

Dizziness in the older adult, part 1

Evaluation and general treatment strategies

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Dizziness is one of the most common complaints among patients age 50 and older who present to primary care physicians. The evaluation of dizziness is challenging due to the wide range of diagnostic possibilities, including four symptom categories each with multiple potential underlying causes. Although each cause has specific treatment options, general management strategies can be applied to all patients presenting with dizziness. These strategies include use of medications to control acute vestibular and autonomic symptoms as well as vestibular rehabilitation exercises. Difficult cases that do not respond to treatment efforts should be referred to an otolaryngologist for further evaluation and treatment.

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An estimated 12.5 million Americans over age 65 are affected by dizziness; in fact, dizziness has been reported to have a prevalence of 28 to 34% in adults over age 60 in several epidemiologic studies of dizziness in the community and primary care settings.¹ The majority of older patients who present with dizziness have an identifiable cause that is benign and self-limited.

The primary care physician must be familiar with the differential diagnosis, evaluation, and management of dizziness

in order to identify and treat underlying, potentially life-threatening conditions. It is inappropriate to attribute dizziness to the normal aging process. Referrals to an otolaryngologist, neurologist, cardiologist, psychiatrist, or neurosurgeon may be necessary as dizziness may be caused by a disturbance in any of the balance control systems managed by these specialists.

This two-part article will discuss the diagnosis and management of the most common causes of dizziness in the older adult. In part 1, we review the evaluation of dizziness in the older patient and make general recommendations for managing symptoms. In part 2 (page 46), we discuss in detail the four most common dizziness symptom categories in the older adult (vertigo, presyncope, dysequilibrium, non-specific dizziness) and the underlying causes of each, as well as make specific treatment recommendations for each type.

Scope of dizziness

The prevalence of dizziness increases with age and is more common in women. In one study of 1,000 adults age 65 and older, 30% reported dizziness.² Another study found that 47% of men and 61% of women over age 70 are affected by dizziness.³ Falls, fear of falling, and secondary limitation of activity are consequences of dizziness in the older population.⁴ Dizziness was directly associated with 6.4 to 7.2% of falls in one large study of older patients.²

Postural stability involves the complex integration of visual, proprioceptive, somatosensory, and vestibular signals; therefore, pathology of any of these signals may lead to the sensation of altered orientation in space typically perceived as dizziness. Unless otherwise specified, the general term “dizziness” will be used in these articles to mean vertigo, presyncope, dysequilibrium, and non-specific dizziness.

Although a large number of disorders may contribute to dizziness, approximately 90% of identified causes of dizziness fall into one of seven broad categories (table 1).⁵ A recent prospective study of ambulatory older patients (mean age 74) with dizziness of more than 1 year’s duration identified a cardiovascular cause in 28%, peripheral vestibular disorder in 18%, central neurologic disorder in 14%, more than one diagnosis in 18%, and no attributable cause in 22%.⁶

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Physiology and changes with aging

The maintenance of postural stability involves the central nervous integration of five sensory modalities: vestibular, visual, proprioceptive, touch and pressure, and hearing. Adults tend to rely more heavily on visual cues (such as the location of stable surroundings) for the maintenance of balance, whereas children rely more on proprioceptive (such as footing) and vestibular (such as head position) cues. In the normal state, vestibular receptors in each labyrinth generate resting activity; subsequent head movement produces equal and opposite alterations in the activity in each ear. This in turn leads to the appropriate compensatory eye and muscle movements for the maintenance of gaze and posture. Therefore, the production of spontaneous false or inadequate information by any of the involved sensory organs may produce dizziness and disorders of balance. Any discrepancy between the senses, slow or inaccurate central integration, or abnormal motor function (caused by orthopedic or neurologic disease in older patients such as arthritis or Parkinson's disease) may lead to a mismatch in input causing dizziness and subsequent imbalance.

Changes with aging. Normal aging increases the older individual's susceptibility to dizziness and contributes to a slower recovery from diseases that cause dizziness. **Normal aging, however, is never the cause of dizziness.** Well-described anatomic and physiologic changes associated with aging that make older adults susceptible to dizziness

include a reduction in sensory receptors located in the semicircular canals, saccule, utricle, proprioceptive end organs, and retina.⁷ Vision and visual-vestibular reflexes are known to decline with advancing age. Because adults rely heavily on vision to compensate for vestibular and postural control deficits, a decline in vision contributes significantly to dizziness and subsequent imbalance in older individuals.

