Vertigo and Dizziness

Understanding and Managing Fall Risk

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KEYWORDS

• Vertigo • Dizziness • Fall risk • Geriatric vestibulopathy

KEY POINTS

- Dizziness is associated with functional disability and risk of falls.
- Falls are the leading cause of fatal and nonfatal injuries among older Americans.
- "Dizziness" can describe several sensations, including spinning or nonspinning vertigo, disequilibrium, imbalance, presyncope, lightheadedness, and floating or a combination thereof.
- Chronic dizziness is often multifactorial and can reflect dysfunction in the vestibular, somatosensory, or visual systems or in their central integration. Systemic processes, such as postural hypotension, arrhythmias, heart failure, medication use, and lower extremity weakness or frailty, also contribute.
- Careful history and physical examination are critical in evaluating dizzy patients and in many cases may preclude the need for more expensive imaging or vestibular testing.

INTRODUCTION

Vertigo and dizziness are common among older adults, defined as those over age 65 years. These symptoms are closely associated with fall risk and portend major implications for geriatric injury and disability. Management can be particularly challenging, because symptoms are often nonspecific and may reflect multiple etiologies. This article reviews relevant definitions, epidemiology, pathophysiology of balance, diagnosis, and clinical management.

DEFINITIONS

Dizziness is a general term that can describe several sensations, including spinning or nonspinning vertigo, disequilibrium, presyncope, lightheadedness, floating, or a combination thereof.

- Vertigo refers to the illusory sensation of movement of the body or the environment.

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- Disequilibrium describes a sense of imbalance, unsteadiness, or postural instability.
- Presyncope connotes lightheadedness and the sense of an impending fainting episode. It is often associated with temporary diffuse cerebral hypoperfusion.

Despite current consensus among medical practitioners as to the use of these terms, patients use these words to describe any or all of the sensations described previously and may also include other sensations, such as weakness, fatigue, floating, fear of falling, or unstable gaze. This discrepancy underlies the importance of asking patients to describe their symptoms in their own words.

EPIDEMIOLOGY

Prevalence studies of dizziness among older adults vary depending on the definition of dizziness, study population (eg, age range or community-dwelling adults vs nursing home), and study setting (eg, emergency department vs primary care). Estimates range from 10% to 35%,[1–4] with rates increasing with age such that up to 50% of community-dwelling adults older than 80 are affected.[5]

Falls are highly associated with vertigo and dizziness.[6] Every 11 seconds, an older adult presents to an emergency room for a fall, and every 19 minutes, an older adult dies from a fall.[7] Even in the absence of falls, dizziness is detrimental to quality of life.[8] Adverse effects include anxiety, decline in mobility, fear of falling, limitations of activities of everyday life, and an increase in indirect health care costs.[8]

PATHOPHYSIOLOGY OF DIZZINESS

Balance involves the central integration of multiple sensory systems: vestibular, vision, and somatosensory proprioception and exteroception. It also involves neuromuscular reflex pathways and core and lower body strength to maintain postural stability in response to perceived stimuli. Dizziness can reflect specific medical conditions affecting single systems or may represent multisensory dysfunction from multiple etiologies (**Table 1**).

The peripheral vestibular system provides information about linear and angular acceleration. Three sets of paired semicircular canals sense angular acceleration. The utricle and saccule (otolithic organs) sense horizontal and vertical linear acceleration, respectively. The canals and otolithic organs are innervated by the vestibular nerve, which projects to the brainstem vestibular nuclei and cerebellum. Efferent tracts from the cerebellum then project to motor nuclei of the extraocular muscles and vestibulospinal tracts, which contribute to gaze stabilization and postural control.

Age-related vestibular loss manifests in decreased vestibular hair cells, fewer vestibular nerve fibers, and loss of cerebellar Purkinje cells. Although older adults consistently show decreased vestibular function on quantitative testing, the presence of dizziness is highly variable: those with objective dysfunction may have no subjective symptoms.[9]

The somatosensory system provides information about proprioception (internal sense of body/limb position) and exteroception (sensation of the environment) from mechanoreceptors in the joints and skin. Information must be relayed through peripheral nerves and the posterior spinal column to reach the central nervous system. In an elevator, the vestibular system's saccule detects vertical acceleration, but mechanoreceptors in the feet simultaneously sense a drop in pressure when going down, and an increase when going up. Common disorders affecting the somatosensory system include arthritis, joint replacements, and peripheral neuropathy from diabetes or vitamin deficiency.

