Diagnosis and Management of Dizziness and Vertigo

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Dizziness is the third most common complaint among all outpatients and the single most common complaint among patients older than 75 years [1]. These patients present to psychiatry clinics, emergency departments, and outpatient offices. In all of these settings, the amount of time that the clinician has to spend with the patient is limited. Chronic cases average five physician visits without resolution (Charles Yanofsky, MD, unpublished data, 2004). For the patient, the ongoing dizziness and imbalance can lead to loss of function, falls, and injuries.

The evaluation of the dizzy patient can certainly be overwhelming for any clinician. Few other complaints have such a broad differential. Dizziness as a chief complaint encompasses weakness, presyncope, neurologic impairment, vertigo, visual disturbance, and psychologic illness. Often difficult and time-consuming to handle, the dizzy patient is commonly referred to medical specialists. Although otolaryngology, neurology, and cardiology all play an important role in the evaluation of the patient, a good history and focal physical examination in the primary care setting can usually reveal the diagnosis.

In addition to diagnosing the patient, the goal of the primary clinician should be to recognize which patients need inpatient management or emergent intervention. This goal becomes particularly important when evaluating older patients. Several acute pathologic conditions can present with dizziness as the initial complaint. This article outlines the diagnostic approach to the dizzy patient, with emphasis on the differentiation of clinical emergencies.

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Diagnostic approach

History

Obtaining a good history is the most critical step in the assessment of the dizzy patient. Because the term dizzy is used by patients to describe a variety of experiences, it is important to clarify the patient’s actual complaint. The sensation of movement or spinning is classic for true vertigo. These patients may complain of objects moving around them (objective vertigo) or that they are spinning relative to their surroundings (subjective vertigo). Other patients may describe light-headedness or weakness. These symptoms should guide the clinician to investigate more systemic diseases consistent with presyncope.

Often, a good history can elicit whether a patient has true vertigo and whether the cause is central or peripheral. Vertigo, which is peripheral in origin, often presents as severe, intense attacks that last several seconds to minutes. Occasionally, more severe episodes last up to several hours and are accompanied by nausea, vomiting, and disequilibrium. Vertigo triggered by a change in position is also suggestive of a peripheral disorder. A central etiology is more concerning in patients who describe mild symptoms that are gradual in onset and last several weeks to months (Table 1).

It is also important for the physician to inquire about associated symptoms. Diseases of the middle and inner ear can cause hearing loss, aural fullness, and tinnitus along with vertigo. The physician should attempt to localize the auditory symptoms to one side. The symptomatic ear is often the one with vestibular damage. Associated neurologic symptoms are more consistent with central vertigo. Headaches may suggest dizziness associated with migraines. Other symptoms suggestive of a central disorder include visual changes, seizures, ataxia, or other gait disturbances. The presence of these symptoms should provoke further investigation and imaging.

A thorough medication history should also be reviewed. Several drugs are directly ototoxic and should be discontinued in any patient complaining of vertigo. These drugs include certain aminoglycosides, furosemide,

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ethacrynic acid, acetylsalicylic acid, amiodarone, quinine, and cisplatinum. Psychotropic medications are also notorious for causing light-headedness and disequilibrium (Box 1), of which the most commonly encountered are anti-Alzheimer’s medications, anticonvulsants, antidepressants, and anxiolytics. In addition, chronic use of vestibular suppressants such as meclizine and scopolamine may result in sensitization. Patients can have severe withdrawal symptoms when these medications are discontinued. A variety of medications can induce toxic labyrinthitis. In these cases, the offending medication should be immediately discontinued.

A complete social history is also important in patients complaining of dizziness. Alcohol, nicotine, and caffeine can all exacerbate vertiginous symptoms. Current or previous use of illicit drugs should be documented. Sexual history should also be noted. Certain sexually transmitted diseases such as syphilis have otologic symptoms. In addition, any history of traumatic head injury or cervical trauma should be investigated. Finally, it is important to remember that depression and anxiety can also manifest as dizziness.