Symptom categories

A key aspect in the evaluation of a dizzy patient is eliciting a detailed description of the character of the sensation in the patient's own words. Although dizziness can be classified into the following four broad symptom categories,⁸ it is important to keep in mind that the clinician may be unable to assign the older adult's symptom to a single category. Also, more than one symptom type may be present.

Vertigo is a sensation in which patients feel that their environment is moving. Although the sensation is often rotational, patients also may feel as though they are falling. Vertigo is usually episodic, begins abruptly, and is often associated with nausea or vomiting when it is severe. Vertigo is typi-

cally caused by a disturbance of the peripheral vestibular apparatus (inner ear or eighth cranial nerve) or connections in the CNS. Differences in the manifestations of peripheral and central vertigo are provided in table 2. Common causes of peripheral vertigo include benign paroxysmal positional vertigo (BPPV), acute labyrinthitis, and Meniere's disease. Central causes of vertigo include mass lesions located in the CNS, and vascular, compromise, and inflammatory disorders involving the CNS. These diseases will be discussed in detail in part 2.

Presyncope is usually described as a sensation of impending faint or loss of consciousness, and may begin with diminished vision or roaring in the ears.

The presyncope symptom complex denotes diffuse cerebral ischemia, due to cardiac causes (eg, dysrhythmias or aortic stenosis), non-cardiac causes (eg, postprandial hypotension, vasovagal episodes, orthostatic hypotension, medications), or both.

Dysequilibrium is a feeling that a fall is imminent and is characterized by unsteadiness or imbalance that occurs only when erect and primarily involves the trunk and lower extremities rather than the head; the sensation disappears when sitting or lying. Dysequi-

Table 1 Seven broad causes of dizziness

Peripheral vestibular disorders	Central and primary neurologic disorders
Cardiovascular disorders	Psychiatric disease
Multisensory dizziness	Hyperventilation syndrome
Brainstem cerebrovascular disease	

Source: Prepared for Geriatrics by Deborah A. Eaton, MD, and Peter S. Roland, MD.

Table 2 Manifestations of peripheral and central vertigo

Cause	Nausea and vomiting	Ataxia	Hearing loss	Neurologic symptoms	Compensation
Peripheral vertigo	Severe	Rare	Common	Rare	Rapid
Central vertigo	Moderate	Common	Rare	Common	Slow

Source: Prepared for Geriatrics by Deborah A. Eaton, MD, and Peter S. Roland, MD.

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librium is often continuous (ie, no intervening periods of a normal sense of balance) and only uncommonly occurs in paroxysms. Dysequilibrium is often caused by dysfunction of more than one sensory modality and frequently involves some deterioration or injury to the nervous system. Common causes of dysequilibrium include severe bilateral vestibular disease, stroke, neurosensory deficits, cerebellar disease, or peripheral neuropathies.

Non-specific dizziness is described by the patient as a vague sensation of light-headedness, and includes symptoms that cannot be distinctly identified as vertigo, presyncope, or dysequilibrium. Patients may use terms such as “heavy-headedness” or “wooziness” to describe this sensation. This type of dizziness may be caused by less severe forms of the above-mentioned disorders, but is often due to anxiety, phobic disorders, or hyperventilation.

Time course

In addition to the patient’s description of the dizziness, the time course of the patient’s symptoms can provide important clues to diagnosis. It is helpful to elicit the temporal component of individual attacks as well as the entire course of the disorder. At the simplest level, one can distinguish between episodic (ie, symptoms that come and go) or continuous symptomatology.

● **Less than 1 minute.** Episodes of acute, rotational vertigo lasting less than 1 minute are most commonly associated with disorders of the peripheral vestibular system such as BPPV.

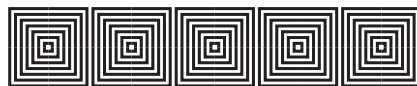
● **Less than 1 hour.** Dizziness lasting a few minutes to 1 or 2 hours can be caused by Meniere’s disease, transient cerebral hypoperfusion (causing presyncope), or phobic/anxiety disorders.

● **Several hours to 24 hours.** This type of dizziness indicates processes that cause continuous dysequilibrium or Meniere’s disease. Viral or vascular labyrinthitis usually presents with acute rotational vertigo of several days’ duration with gradual improvement.

History

A complete medication history is critical to the evaluation. Cardiovascular drugs including diuretics, beta blockers, and vasodilators may produce presyncope. Ototoxic drugs (eg, aspirin, aminoglycosides) typically cause dysequilibrium and oscillopsia (ie, oscillating vision in which objects seem to jerk or wiggle with head movement). Psychotropic medications, muscle relaxants, and anticonvulsants in therapeutic and toxic dosages have also been associated with dysequilibrium. Alcohol, caffeine, and over-the-counter drug use (including dietary supplements) should also be assessed.