Table 1
Causes of and contributors to dizziness in the elderly

Peripheral vestibular	BPPV Vestibular neuritis Labyrinthitis Late-onset Meniere's disease Bilateral deafferentation Perilymphatic fistula Vestibular schwannoma
Central nervous system	Stroke or transient ischemic attack Vertebrobasilar insufficiency Vestibular migraine Neoplastic Neurodegenerative disease (Parkinson's disease, cerebellar ataxia, degenerative dementias) (Normal pressure) hydrocephalus Multiple sclerosis Posttraumatic Neurosyphilis
Somatosensory	Peripheral neuropathy (diabetes, vitamin deficiency) Cervicogenic vertigo Arthritis
Vision	Cataracts Use of bifocals/multifocals
Cardiovascular and orthostatic	Arrhythmia Heart failure Postural hypotension Postprandial hypotension Hypovolemia
Other systemic	Alcohol Heavy metal exposure Hypothyroidism Hypoglycemia, metabolic imbalance Medications, polypharmacy Psychophysiologic

Vision provides information about spatial orientation. Visual acuity, depth perception, accommodation, and contrast sensitivity worsen with age. Furthermore, pathologies, such as cataracts and macular degeneration, become more frequent.

Appropriate central integration of sensory information is integral to balance. The brainstem, cerebellum, and higher cortical structures all undergo age-related changes. Neurodegenerative diseases, such as Parkinson's disease and Alzheimer's disease, become more prevalent with age. Stroke, multiple sclerosis, and neoplasias are also possible.

Although the cardiovascular system is not traditionally believed to be directly involved with balance, disorders, such as arrhythmias and orthostatic hypotension, can lead to cerebral hypoperfusion and presyncopal dizziness. Vertebrobasilar insufficiency reflects ischemia of the brain's posterior circulation. Symptoms include vision changes, vertigo, ataxia, and bilateral numbness or weakness. Insufficiency can occur during episodes of reduced blood pressure or flow (such as after periods of standing, hypovolemia, or external vessel compression from neck turn). Other etiologies include atherosclerosis, embolic events, or arterial dissection.

Psychological disease, such as anxiety and depression in particular, are risk factors. They may reflect a psychogenic etiology for dizziness or a psychological response to somatic disease.[10] Other systemic changes, such as electrolyte imbalance, anemia, or hypothyroidism, can lead to fatigue or confusion, which can exacerbate dizziness.

Just as studies of dizziness prevalence vary depending on definitions, population, and setting, studies of etiologies also vary. The most consistent finding is that a majority of patients have multifactorial disease[1,11] (see **Table 1**). Maarsingh and colleagues[1] found that among patients ages 65 to 95 presenting to primary care, cardiovascular (including cerebrovascular) disease represented 57% of all main causes and peripheral otogenic vestibular disease, 14%. Colledge and colleagues[11] studied community-dwelling adults greater than age 65 years and similarly found 70% of persistent dizziness to be caused by "central vascular disease." In contrast, in a younger population with average age 63, Kroenke and colleagues[12] found the peripheral vestibular system the cause in a majority of patients, and benign paroxysmal positional vertigo (BPPV) represented the most common specific etiology.

ADDITIONAL SPECIFIC ETIOLOGIES OF DIZZINESS

Although multisystem and cardiovascular etiologies are the most common causes of dizziness in the elderly, other specific etiologies that are amenable to intervention, such as BPPV, episodic hypotension, and medication-related dizziness, are reviewed.

Benign Paroxysmal Positional Vertigo

Older adults are at increased risk for BPPV, have a higher rate of recurrence, and may report atypical symptoms. BPPV arises from the abnormal displacement of otoconia into the semicircular canals from the saccule or utricle. In canalithiasis, loose debris produces abnormal movement in the endolymph of the canal. In the much rarer cupulolithiasis, debris attaches to the cupula of one of the canals. The posterior canal is most commonly affected, with the lateral canal second.

Most cases of BPPV can be diagnosed using history and provocative maneuvers. A typical patient describes an episodic rotational vertigo lasting less than a minute, provoked when changing head position relative to gravity, such as turning over in bed.

