ENT Series

Dizziness in the older adult, part 2

Treatments for causes of the four most common symptoms

Deborah A. Eaton, MD • Peter S. Roland, MD

Dizziness is a common presenting complaint among older patients in primary care. A thorough examination and history can identify the type of dizziness and point to a specific differential diagnosis. Vertigo, presyncope, dysequilibrium, and non-specific dizziness are associated with a variety of underlying causes, each with specific treatment options.

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The evaluation of dizziness in the older adult is challenging due to the wide range of diagnostic possibilities. Vertigo, presyncope, dysequilibrium, and non-specific dizziness are the four most common dizziness symptom categories in the older adult, all with multiple underlying causes. In part 1 of this article (page 28), we defined these four symptom categories and discussed the evaluation and general management of dizziness. In part 2, we discuss in detail the underlying causes of the four symptom categories and provide specific treatment approaches for each cause.

As a review, definitions of the four symptom categories precede the differential diagnosis of each. The table outlines pharmacologic treatment options for older patients with dizziness.

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Vertigo

Vertigo is a sensation in which patients feel that their environment is moving or that they are falling or spinning. It is usually episodic, begins abruptly, and is often associated with nausea or vomiting.

Benign paroxysmal positional vertigo. Episodic vertigo in older adults is most often positional. After serious disease has been excluded, positional vertigo in older adults is frequently referred to as benign positional vertigo. Benign paroxysmal positional vertigo (BPPV) is a specific variety of benign positional vertigo characterized by fatigable nystagmus with a short latency period that occurs when the Dix-Hallpike maneuver (see part 1) is performed. Patients with BPPV typically present with brief episodes of vertigo associated with a change in head position often while turning in bed. Twenty to sixty seconds of rotational vertigo when turning to one side in the supine position is the classic complaint and is almost diagnostic. Extending the neck to look and reach up also typically produces symptoms in patients with BPPV. This cause of vertigo is thought to result from a degenerative process involving the otoconial membrane of the utricle and saccule that causes displacement of otoconial crystals into the perilymph. Subsequent head position changes in the plane of the posterior semicircular canal provoke vertigo. Prior episodes of viral labyrinthitis, Meniere's disease, and head trauma are thought to be predisposing factors.

Treatment. Most cases will eventually remit spontaneously; therefore reassuring patients that their prognosis is favorable is an important aspect of treatment. Repositioning maneuvers that involve falling or rolling several times in succession in a manner that provokes the dizziness may be performed in the physician's office and the patient's home in order to accelerate remissions. This repositioning maneuver, also known as the Epley maneuver or canalith repositioning procedure, has a success rate near 90%. Antivertiginous medications (eg, meclizine, diazepam, or promethazine) can be used for symptomatic treatment as needed, although caution must be used when prescribing sedating medications to older adults.

Acute labyrinthitis. Acute labyrinthitis is characterized by the sudden onset of severe vertigo often accompanied by visceral autonomic symptoms including nausea, vomiting, and diaphoresis. Hearing loss is absent. Severe symptoms usually last for 1 to 5 days followed by a 2- to 3-week period of gradual, progressive improvement.

continued

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Mehta NN, Greenspon AJ. Atrial fibrillation: Rhythm versus rate control. Geriatrics 2003; 58 (April):39-44.

1. All of the following are clinical risk factors for stroke in older patients with atrial fibrillation EXCEPT:
   - ☐ a. hypertension
   - ☐ b. congestive heart failure
   - ☐ c. diabetes mellitus
   - ☐ d. prior myocardial infarction
   - ☐ e. prior TIA

2. A 75-year-old woman comes to the hospital with the recent onset of dyspnea and is found to be in atrial fibrillation at a rate of 120 beats per minute. She has no prior cardiac history. Physical examination reveals BP 130/80, pulse 120/min, clear lungs, no cardiac murmur. ECG shows atrial fibrillation with no ST or T wave changes. Diagnostic laboratory evaluation should include which of the following:
   - ☐ a. serum calcium
   - ☐ b. serum magnesium
   - ☐ c. troponin
   - ☐ d. serum potassium
   - ☐ e. TSH

3. Stroke prevention in patients with atrial fibrillation is best achieved when warfarin is dosed to a target INR of:
   - ☐ a. 1.5 to 3.5
   - ☐ b. 2 to 3
   - ☐ c. 2.5 to 3.5
   - ☐ d. 3 to 4
   - ☐ e. dose by body weight. No target INR is important.