The history should specifically seek information about systemic disorders that interfere with cerebral blood sup-



It is important to determine the relationship between dizziness, position, and motion



ply (such as vasculitis) and may produce vertigo due to either focal brain stem involvement or diffuse cerebral ischemia. Non-specific constant dizziness can follow head trauma. Cardiac abnormalities (such as arrhythmia or valvular stenosis) may cause recurrent presyncopal episodes.

Associated symptoms such as hearing loss, tinnitus, nausea and vomiting, and cranial nerve deficits should also be noted because the presence of such symptoms localizes the pathology to the labyrinth, neurovascular bundle, or brainstem. Hearing loss and tinnitus suggest a labyrinthine source, whereas cra-

nial nerve deficits tend to localize pathology to the brainstem or peripheral nerve.

It is also important to determine the relationship between dizziness and position and motion. For example, BPPV occurs with a rapid change in position whereas orthostatic hypotension occurs when the patient is standing. Symptoms from Meniere’s disease, stroke, or cardiac disease are unrelated to position or motion. Dysequilibrium due to multisensory loss occurs when the patient is moving (typically during ambulation) but is absent when the patient is sitting still.

Examination

The dizziness simulation battery has been advocated for the office evaluation of dizziness (table 3).⁵ Patients are asked to identify which of eight different maneuvers most closely reproduces their dizziness. This test battery includes an assessment of hyperventilation, orthostatic hypotension, peripheral vestibulopathy, and carotid sinus stimulation as well as multisensory disturbances.

The eight tests are designed to be easily administered in the office setting. After performing one or more maneuvers, the patient is asked to identify which maneuvers most closely reproduced his dizziness symptoms. Orthostatic blood pressure testing can identify orthostatic hypotension if there is a significant decrease in systolic blood pressure (20 to 25 mm Hg decrease) between the first blood pressure measurement (taken when the patient is lying down) and the second blood pressure measurement (taken when the patient is standing). This test can identify patients with presyncope. The potentiated Valsalva maneuver is also designed to cause presyncope symptoms, and requires the patient to squat for 30 seconds then stand and blow into a sphygmomanometer at 40 mm Hg for 15 seconds. Straining against a closed glottis and carotid sinus stimulation may also produce presyncope symptoms.

The head-hanging positioning maneuver (Dix-Hallpike) is a simple in-office screen for BPPV. The patient is

Table 3 Dizziness simulation battery

Maneuver	Description	Dizziness produced
1. Orthostatic blood pressure testing		
	Patient's blood pressure is measured first while the patient is lying down, and then while the patient is standing. A 20 to 25 mm Hg decrease in systolic blood pressure indicates orthostatic hypotension.	Produces presyncope resulting from orthostatic hypotension.
2. Potentiated Valsalva maneuver		
	Patient squats for 30 seconds then stands up and blows into a sphygmomanometer at 40 mm Hg for 15 seconds.	Produces presyncope indicating presence of orthostatic hypotension, vasovagal attacks, or decreased cardiac output.
3. Carotid sinus stimulation		
	Gently massage the area of the patient's carotid bulb for a few seconds with continuous ECG monitoring.	Produces presyncope indicating presence of orthostatic hypotension, vasovagal attacks, or decreased cardiac output.
4. Dix-Hallpike maneuver		
	Patient is seated on a table and is rapidly lowered until the head hangs over the table. The position is held for at least 10 seconds. See www.geri.com for an animated illustration of this maneuver.	Produces vertigo in patients with BPPV.
5. Barany rotation		
	Patient is seated in a swivel chair with the head tilted down 30 degrees. The chair is then spun 10 times.	Produces vertigo in anyone who maintains some vestibular function.
6. Walk and turn		
	Patient walks 6 to 10 feet, turns around, and walks back to the starting point.	Produces dysequilibrium caused by multisensory loss.
7. Seated head turn		
	While seated in a chair, patient is asked to turn the head as if watching an airplane fly across the sky. Having the patient perform this maneuver once is enough to elicit symptoms.	Produces dysequilibrium caused by multisensory loss.
8. Hyperventilation (30 seconds)		
	Patient breathes into a paper bag for 30 seconds.	Produces non-specific dizziness caused by hyperventilation, indicating the possible presence of anxiety or a phobic disorder.

Source: Prepared for Geriatrics by Deborah A. Eaton, MD, and Peter S. Roland, MD, using information from reference 5.