Physicians should perform the Dix-Hallpike maneuver for further evaluation (discussed later; **Fig. 1**). In cases where history suggests BPPV but the Dix-Hallpike maneuver is negative, the supine roll test can be performed to assess for lateral canal BPPV. At presentation, multiple canals may be involved, and treatment of posterior canal BPPV can lead to otoconia being displaced into the lateral canal. The American Academy of Otolaryngology–Head and Neck Surgery recommends that patients who meet criteria for BPPV without inconsistent signs/symptoms should not obtain routine imaging or vestibular testing.

Episodic Hypotension

Patients with orthostatic hypotension present with dizziness on standing or sitting up. Orthostatic hypotension is defined as a decrease in systolic blood pressure by 20 mm Hg or in diastolic blood pressure by 10 mm Hg within 3 minutes of standing. Hypovolemia, vasodilation, and disorders of the cardiovascular, neurologic, or endocrine systems can be causative.

In the elderly, postprandial hypotension is another consideration. It is characterized by systolic blood pressure decrease by 20 mm Hg or fall below 90 mm Hg within

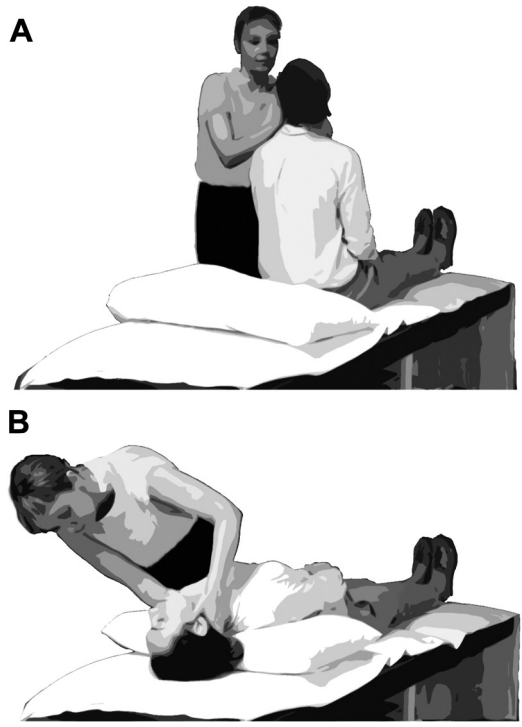


Fig. 1. Dix-Hallpike maneuver. This patient is being assessed for left posterior canal BPPV. (*A*) The examiner stands beside the patient, whose head is turned 45° to the left. (*B*) The patient is guided from seated to supine, with neck extended. Neck extension is achieved by allowing the patient's head to extend beyond the end of the table or by placing a cushion under the shoulders. The patient keeps her eyes open, allowing the physician to examine for nystagmus. After nystagmus resolves, or after 30 seconds, the patient is guided back to a seated position. (*Courtesy of* Jennifer C. Alyono, MD, MS, Stanford, CA.)

2 hours after meals.[13] It is believed to result from a deficient sympathetic response after eating.[13] Treatment includes increasing preprandial water intake, decreasing carbohydrates, and eating more frequent, smaller meals.

Medication-Related Dizziness

Many drugs can cause or exacerbate dizziness (**Box 1**). This is particularly troublesome in the elderly, who have reduced hepatic and renal clearance of drugs, and may have increased sensitivity to drugs. Dizziness can be potentiated through numerous mechanisms: direct ototoxicity, lowering of blood pressure, volume depletion, or direct central nervous system depression and sedation.

DIAGNOSTIC APPROACH TO PATIENTS WITH DIZZINESS

Evaluation of patients with dizziness can be challenging for both patients and physicians alike—symptoms can be difficult to describe and potential diagnoses broad. Although a single cause is not always identifiable, addressing contributors to dysfunction can alleviate, if not resolve, dizziness symptoms in addition to reducing the risk of fall.

Box 1
Medications that may cause dizziness

Ototoxic—peripheral vestibular impairment
- Aminoglycosides
- Loop diuretics
- Platinum-based and vinca alkaloid chemotherapeutics

Cardiovascular—hypotension
- β-blockers and calcium channel blockers
- Vasodilators
- Diuretics
- Angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers

Psychoactive
- Benzodiazepines
- Barbiturates
- Benzodiazepine receptor agonists (eg, zolpidem, zopiclone, and zaleplon)
- Anticonvulsants
- Antidepressants
- Opioids
- Antipsychotics

Antihistamines

Anticholinergics

Muscle relaxants

History

A detailed history is invaluable. Patients should describe their dizziness in their own words. Physicians should inquire about the duration and intensity of symptoms, whether dizziness is constant or episodic, and the length of specific episodes, if applicable. Precipitating factors (such as meals and certain movements) and associated symptoms (such as hearing loss, vision changes, headache, palpitations, chest pain, numbness, and weakness) should be elicited. Recent events, such as trauma and bleeding, may be relevant.