4. Atrial fibrillation independently increases the risk for new cardiac events.
   - ☐ a. True
   - ☐ b. False

5. Atrial fibrillation itself induces electrical changes within atrial myocardium which promote further development of atrial fibrillation.
   - ☐ a. True
   - ☐ b. False

6. A 78-year-old man complains of chest pain and lightheadedness of 3 hours duration. He has a past history of hypertension and coronary artery disease. Five years ago he sustained an uncomplicated inferior wall myocardial infarction. His medications include enalapril and pravastatin. On physical examination, BP is 80/50 with a pulse rate of 140 beats per minute. Neck-no JVD; Lungs-bibasilar rales; heart-grade III/VI holosystolic murmur at the apex. ECG shows atrial fibrillation, old inferior wall MI, ST segment depression in leads V4-6. Which is the most appropriate next step in therapy?
   - ☐ a. oral metoprolol
   - ☐ b. oral diltiazem
   - ☐ c. intravenous verapamil
   - ☐ d. intravenous digoxin
   - ☐ e. direct current cardioversion

7. Dual chamber pacing for patients with symptomatic sinus node dysfunction prevents the onset of atrial fibrillation.
   - ☐ a. True
   - ☐ b. False

8. A 78-year-old woman with a history of hypertension develops the new onset of atrial fibrillation and is started on intravenous heparin. A transesophageal echocardiogram is performed and no intracavitary thrombus is seen. Direct current cardioversion is successfully performed. Appropriate management at this point should include:
   - ☐ a. continue heparin for additional 24 hours
   - ☐ b. oral warfarin for 4 weeks
   - ☐ c. no anticoagulation is needed
   - ☐ d. warfarin for 1 week and repeat TEE
   - ☐ e. aspirin for 4 weeks

In addition to the exam questions, answer the following evaulation questions: (1=strongly agree, 6=strongly disagree)

1. The information presented in this article was useful.
   1 2 3 4 5 6

2. The information presented was fair, objective, and balanced.
   1 2 3 4 5 6

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...l origin is the most likely cause, and patients have an associated upper respiratory infection in approximately 50% of cases.

Treatment. Treatment is directed at relieving symptoms by using antivertiginous medications (meclizine, 12.5 to 25 mg every 6 to 8 hours as needed, or low-dose diazepam, 2.0 mg/d). Five to ten days of systemic steroids have been shown to shorten the course of severe symptoms.[1] Vestibular exercises to accelerate compensation are usually all that is necessary. Recovery tends to be slow in older adults. Acute labyrinthitis must not be confused with Ramsay-Hunt syndrome (herpes zoster oticus). Herpes zoster oticus is a symptom complex caused by reactivation of the dormant herpes zoster virus in the cranial nerves. It typically presents as facial paralysis associated with otalgia, external ear vesicular eruptions, and sometimes sensorineural hearing loss and dysequilibrium. Treatment of herpes zoster oticus includes analgesia, oral steroids, and antiviral agents (eg, valacyclovir).

Meniere's disease. Meniere's disease is characterized by paroxysmal vertigo lasting minutes to hours accompanied by fluctuating low-frequency hearing loss, tinnitus, and aural fullness. Typically, attacks occur suddenly with the onset of incapacitating vertigo, roaring unilateral tinnitus, and ipsilateral hearing loss. These attacks are often associated with nausea and vomiting. Resolution usually occurs over several hours. Age of onset is typically between the third and sixth decades, and up to 20% of patients will have a family history of Me-