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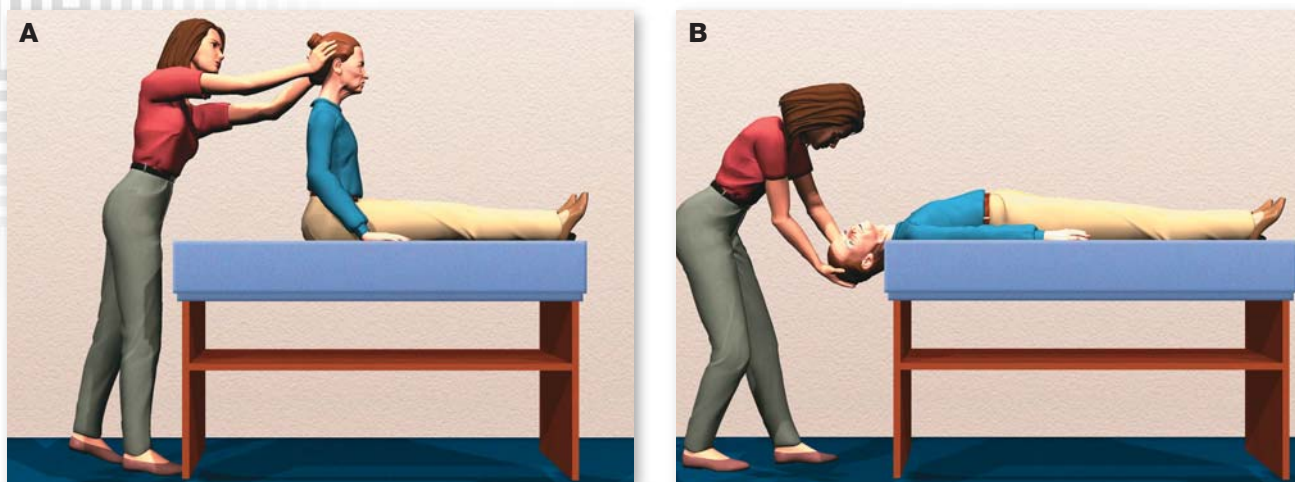


Figure. The Dix-Hallpike maneuver is one component of the dizziness simulation battery. It begins with the patient seated on a table with the head turned 45 degrees (A). The examiner then rapidly brings the patient to a supine head-hanging position (B). An animated illustration of this maneuver can be viewed on the Geriatrics web site at www.geri.com.

Illustrations by Medical Imagery

placed in a seated position on a table and then rapidly lowered until the head hangs over the table (figure). The position is held for at least 10 seconds. A delayed (few seconds) onset of brief, fatigable horizontal-rotary nystagmus is diagnostic of BPPV. An animated illustration of the Dix-Hallpike maneuver can be viewed on the Geriatrics web site at www.geri.com.

The Barany rotation is conducted by spinning the patient in a swivel chair 10 times while the patient's head is tilted down 30 degrees. This maneuver is designed to stimulate the horizontal semicircular canals and will produce vertigo in anyone who retains some vestibular responsiveness. The walk and turn assessment and seated head turn will identify patients with dysequilibrium due to multisensory loss. Having the patient breathe into a paper bag for 30 seconds will cause dizziness due to hyperventilation.

Cardiac arrhythmias, murmurs—particularly aortic stenosis, and evidence of peripheral vascular disease should also be carefully assessed during the primary evaluation, as these signs will further focus the differential diagnosis and guide the diagnostic evaluation.

Diagnostic testing

The evaluation often can be limited to a careful history and physical examina-

tion, particularly in straightforward cases of BPPV, which can be identified by conducting the Dix-Hallpike maneuver.

An audiogram is helpful in the evaluation of Meniere's disease and acoustic neuroma. Abnormalities in brainstem auditory evoked potentials may indicate the presence of suspected eighth-nerve diseases such as multiple sclerosis and acoustic neuroma. Specific screening laboratory tests, which may be helpful in difficult cases of dizziness, include complete blood cell count, blood glucose, blood urea nitrogen, calcium, liver function, VDRL, and thyroid function.

MRI is the diagnostic procedure of choice for the detection and evaluation of tumors. The use of Holter monitoring to identify arrhythmias that may be contributing to presyncope is controversial, as one report found that only 7% of exams showed abnormalities in the absence of other cardiac symptoms.⁶ Symptoms suggesting transient ischemic attacks should be evaluated with carotid and vertebral artery Doppler examinations.