A full medication history should be taken, including over-the-counter drugs, supplements, recreational drugs, and alcohol. A complete medical history should be asked for, with particular attention to disorders that may contribute to fall risk, including cardiovascular disease, arrhythmias, ophthalmologic disease, anemia, arthritis, diabetes, depression, or anxiety.

Physical

Physical examination should be thorough but also focused on the neurologic system. Screening for orthostatic hypotension is recommended as part of routine vital signs. Pulse palpation and heart auscultation can reveal arrhythmias.

Examination of the ears should assess for otitis media or middle ear effusion. Finger rub and tuning fork tests can screen for asymmetric hearing loss.

A complete cranial nerve examination is recommended. On assessing extraocular movements, observe for spontaneous or gaze-evoked nystagmus (**Table 2**). The extremities should be evaluated for sensation and strength. Cogwheeling may suggest Parkinson's disease.

The Romberg test assesses the vestibular and proprioceptive systems. A patient is asked to stand with eyes closed and feet next to each other. The sharpened Romberg is more challenging, with a patient asked to stand with 1 foot in front of the other, in

Table 2
Peripheral versus central oculomotor abnormalities

	Peripheral	Central
Gaze-evoked nystagmus	Unidirectional	Direction may change with gaze position
Spontaneous nystagmus	Horizontal and/or torsional; suppressed by gaze fixation	Often pure vertical, horizontal, or torsional; not suppressed by gaze fixation
Head impulse test	Impaired	Intact
Provocative positional testing (ie Dix-Hallpike maneuver)	Longer latency; fatigability present	Shorter or absent latency; no fatigability present
Smooth pursuit	Smooth	Catch-up saccades

tandem. Regular gait and tandem gait are assessed. A cerebellar examination also includes assessment for dysdiadochokinesia, using rapid alternating hand movements, and for dysmetria, using the finger to nose test.

Provocative Maneuvers

Provocative maneuvers can further assess the vestibular system.

The Dix-Hallpike maneuver is used to evaluate for posterior canal BPPV (see **Fig. 1**). Three key features should be observed to confirm the diagnosis of posterior canal BPPV:

- Nystagmus is torsional and upbeating.
- There is a latency period of approximately 5 seconds to 20 seconds between maneuver completion and onset of subjective vertigo and objective nystagmus.
- The vertigo and nystagmus crescendo and then decrescendo, resolving within 60 seconds.

In the head impulse test (also known as head thrust test) (**Fig. 2**), the examiner sits face-to-face with the patient, with head upright. The patient fixes the gaze on a target (such as the examiner's nose). The examiner rapidly rotates the patient's head left or right or back to midline while observing for corrective saccades. A corrective saccade (where the eyes initially follow the head movement before saccading back to the target) indicates an abnormal test, reflective of vestibular disease. The test is abnormal when the head is thrust toward the side of vestibulopathy. For example, a patient with left vestibular neuritis has corrective saccades when the head is thrust to the left.

In the test of skew (also known as alternate cover test), the examiner covers 1 eye, then the other, switching back and forth. In a healthy patient, the eyes remain motionless. The test is positive, or abnormal, when the eye has a refixation saccade after the cover is moved, indicating ocular misalignment. A positive test can reflect longstanding strabismus; on the other hand, in an acutely vertiginous patient, vertical ocular misalignment strongly suggests posterior fossa stroke.

STUDIES

Laboratory

No consensus exists on standardized laboratory tests. Instead, testing should be tailored to a patient's specific situation. For example, a patient meeting diagnostic criteria for BPPV may need no further testing. In contrast, a patient with acute