Table Pharmacologic treatment options for older patients with dizziness*

Agent	Recommended dosing	Comments/precautions
Anticholinergic		
Meclizine (Anivert)	12.5 to 25 mg every 6 to 8 hours as needed	Can be used for symptomatic treatment of BPPV, acute labyrinthitis, Meniere's disease, stroke.
Antiplatelet agents		
Aspirin	81 to 325 mg/d	Prevention of subsequent brain stem stroke, treatment of vertebrobasilar insufficiency.
Clopidogrel (Plavix)	75 mg/d	Treatment of vertebrobasilar insufficiency.
Benzodiazepine		
Diazepam (Valium)	2.0 to 7.5 mg/d. Start with 2 mg/d then increase dose and frequency as needed to alleviate symptoms.	Can be used for symptomatic treatment of BPPV, acute labyrinthitis, Meniere's disease, brain stem stroke. Use with caution in older adults as diazepam has a long half-life and is associated with delirium in this population.
Corticosteroid		
Prednisone	Start at 1.0 mg/kg/d for 5 days, then slowly decrease over 2 to 3 weeks until the drug is discontinued.	Can be used for symptomatic treatment of acute labyrinthitis.
Diuretic		
Hydrochlorothiazide	12.5 to 25 mg/d	Treatment of Meniere's disease.
Nonselective phenothiazine		
Promethazine HCl (Phenergan)	12.5 to 25 mg qid	Can be used for symptomatic treatment of BPPV; relief of nausea and vomiting associated with brain stem stroke.

*Before prescribing any agent, check for possible Black Box warnings, investigate possible drug-drug interactions, review potential adverse effects, and verify dosing recommendations.

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

niere's disease. The underlying pathology is endolymphatic hydrops, which results in excess endolymph and subsequent displacement of inner ear structures. The clinical course is highly variable, with reversible hearing loss in early stages and permanent hearing loss in later stages of the disease. The resulting vertigo, however, is disruptive to the patient's quality of life.

Treatment. Salt restriction and diuretics are the mainstays of treatment. Vestibular suppressants can often reduce the severity of acute attacks of vertigo. Low-dose diazepam, 2 to 7.5 mg/d, is generally more effective and less sedating than meclizine, 12.5 to 25 mg every 6 to 8 hours as needed. Both medications are best used with caution on an as needed basis to treat or prevent individual episodes. Surgery for the treatment of endolymphatic hydrops may be necessary in severe cases and consists of decompression with or without shunt placement. Vestibular nerve section has also been successful in patients with disabling vertigo associated with Meniere's disease.

Vertigo is a feeling that the environment is moving, or that the patient is falling or spinning

Vertebrobasilar insufficiency. Ischemia of the labyrinth or the central vestibular nuclei within the brain stem may result in episodes of acute vertigo associated with focal neurologic deficits such as diplopia, hemiparesis, dysarthria, headache, and blurred vision. The diagnosis is usually based on clinical examination because there are no definitive diagnostic tests for vertebrobasilar insufficiency. Magnetic resonance angiography evaluates the posterior circulation and may show infarction in the distribution of the vertebrobasilar system. Atherosclerotic disease of the vertebral and basilar arteries and compression of the vertebral arteries by the cervical vertebrae can cause transient posterior fossa hypoperfusion during head turning and head extension, resulting in vertigo.

Treatment. Treatment consists of controlling risk factors (eg, diabetes, hypertension, hyperlipidemia) and using antiplatelet drugs (eg, aspirin, clopidogrel).

Brain stem stroke. Occasionally, a brain stem stroke is misdiagnosed as acute labyrinthitis because the patient only presents with vertigo, nausea, and vomiting following even small, brief head movements. The patient may, therefore, be quite unaware of the other neurologic deficits produced by the infarct. A screening neurologic examination will rapidly uncover these additional symptoms and physical findings.

Lateral medullary infarction produces a syndrome consisting of vertigo, nausea, ipsilateral facial numbness, contralateral loss of pain and temperature sense, Horner's syndrome, and a tendency to fall toward the side of the lesion due to posterior inferior cerebellar artery infarction. Lateral pontomedullary infarction results from occlusion of the anterior inferior cerebellar artery and results in severe vertigo, nausea, vomiting, unilateral hearing loss, tinnitus, facial paralysis, and asymmetric cerebellar dysfunction.

Treatment. Patients who experience a brain stem stroke usually have risk factors for atherosclerotic vascular disease (ie, diabetes, hypertension, hyperlipidemia) that must be addressed. Aspirin therapy should be instituted unless it is contraindicated. Management of the associated vertigo is directed at relieving symptoms by using antivertiginous and antiemetic medications as well as vestibular exercises to accelerate compensation. However, antivertiginous medications and vestibular exercises are often not effective for controlling vertigo of central origin.

Tumors. Cerebellopontine angle, brain, and temporal bone tumors may present with a sensation of vertigo or dysequilibrium. Tumors involving the middle or inner ear will generally cause hearing loss associated with vertigo. The internal auditory canal is an occasional site of metastatic tumor growth, and symptoms of compression of the seventh and eighth cranial nerves will evolve.