**Physical examination**

After a good history has been obtained, the next important step is a thorough physical examination. Particular emphasis should be placed on the ocular examination. It is important to test pupillary reactivity and extraocular movements. Subtle ocular abnormalities can sometimes be the only clue to cerebellar disease. A fundoscopic examination should always be performed.

### Box 1. Psychotropic medications that may cause dizziness

**Anti-Alzheimer’s** (memantine, rivastigmine, tacrine)

**Antipsychotics**
- Typical (chlorpromazine, prochlorperazine, fluphenazine, perphenazine, thioridazine, trifluoperazine)
- Atypical (all except olanzapine)

**Antidepressants**
- Selective serotonin reuptake inhibitors (all)
- Tryptamines (amitriptyline, nortriptyline, trazodone, imipramine)
- Monoamine oxidase inhibitors (selegiline, phenelzine)
- Others (nefazodone, venlafaxine, mirtazapine, bupropion)

**Anxiolytics** (alprazolam, clonazepam, diazepam, lorazepam, oxazepam, clordiazepoxide)

**Mood stabilizers** (gabapentin, carbamazepine, oxcarbazepine, lamotrigine)

**Anticonvulsants** (phenytoin)
Papilledema usually presents bilaterally and is indicative of elevated intracranial pressure. In these patients, vision is usually well preserved and visual acuity testing does not offer significant additional information.

In patients who have unilateral vestibular disorders, horizontal beating nystagmus may be observed away from the side of the lesion. The abnormal jerk nystagmus that is classical for inner ear disease consists of slow and quick components. Patients exhibiting coarse vertical nystagmus may have a central lesion which is thought to be related to asymmetric vestibular input from both sides. Patients who have peripheral vestibular disease should be able to suppress the nystagmus by focusing their vision on a stationary target. The inability to suppress the nystagmus is suggestive of a central abnormality. It is often helpful to ask the patient’s family whether they noted any unusual eye movements during the acute vertiginous episode which is particularly important with pediatric patients [2].

If nystagmus is not present at rest, then positional testing should be performed. The patient’s eye movement should be noted while lying supine with the head extended and turned to one side. The test should be repeated with the head turned to the other side. Positional nystagmus is strongly suggestive of vestibular disease. This maneuver often reproduces vertiginous symptoms in patients who have a peripheral disorder. Because of the risk of dislodging atheromatous plaques in the vertebrobasilar vessels with sudden turning movements, this maneuver should be avoided in elderly patients.

When examining the ear, the clinician should use an otoscope to look for impacted cerumen or any foreign object in the ear canal. Often, removal of the foreign body relieves the symptoms of vertigo. It is also important to recognize signs of middle ear disease such as fluid behind the eardrum, perforation, or extensive scarring. The patient should be tested for any subtle hearing loss. If the hearing is abnormal, the Rinne and the Weber’s tuning-fork tests can help determine whether the hearing loss is conductive or sensorineural.

The heart and carotid arteries should be auscultated because occasionally, a positive finding points to vascular causes of dizziness. Examinations significant for a carotid bruit, heart murmur, or irregular rhythm should impress upon the physician the need for a cardiovascular work-up. This work-up is particularly important in older patients or those who are at high risk for cerebrovascular disease.

A thorough neurologic examination is important in patients complaining of dizziness. A complete cranial nerve evaluation may help localize lesions of the midbrain, pons, and medulla. The patient’s cerebellar function can be assessed with finger-to-nose pointing and rapidly alternating movements. Romberg’s test is also useful in assessing the dizzy patient. The patient is asked to stand with feet together and arms folded. The inability to maintain posture in this position is suggestive of abnormal proprioception.

Any gait abnormality should arouse suspicion of a central lesion. The main features of an ataxic gait consistent with cerebellar disease are
a wide base, unsteadiness, irregularity of steps, tremor of the trunk, and
lurching from side to side. This unsteadiness is most prominent on arising
quickly from sitting, turning sharply, and stopping suddenly while walking.