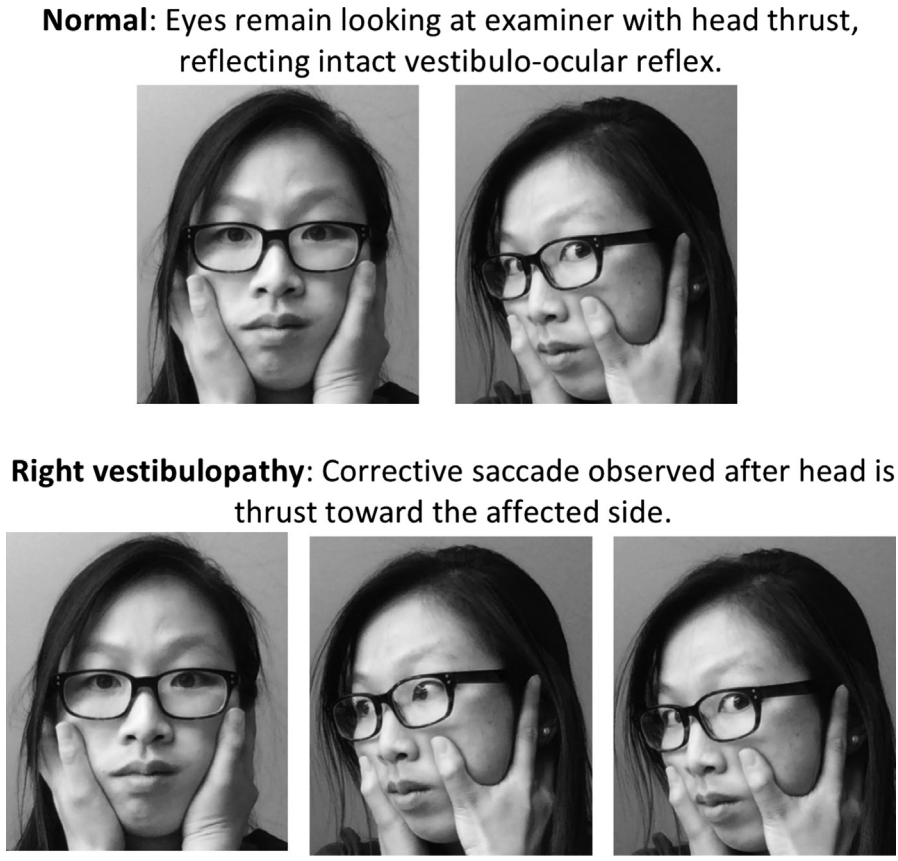


Fig. 2. Head impulse test.

dizziness and chest pain warrants cardiac enzymes. In chronic multifactorial dizziness, hematocrit, basic metabolic panel, hemoglobin A_{1C}, and vitamin B_{12} levels may help rule out systemic contributors.

Audiometry

Many patients with age-related loss of vestibular function also have age-related hearing loss. In corollary, insults to the vestibular system (Meniere's disease and ototoxic medications) are also likely to affect hearing. Screening for hearing loss is recommended, because hearing loss is an additional risk factor for social isolation, depression, and cognitive decline.

Vestibular Testing

Often, careful history and physical examination are sufficient for diagnosis and management of dizziness. In some cases, judicious use of specialized vestibular testing is helpful (**Table 3**). Formal vestibular testing is indicated when the side or site of pathology cannot be localized by history and physical, in documentation of contralateral function when vestibular ablation in considered, and at times, when tracking disease progression or recovery.

Table 3
Vestibular testing

Assessment	Description	System Tested	Clinical Utility
Electronystagmography or videonystagmography			
Eye movements are quantitatively recorded in these subtests:			
Oculomotor	Smooth pursuit, saccadic, and optokinetic tracking	Central and peripheral vestibular	May distinguish central vs peripheral etiology (see Table 2)
Spontaneous and static positional nystagmus	Sitting, supine, body right/left, head turned, etc.		
Caloric testing	Irrigate ear canal with 30°C cool and/or 44°C warm water	Horizontal semicircular canals	Quantifies relative weakness of left vs right.
Dix-Hallpike maneuver	See Fig. 1 for maneuver description	Canalithiasis or cupulolithaisis	Helpful when clinical Dix-Hallpike maneuver is equivocal or atypical
Rotational chair	Compares eye movement responses to head/body rotation.	Vestibulo-ocular reflex	Only test that can detect bilateral vestibular deficits
VEMPs			
Cervical VEMP	Surface electrodes record the sternocleidomastoid (cervical VEMP) or the inferior oblique eye muscles (ocular VEMP) in response to sound stimuli	Saccule, inferior vestbular nerve, vestibulocolic reflex	Diagnostic for superior semicircular canal dehiscence
Ocular VEMP		Utricle, superior vestibular nerve	Unreliable if conductive hearing loss present
Head impulse test with video-oculography	Eye movements are recorded while the head is thrust multidirectionally. Accelerometers record head velocity and direction.	Semicircular canals, vestibulo-ocular reflex	High-frequency, high-acceleration test of the semicircular canals
Computerized dynamic posturography			
Sensory organization test	Ability to maintain balance with eyes open vs closed, with stable vs moving platform, with or without visual surround discordant to sway	Assesses ability to use vestibular vs visual vs proprioceptive systems to maintain balance	Guides and tracks progress to vestibular therapy. Can also use in cases of suspected malingering.