Treatment. Surgical resection is the treatment of choice for acoustic neuromas, glomus tumors, and most other cerebellopontine angle tumors. Radiation therapy should be considered in non-resectable cases.

Perilymph fistula. A perilymph fistula, a rare cause of dizziness, results in a loss of perilymph from the inner ear, and can cause episodic vertigo. Head injury, a very forceful Valsalva maneuver, or barotrauma are thought to induce a fistula, and the cause should be apparent from the patient's history. When loss of perilymph is rapid and voluminous, severe rotational vertigo and hearing loss are the result. Very low volume leak, or episodic leaks may produce only episodic vertigo or dysequilibrium. Moreover, when leaks are slow, hearing loss may be absent.

Treatment. Surgical treatment consists of exploring the middle ear and grafting in the area of the oval and round windows.

Cervical vertigo. Cervical dizziness is episodic vertigo brought on by neck disease. It is characterized by vertigo that occurs during neck movements, especially extension of the neck. There are two types of cervical vertigo. The first type results from decreased cerebral perfusion caused by extrinsic vascular compression (principally of the vertebral arteries) in the neck due to bony abnormalities of the cervical spine.

The second type is caused by altered proprioceptive feedback in the neck joints and muscle stretch receptors in the neck. This type of cervical vertig

ually associated with chronic cervical spine disease such as facet joint osteoarthritis.

Treatment. Neck exercises and improved posture usually alleviate these symptoms. Further management in the case of extrinsic vascular compression is discussed in the "Proprioceptive and somatosensory loss" section.

Presyncope

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

Orthostatic hypotension. This form of dizziness occurs when the patient is standing. In older adults, postural dizziness often occurs in patients whose symptoms do not strictly meet the criteria for orthostatic hypotension (ie, a 20 mm Hg drop in systolic blood pressure or a 10 mm Hg drop in diastolic blood pressure 2 minutes after standing that produces postural dizziness and light-headedness).[2] Older patients may present with classic presyncope symptoms, but their blood pressure readings may not precisely meet these criteria. Postural hypotension may be caused by pooling of blood in the lower extremities, medications, prolonged bed rest, or autonomic dysfunction.

Vasovagal attacks. Vasovagal attacks may occur as dramatic blood pressure drops between 10 and 30 minutes after assuming upright posture (ie, sitting upright or standing). Patients typically exhibit nausea, light-headedness, and pale, cold, and clammy skin. These "faints" can be caused by swallowing, GI conditions such as diarrhea and fecal impaction, fright, and pain. They may also be induced when strong emotions initiated in the limbic system activate brain stem medullary vasodepressor centers. Parasympathetic hyperactivity causes a decrease in cardiac output leading to a decrease in cerebral blood flow and the resulting attack.

Decreased cardiac output. Any cardiac condition causing impaired cardiac output, including arrhythmia, congestive heart failure, MI, and valvular disease such as aortic stenosis may lead to presyncopal light-headedness.

Presyncope is a sensation of impending faint or loss of consciousness

Treatment. Orthostatic hypotension and vasovagal attacks are usually treated by addressing the underlying medical problem (eg, cardiac arrhythmia, volume depletion) and by preventing blood pooling in the lower extremities with elastic stockings. Increased salt intake may also increase blood volume in patients with autonomic insufficiency. To prevent orthostatic hypotension, patients should sit on the edge of the bed and flex their feet before standing. Decreased cardiac output should be treated according to the underlying cause (eg, congestive heart failure, aortic stenosis).

Dysequilibrium

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.

Vestibular loss. A slow unilateral loss of vestibular function (such as in association with an acoustic neuroma) or bilateral symmetric vestibular loss results in persistent unsteadiness. Patients may say they feel "a little dizzy." This type of imbalance is typically worse in the dark when visual cues are not available to help compensate for the loss in vestibular function. A common etiology of bilateral vestibulopathy is ototoxic drug exposure, especially in patients with renal dysfunction. Oscillopsia, or oscillating vision in which objects seem to jerk or wiggle with head movement, is a common associated symptom.

Treatment. Bilateral severe vestibular loss is often a side effect of ototoxic medications, which should be altered or withdrawn. A slow, unilateral loss of vestibular function is usually the result of a tumor (such as an acoustic neuroma), which should be treated appropriately. Vestibular rehabilitation can often recover significant function.

