensitivity; primary glaucoma (including initial stages); pyloric obstruction; toxic megacolon; hepatic disease; paralytic ileus; severe ulcerative colitis; renal disease; obstructive uropathy; myasthenia gravis
Interactions	Antipsychotic effectiveness of phenothiazines

	may be decreased with coadministration; concurrent therapy may increase anticholinergic adverse effects (adjust phenothiazine dosages prn); coadministration with tricyclic antidepressants (TCAs) may increase anticholinergic adverse effects (eg, dry mouth, constipation, urinary retention) due to additive effect (TCAs with decreased anticholinergic activity may be beneficial)
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in elderly patients because of increased incidence of glaucoma; large doses may suppress intestinal motility and precipitate or aggravate toxic megacolon; anticholinergics may aggravate hiatal hernia associated with reflux esophagitis; patients with prostatism can have dysuria and may require catheterization; use cautiously in patients with asthma or allergies; reduction in bronchial secretions can lead to inspissation and formation of bronchial plugs
Drug Name	Glycopyrrolate (Robinul) -- Blocks action of acetylcholine at parasympathetic sites.
Adult Dose	1-2 mg PO bid/tid
Pediatric Dose	40-100 mcg/kg/dose PO tid/qid
Contraindications	Documented hypersensitivity; narrow-angle glaucoma; tachycardia; ulcerative colitis; paralytic ileus; acute hemorrhage
Interactions	Levodopa decreases effects of glycopyrrolate; both amantadine and cyclopropane increase glycopyrrolate toxicity
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	May increase megacolon, hyperthyroidism, congestive heart failure (CHF), coronary artery disease (CAD), hiatal hernia, and benign prostatic hyperplasia (BPH); not recommended for children <12 y or patients with Down syndrome

Drug Category: *Benzodiazepines* -- By binding to specific receptor sites, these

agents appear to potentiate effects of gamma-aminobutyric acid (GABA) and facilitate inhibitory GABA neurotransmission and other inhibitory transmitters. These effects may prevent vertigo and emesis.

Drug Name	Diazepam (Valium) -- Effective in treating vertigo. Depresses all levels of CNS, including limbic and reticular formation, possibly by increasing activity of GABA, major inhibitory neurotransmitter. Individualize dosage and cautiously increase to avoid adverse effects. Effective for acute episodes. Discontinue as quickly as possible to maximize cerebellar vestibular compensation process.
Adult Dose	5-10 mg PO/IV/IM q4-6h
Pediatric Dose	<6 months: Not recommended >6 months: 0.05-0.3 mg/kg/dose IV/IM over 2-3 min, repeat in 2-4 h prn 0.12-0.8 mg/kg/d PO divided q6-8h; not to exceed 10 mg/dose
Contraindications	Documented hypersensitivity
Interactions	Increases toxicity of benzodiazepines in CNS with coadministration of phenothiazines, barbiturates, alcohols, and monoamine oxidase inhibitor (MAOIs)
Pregnancy	D - Unsafe in pregnancy
Precautions	Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity)

Drug Category: *Phenothiazines* -- These drugs are effective in treating emesis, possibly because of their effects in the dopaminergic mesolimbic system.

Drug Name	Promethazine (Phenergan) -- Antidopaminergic effective in treatment of emesis. Blocks postsynaptic mesolimbic dopaminergic receptors in brain and reduces stimuli to brainstem reticular system. Robinul safer and has fewer adverse effects. Well tolerated in elderly patients and does not have potential for extrapyramidal syndrome.

Adult Dose	25 or 50 mg PO/IM/PR q4-6h
Pediatric Dose	<2 years: Contraindicated >2 years: 0.25-1 mg/kg PO/IV/IM/PR 4-6 times/d prn
Contraindications	Documented hypersensitivity; children younger than 2 y (incidences of death due to respiratory depression)
Interactions	May have additive effects when used concurrently with other CNS depressants or anticonvulsants; coadministration with epinephrine may cause hypotension
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Can be associated with CNS depression, dry mouth, extrapyramidal symptoms, hypertension, and skin rash; caution in cardiovascular disease, impaired liver function, seizures, sleep apnea, and asthma
Drug Name	Prochlorperazine (Compazine) -- Antidopaminergic drug that blocks postsynaptic mesolimbic dopamine receptors. Has anticholinergic effect and can depress reticular activating system (possibly responsible for relieving nausea and vomiting).
Adult Dose	5-10 mg PO/IM q6h 25-mg supp PR q12h
Pediatric Dose	2.5 mg PO/PR q8h or 5 mg PO/PR q12h prn; not to exceed 15 mg/d 0.1-0.15 mg/kg/dose IM; change to PO when possible
Contraindications	Documented hypersensitivity; bone marrow suppression; narrow-angle glaucoma; severe liver or cardiac disease
Interactions	Coadministration with other CNS depressants or anticonvulsants may cause additive effects; administration with epinephrine may cause hypotension
Pregnancy	D - Unsafe in pregnancy
Precautions	Drug-induced Parkinson syndrome or pseudoparkinsonism frequent; akathisia most

	common extrapyramidal reaction in elderly; lowers seizure threshold; caution in history of seizures

Drug Category: *Monoaminergics* -- These agents may be used to treat vertigo, possibly by modulating the sympathetic system.

Drug Name	Ephedrine (Pretz-D) -- Stimulates release of epinephrine stores, producing alpha- and beta-adrenergic receptors.
Adult Dose	5-10 mg PO/IM q6h 25-mg supp PR q12h
Pediatric Dose	<2 years: Not recommended 2-5 years: 3 mg PO q6-8h >5 years: 6.25 mg PO q6-8h
Contraindications	Documented hypersensitivity; angle-closure glaucoma; cardiac arrhythmias
Interactions	Theophylline, atropine, or MAOIs may increase toxicity; alpha- and beta-blockers decrease vasopressor effects of ephedrine; cardiac glycosides and general anesthetics increase cardiac stimulation of ephedrine
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Adverse effects (eg, excitation, tremulousness, insomnia, nervousness, palpitation, tachycardia, other symptoms associated with sympathetic activation); bladder sphincter spasm (may cause a transient acute urinary retention); caution in elderly and in patients with diabetes mellitus, hyperthyroidism, hypertension, cardiovascular disease, prostatic hypertrophy, or cerebrovascular insufficiency

Dizziness and related symptoms are among the most common reasons why individuals

seek medical evaluation. Primary care physicians evaluate the vast majority of these symptoms, and most are treated successfully.

The patient's history and findings on vestibular examination are critical in identifying underlying causes. Auditory, vestibular, complementary blood and radiologic tests help in narrowing the differential diagnosis and tailoring treatment. Vestibular tests should be ordered after careful history taking and examination because they do not provide the clinician with diagnostic information.

Most patients are treated medically and with vestibular rehabilitation. In addition to the appropriate medical and rehabilitative managements, safety must be emphasized and discussed with patients and their families. Occupational and physical therapists are helpful in addressing home safety and providing a structured balance-rehabilitation program.

Vestibular research will increase the utility of current tests and further expand the role of vestibular rehabilitation. New knowledge of molecular biology and genetics of vestibular disorders (eg, the genetics of motion sickness) will probably lead to new biotechnology, vestibular pacers, implants, and pharmacologic modulators of the vestibular and balance system.

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