Abbreviation: VEMP, vestibular-evoked myogenic potentials.

Imaging

Many investigators agree that routine neuroimaging is unlikely to reveal the cause of dizziness in most patients.[14,15] Often, imaging findings are present, such as cortical atrophy, but do not alter management, thus requiring nuanced interpretation.[14]

Commonly accepted indications for neuroimaging include focal neurologic signs, suspicion for acute stroke, asymmetric symptoms (such as unilateral hearing loss), known vascular abnormalities, or recent trauma. CT is superior in demonstrating bony abnormalities (eg, superior semicircular canal dehiscence), whereas MRI is superior in demonstrating soft tissue.

ACUTE VESTIBULAR SYNDROME

Special mention is warranted in assessing acute vestibular syndrome (AVS). Acute unilateral injury to the peripheral or central vestibular system produces AVS and is manifested by continuous vertigo, nausea/vomiting, nystagmus, and motion intolerance that evolve over the course of hours.[16,17] Although vestibular neuritis is a common etiology and is self-limited with good long-term prognosis, stroke involving the posterior circulation can present similarly but with potentially more devastating consequences. For example, edema from inferior cerebellar stroke can lead to brainstem compression and death.[16] Misdiagnosis of stroke occurs far more frequently in those presenting with dizziness (35%) compared with those with motor deficits (4%).[18]

Acute vertigo from brainstem stroke is often but not always accompanied by additional symptoms, such as diplopia, dysarthria, other cranial neuropathies, or focal sensory or motor deficits.[16] One study found that up to three-fourths of patients with posterior circulation stroke had isolated AVS with no other symptoms.[17] Dysmetria, pathognomonic for cerebellar dysfunction, may be minimal or even absent after inferior cerebellar stroke.[16] Patients with acute unilateral peripheral vestibulopathy often fall toward the affected side but are typically able to ambulate. In contrast, those with acute cerebellar stroke are usually unable to walk.[16]

The HINTS "plus" battery is a useful three-step bedside examination for patients with AVS. HINTS stands for Head Impulse, Nystagmus, and Test of Skew (see **Table 4**). Compared with early MRI, which can be falsely negative in half of patients up to 48 hours after symptom onset, HINTS "plus" is highly accurate, with a sensitivity of 98%, specificity of 85%, and negative likelihood ratio of 2%.[17,19]

Stroke should be suspected if any one of the following is true: the head impulse test is normal (see **Fig. 2**), nystagmus is direction-changing or vertical (see **Table 3**), or skew deviation (ocular misalignment) is present on the alternate cover test. Conversely, a patient with peripheral vertigo should have an abnormal head impulse test, unidirectional non-vertical nystagmus, and no skew deviation.

HINTS "plus" includes acute hearing loss (in the absence of middle ear disease) as a sign of anterior inferior cerebellar artery infarction. Although including hearing loss in this battery decreases specificity, it increases sensitivity, and therefore decreases the risk of missed stroke. Contrary to conventional teaching, recent studies suggest

Table 4
H.I.N.T.S. examination for acute vestibular syndrome

Examination Steps: H.I.N.T.S.	Findings in Stroke: I.N.F.A.R.C.T.
*H*ead *I*mpulse test (head thrust)	*I*mpulse *N*ormal
*N*ystagmus	*F*ast-phase *A*lternating (direction changes with gaze or vertical)
*T*est of *S*kew (alternate cover test)	*R*efixation on *C*over *T*est (skew deviation present)

Adapted from Newman-Toker DE, Kerber KA, Hsieh YH, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. Acad Emerg Med 2013;20(10):988; with permission.

that the presence of associated acute hearing loss more often reflects vascular infarction rather than viral labyrinthis.[20]

A neurologist should be consulted early should stroke be suspected.