A vestibular assessment including electronystagmography (ENG), rotational testing, and posturography may be helpful in select cases of dizziness where the diagnosis is still unclear after history and physical exam. The ENG records eye movements by measuring

corneoretinal potentials during different maneuvers. The entire ENG evaluation consists of several subtests: the Dix-Hallpike maneuver, caloric testing (which assesses the horizontal semicircular canals), and the saccade, tracking, and optokinetic tests. The caloric test is very sensitive in identifying unilateral peripheral vestibular deficits, and is not affected by aging.⁷

Rotational chair testing assesses the vestibulo-ocular reflex whereas posturography tests the integrity of the vestibulospinal tracts. Test results indicate dysfunction in these areas and help to focus rehabilitation efforts.

Consultation with a neurologist or otolaryngologist should be considered in patients with chronic dizziness and no apparent underlying cause.

General management strategies

When possible, treatment should be directed at the underlying cause of the dizziness and will be discussed further in part 2 of this article. In general, medical treatment for patients with a sudden loss of vestibular function is aimed at controlling the acute vestibular and autonomic symptoms. Five main classes of drugs are used: antihistamines (eg, meclizine), phenothiazines (eg, promethazine), anticholinergics (eg, scopolamine), 5-HT₃ antagonists (eg, on-

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dansetron), and benzodiazepines (eg, diazepam). All medications should be administered sparingly and for short durations, however, as they cause a reduction in central nervous system compensation in all patients. Meclizine is typically administered orally in doses of 12.5 to 25 mg tid as needed for acute peripheral vertigo only (such as acute attacks of labyrinthitis, vestibular neuritis, or Meniere's disease). Older patients should be started on the lowest dose of this medication. Benzodiazepines (usually diazepam prescribed orally at 2.0 to 7.5 mg/d) are helpful in suppressing the central response to chronic vestibular vertigo. 5-HT₃ antagonists may be quite helpful if nausea is a significant component of the clinical picture but are not helpful in reducing vestibular symptoms. All of these medications should be used cautiously in older individuals in order to avoid sedative side effects. Use the lowest dose possible to achieve symptom control.

Vestibular rehabilitation has been used as adjunctive therapy in the United States since the 1940s. It involves specific habituation exercises designed to enhance the normal adaptive mechanisms in the central nervous system. Program strategies vary depending on the patient's primary problem, but are aimed at stabilizing gaze and posture, improving central nervous system adaptation, and reconditioning. Essential components of effective vestibular rehabilitation include gaze stabilization, balance retraining, and desensitization. Each aspect is addressed separately using different exercises.

- Gaze stabilization exercises promote vestibular adaptation through exercises that stimulate the vestibular ocular reflex (such as moving the head while reading text and vice versa).

- Balance retraining starts with activities that progressively decrease the patient's base of support and progresses to gait exercises performed on varied surfaces (such as stairs, balance beams, etc.). The tandem gait is an appropriate alternative for older patients who have difficulty on the balance beam.

Having patients attempt to walk while reading, carry objects while walking, and walk through crowded hallways challenges the balance system. Strengthening exercises are prescribed to improve muscle weakness and flexibility.

- Repetitive head and eye movements designed to provoke vertigo and unsteadiness enhance the central nervous system adaptive mechanisms.


- These exercises are usually repeated until they are no longer tolerated, and the number of repetitions is slowly increased over a 6- to 8-week period. Vestibular rehabilitation is usually administered as adjunctive therapy by a physical therapist with experience treating older patients. These habituation strategies have been found most effective in patients with positional vertigo or a sudden loss of vestibular function.⁹

- A patient handout on the Geriatrics web site (www.geri.com) provides examples of these exercises that older patients can do at home. Physicians can download the handout and print out copies for their patients.

Most cases of dizziness in older adults can be treated once the cause(s) have been identified. Often, simple measures such as correcting vision, modifying a drug regimen, balance retraining, or treating a cardiac arrhythmia are all that is necessary to improve the patient's quality of life. A minority of patients, however, will have no single identifiable cause for their dizziness and will be impossible to cure. These patients typically have multiple medical problems with significant impairment and are most appropriately treated with supportive measures such as vestibular rehabilitation exercises, physical therapy, and tools that provide additional proprioceptive input, such as canes and walkers. Particularly challenging cases should be referred to an otolaryngologist for further work-up and possible chemical or surgical ablative therapy.

Conclusion

Dizziness is a common presenting complaint among older patients in primary care. Physicians investigating complaints of dizziness can use the four

dizziness symptom categories to narrow down the possible causes. Thorough history, examination, and diagnostic testing help to tease out the differential diagnosis. General management strategies that can be applied to all older adults include use of medications to control acute vestibular and autonomic symptoms as well as vestibular rehabilitation exercises. In part 2, we discuss in detail the underlying causes of the four symptom categories and provide specific treatment approaches for each cause. 

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