Laboratory tests

Most routine testing is not helpful in the evaluation of the patient who
has vertiginous symptoms; however, in the absence of clinical findings or
in the evaluation of the patient who has near syncope, a complete blood
count and chemistry panel can be helpful. Some clinicians also recommend
thyroid function tests, fasting glucose, and rheumatoid factor [3].

Electrocardiography

Because myocardial ischemia can present atypically in many patients, it is
important to obtain an ECG on those patients who are older or have signif-
icant cardiac risk factors. In addition, any patient who requires an emergency
room evaluation after being seen in the outpatient office should have an ECG
reviewed before transfer.

Electronystagmography

Electronystagmography is an examination that records eye movements in
response to vestibular, visual, cervical, caloric, rotational, and positional
stimulation [4]. Electrodes are placed at the outer and inner canthi for hor-
izontal recordings and above and below the eye for vertical recordings. Elec-
tronystagmography testing is helpful in assessing vestibular dysfunction but
is limited in diagnosing nonvestibular disorders [5].

Radiologic imaging

When cerebellar hemorrhage, cerebellar infarction, or other central le-
sions are suspected, an emergent CT or MRI of the brain is indicated. These
patients should be immediately transferred to an emergency department that
has neuroimaging capabilities.

In patients at particularly high risk for cerebrovascular disease, magnetic
resonance angiography can be used to visualize the intracranial vasculature.
Although a less sensitive study than cerebral angiography because of its lim-
ited visualization of small vessels, magnetic resonance angiography remains
more readily used by neurologists to evaluate high-risk patients.

MRI with gadolinium enhancement is particularly useful in detecting
smaller intracanalicular tumors such as acoustic neuromas. It is also recom-
mended for identifying sclerotic and demyelinating white matter lesions char-
acteristic of multiple sclerosis. Although not indicated in younger patients
who have a clear peripheral cause, radiologic imaging should be considered
in all patients who have new-onset vertigo or neurologic findings [6,7].
Peripheral vertigo

Peripheral causes of vertigo arise from abnormalities in the vestibular end organs (semicircular canals and utricle), the vestibular nerve, and the vestibular nuclei. Most of these causes are benign and readily treatable.

Benign paroxysmal positional vertigo

The most common cause of peripheral vertigo is benign paroxysmal positional vertigo (BPPV). As the name implies, this condition is paroxysmal (sudden onset) and positional. Most patients report attacks provoked by turning their head. BPPV is characterized by its fatigability. The patient develops a tolerance to head movements, leading to a reduction in symptoms.

The condition occurs when debris (otoconia) from the utricle circulates within the endolymphatic system, causing positional irritation of the cupula and stimulating vertigo and nystagmus. Occasionally, the debris attaches to the cupula (cupulolithiasis) and symptoms persist for weeks.

The treatment of acute attacks of BPPV centers around symptomatic relief. Benzodiazepines, intravenously and orally, effectively relieve vertiginous symptoms because of their sedative effect. Anticholinergics and antihistamines have also been shown to be helpful in alleviating symptoms by mediating the amount of acetylcholine involved in vestibular reactions. A short course of meclizine or diphenhydramine may resolve the vertiginous symptoms. Antiemetics such as promethazine may improve the nausea associated with vertigo.

Canolith repositioning procedures treat BPPV by directing the otoconia back to the utricle where it is absorbed. The Epley and Semont maneuvers have been shown to be 85% to 95% effective in treating patients who have BPPV [8–10]; however, in more than half of patients, a recurrence of symptoms occurs [11]. In these cases, patient education and reassurance are important.