MANAGEMENT OF PATIENTS WITH DIZZINESS

Disease-directed therapy should be initiated in cases with specific diagnoses, such as BPPV. In the absence of focal diagnosis, treatment consists of reducing symptoms, managing risk factors, and reducing fall risk.

Medications

Drugs commonly used as vestibular suppressants include antihistamines (eg, meclizine and diphenhydramine), anticholinergics (eg, scopolamine), antidopaminergics (eg, metoclopramide), benzodiazepines (eg, diazepam and clonazepam), and phenothiazines (eg, promethazine and prochlorperazine, which have antidopaminergic, antihistaminergic, and anticholinergic properties). These medications may be useful in the acute setting for

- Relief of nausea/vomiting in acute vertigo
- Prevention of nausea prior to planned canalith repositioning maneuvers in BPPV
- Prevention of motion sickness

None, however, is appropriate for long-term use in chronic dizziness, and many investigators recommend discontinuation after 3 days.[21] Vestibular suppressants compromise physiologic compensation, prolonging symptoms of dizziness, and may lead to dependency. Side effects are particularly troubling in the elderly and include delirium, cognitive impairment, and urinary retention.

Certain pharmaceuticals are indicated for specific pathologies:

- Vestibular neuronitis/labyrinthitis: steroids have been shown to decrease acute symptom length and improve long-term recovery of vestibular function.
- Vestibular migraine: prophylactic β-blockers, calcium channel blockers, anticonvulsants, and antidepressants
- Meniere's disease: prophylactic diuretics; for exacerbations: intratympanic or systemic steroids, intratympanic gentamycin for ablation

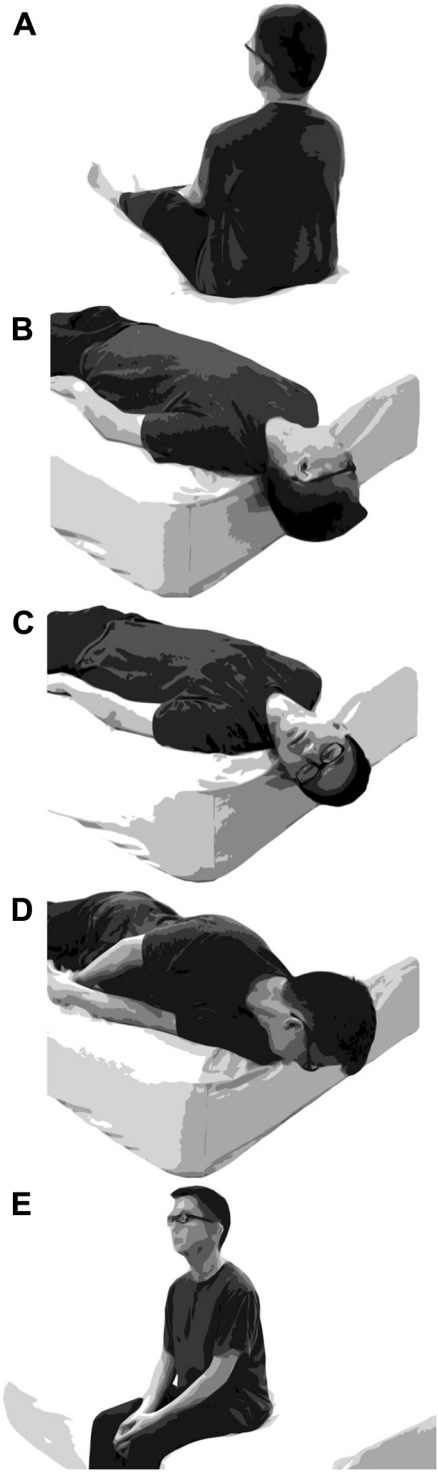
Canalith Repositioning Maneuvers for Benign Paroxysmal Positional Vertigo

The mainstay of BPPV treatment is canalith repositioning maneuvers, such as the Epley or Semont maneuvers. Brandt-Daroff maneuvers are exercises patients perform at home and are intended to catalyze compensation and habituation.

The Epley begins similarly to a Dix-Hallpike test (**Fig. 3**):

- Starting seated with the head turned toward the symptomatic side (for example, the right), the patient is laid back to supine with the head gently extended.
- The patient's head is then rotated 90° toward the opposite, left side.
- Next, the patient continues rotating the head to the left, by looking down toward the ground as he or she rolls onto the left shoulder, into lateral decubitus position.
- In the final step, the patient then is sat upright.
- Each position is maintained for 20 seconds to 30 seconds, allowing vertigo to resolve.