Proprioceptive and somatosensory loss. Dysequilibrium caused by a somatosensory disturbance is usually worst in the dark and is often the result of a peripheral neuropathy, which is common in patients with diabetes or renal failure. Osteoarthritis of the cervical spine may lead to proprioceptive disturbance and cause dysequilibrium resulting from spinal cord compression; these patients also demonstrate weakness in the cervical spine motor distribution and bowel or bladder dysfunction.

Treatment. If the dysequilibrium is caused by cervical osteoarthritis and subsequent nerve compression, appropriate orthopedic and neurosurgical referrals should be made. Measures such as balance training with a physical therapist and the use of a walking stick to introduce additional proprioceptive input are often beneficial.

Motor and cerebellar lesions. Lesions of the frontal lobes or basal ganglia will cause dysequilibrium associated with weakness, rigidity, or tremor. Cerebellar lesions cause severe dysequilibrium whether or not the patient attempts to stabilize himself by visual fixation and are often associated with oscillopsia, a wide-based gait, and truncal ataxia. Lesions of the cortical and subcortical motor centers lead to gait disturbance and may be caused by Parkinson's disease, multiple subcortical infarcts, tumors, and communicating hydrocephalus.

continued

Treatment. Most gait disorders are not reversible, however, and supportive care with physical therapy and canes or walkers is the only treatment option.

Multiple neurosensory impairments. These impairments are sometimes referred to as presbyastasis or dysequilibrium of aging, and are caused by impaired physiologic function in several systems commonly affected by aging (such as the visual system and the musculoskeletal system). One, or more, of the systems involved in postural control are disturbed to such an extent that the other systems are unable to adequately compensate. Multiple neurosensory impairments typically present as a sense of dysequilibrium during standing or walking. Common abnormalities include visual impairment, physical deconditioning, cervical spine disease, peripheral neuropathy, medication side effects, and vestibular hypofunction. This diagnosis should only be considered once other distinct etiologies have been ruled out.

Dysequilibrium is a feeling that a fall is imminent, producing unsteadiness

Treatment. Treatment should be aimed at optimizing those systems that are contributing to the problem. This often includes vision correction, strength training exercises, and vestibular rehabilitation.

Parkinson's disease. Common among older adults, Parkinson's disease produces characteristic postural and motor abnormalities that result in a festinating gait. Patients typically stand in a posture of flexion with the thoracic spine bent forward and the head bent downward. With forward locomotion, the patient takes short, shuffling steps that become successively more rapid and cause the patient to fall if not given assistance. This disturbance of posture and motor control often results in dysequilibrium. The postural instability typically does not respond to levodopa.

Treatment. Management of dysequilibrium associated with Parkinson's disease consists of vestibular rehabilitation exercises.

Non-specific

Non-specific dizziness is described by patients as a vague sensation of lightheadedness, heavy-headedness, or wooziness and includes symptoms that cannot be distinctly identified as vertigo, presyncope, or dysequilibrium.

Hyperventilation. Anxiety or phobic disorders may lead to hyperventilation. In this case, diffuse cerebral ischemia results from constriction of the cerebrovasculature due to a decreased carbon dioxide content in the blood.

Sloane et al[3] found that 37.5% of patients aged 60 and older with chronic dizziness met DSM-III criteria for psychological disorders, with anxiety disorders, adjustment reactions, and depressive disorders being the most common diagnoses. Patients with dysequilibrium caused by psychological disorders often describe a chronic feeling of "wooziness." Two mechanisms have been proposed by the authors: (1) patients with underlying primary psychological disorders may be more susceptible to impairment by diseases affecting neurosensory systems, and (2) dizziness syndromes themselves impair function and may cause secondary psychological symptoms.

Treatment. Management consists of appropriate treatment of the underlying disorder, usually with antidepressants and anxiolytics or psychotherapy.

Conclusion

Dizziness is one of the most common complaints among patients age 50 and older who present to primary physicians. The primary care physician must be familiar with the four most common dizziness symptom categories in order to identify and treat underlying potentially life-threatening conditions. The majority of older patients who present with dizziness have an identifiable cause that is benign and self-limited. A stepwise investigation in the primary care setting can identify underlying, potentially treatable disease. Referrals to the otolaryngologist, neurologist, cardiologist, psychiatrist, or neurosurgeon may be necessary as dizziness may be due to a disturbance in any of the balance control systems managed by these specialists.

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