Additional detail regarding the Semont maneuver, treatment of cupulolithais, and disease affecting the lateral canal can be found in the American Academy of Otolaryngology–Head and Neck Surgery clinical practice guidelines.[22]


A
B
C
D
E

Vestibular Therapy

Vestibular rehabilitation not only is useful in reducing falls but also helpful in decreasing dizziness symptoms, regardless of etiology. Vestibular therapy catalyzes compensation in 3 ways[23]:

- Adaptation (central gain readjustment in vestibulospinal or vestibulo-ocular reflexes)
- Substitution (strengthening nonvestibular components of balance)
- Habituation (reducing maladaptive responses and increasing sensory thresholds through repetitive exposure to provocative situations)

Fall Risk Assessment

In addition to treating patients' symptoms of vertigo and dizziness, directed assessments of fall risk should be made. The American Geriatrics Society and British Geriatrics Society published a joint recommendation for annual multifactorial fall risk assessment in all adults over 65 years.[24] Patients are considered high risk if they answer yes to any of the following:

1. Two or more falls in the past 12 months?
2. Presenting with an acute fall?
3. Difficulty with walking or balance?

Given the widespread impact of falls, multiple national and international organizations have outlined algorithms for fall risk assessment and reduction.[7,24] The Centers for Disease Control and Prevention Stopping Elderly Accidents, Deaths, and Injuries (STEADI) toolkit is a good example (https://www.cdc.gov/steadi/).

Patients who screen positive can then be assessed for gait, strength, and balance using standardized tests, such as the Timed Up and Go, 30-Second Chair Stand, or 4-Stage Balance Test. Additional assessment includes history of postural hypotension, medication review, feet and footwear examination, and vision assessment. Dementia is another important consideration, because its presence not only impairs balance but also decreases hazard recognition.

Fall Risk Reduction

Once patients have been identified as high risk for falls, multifactorial intervention should be implemented.

Medication minimization

Polypharmacy, especially psychotropic medication, has consistently been associated with falls. If discontinuation of medications is not possible, dose reduction should be considered.

Exercise programs

In addition to formal vestibular therapy, home-based and community exercise programs[25] (such as tai chi) that emphasize strength and balance are effective.

Fig. 3. Epley maneuver. This patient is being treated for right posterior canal BPPV. (*A*) The patient starts seated, then (*B*) is laid supine with head turned to the symptomatic side (*right*) and neck extended. (*C*) The patient's head is then rotated 90° to the opposite side (*left*). (*D*) The patient continues rotating the head to the left, looking down toward the ground as he rolls onto his left shoulder, into lateral decubitus position. (*E*) In the final step, the patient is sat upright. Each position is maintained for 20 seconds to 30 seconds, allowing vertigo to resolve.

Assistive devices
A physical therapist can determine whether a patient would benefit from a mobility aid, such as a cane or walker.

Vision impairment
Cataract surgery has shown the most effectiveness in reducing falls.[24] Importantly, patients should avoid multifocal or bifocal lenses while walking, especially on stairs. Bifocals distort depth perception, making tripping accidents more likely.

Vitamin D
The American Geriatrics Society instructs that vitamin D should be recommended in all patients with deficiency and considered in those otherwise at high fall risk.[24] Vitamin D supplementation is believed not only to reduce falls by improving neuromuscular function but also to decrease fracture risk in the event of a fall.[26]

Foot and footwear intervention
Bunions or toe deformities may require podiatry consultation. Footwear choices are also important: shoes with low heel height, high contact area with the ground, and good fit are recommended.

Home modification
Patients should evaluate their surroundings for modifiable hazards:

- Removing trip hazards inside the home, like throw rugs and low-lying furniture
- Removing trip hazards outside the home, such as cracked sidewalks and exposed tree roots
- Improving nighttime illumination
- Installing hand-rails in bathrooms and stairs

SUMMARY

Vertigo and dizziness are common among older adults and are associated with heightened fall risk. Multisensory deficits are common, and multifactorial etiologies may contribute. History and physical examination are integral to diagnosis and may preclude the need for imaging or specialized vestibular testing. In AVS, HINTS is a useful screening battery for stroke. In patients with chronic dizziness, formal fall risk assessment should be performed, and multilimbed interventions implemented. Vestibular therapy is effective in reducing both symptom severity and fall risk.

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