Otitis media

Patients who have otitis media often complain of vertigo. Because of the proximity of the vestibular end organs to the middle ear, the infectious process may extend to these structures. These patients are at risk for hearing loss and often end up with permanent labyrinthine deficits if left untreated. Extension of the infection into the mastoid can also occur, and these patients may develop an epidural abscess. With the early use of antibiotics and the treatment of the underlying otitis, these complications can usually be avoided.

Labyrinthitis

Labyrinthitis is a peripheral disorder characterized by inflammation of the canals of the inner ear. The cause of labyrinthitis is unknown, but because it
commonly occurs following otitis media or an upper respiratory infection, it is thought to be a consequence of viral or bacterial infection [12]. It may also follow allergy, cholesteatoma, or the ingestion of certain drugs that are toxic to the inner ear. Patients who have acute labyrinthitis usually present with severe vertigo, hearing loss, nausea, vomiting, and fever.

Bacterial infections may directly invade the perilymphatic space, causing a suppurative labyrinthitis. These infections usually extend from the middle ear through a ruptured membrane or perilymph fistula. In patients who have meningitis, the infected cerebrospinal fluid enters the labyrinth through the cochlear aqueduct or internal auditory canal.

Patients who have bacterial labyrinthitis appear ill and require hospital admission and intravenous antibiotics. Occasionally, these patients also need surgical drainage and debridement. Bacterial labyrinthitis is one of the few causes of peripheral vertigo that requires early detection and transfer to the emergency department.

Vestibular neuritis

Vestibular neuritis usually results as a complication of an upper respiratory infection. The prevalence of vestibular neuritis peaks at age 40 to 50 years [13]. The virus affects the vestibular nuclei and causes sudden and severe vertigo, nausea, and vomiting. Auditory symptoms are usually absent. The diagnosis can be made on clinical presentation alone. Treatment with prednisone in the first 10 days of the attack may shorten the course of the illness. The acute attack is debilitating and patients often require bedrest.

Ramsay Hunt syndrome is caused by varicella zoster and is a variant of vestibular neuritis, with involvement of cranial nerves VII and VIII. It causes facial paresis, tinnitus, hearing loss, and a vestibular defect [14]. Patients who have Ramsay Hunt syndrome respond well to early initiation of prednisone and acyclovir.

Cholesteatoma

Cholesteatoma is a benign skin growth that occurs in the middle ear behind the ear drum. It is usually due to repeated infection, which causes an ingrowth of the skin of the eardrum. Over time, the cholesteatoma can increase in size and destroy the surrounding delicate bones of the middle ear. When this benign tumor erodes into the labyrinth, it causes hearing loss and vertigo. The vertigo tends to be severe in these patients but typically does not last beyond a few seconds. Surgical removal of the cholesteatoma is indicated in symptomatic patients.

Trauma

The incidence of dizziness and dysequilibrium following head or neck injury is between 40% and 60%, even following mild or moderate head
injuries not requiring acute hospitalization [15]. Any evidence of significant traumatic injury should incite a complete trauma evaluation [16]. Blunt head injury can concuss the membranous labyrinth with preservation of the otic capsule. Patients may complain of mild vertigo, disequilibrium, and nausea [17]. Symptoms tend to resolve spontaneously over several days to weeks.

Explosive blasts can also result in symptoms of vertigo. Pressure waves classically injure the ear by rupturing the tympanic membrane and disrupting the ossicular chain. The cochlea and hair cells can shear off the basilar membrane, causing significant inner ear damage.

Barotrauma to the inner ear is rare. It results from acute changes in atmospheric pressure. Deep-sea divers and pilots are particularly at risk for this type of injury. A perilymphatic fistula occurs when there is rupture of the oval or round windows that separate the perilymphatic space from the middle ear. Patients who have perilymphatic fistulas from barotrauma usually complain of a sudden onset of vertigo or dizziness. Patients are put on bedrest for 1 to 2 weeks and instructed to avoid any activities that would produce Valsalva-type maneuvers. Most patients heal spontaneously, but surgical repair is recommended in severe cases.

**Endolymphatic hydrops**

The most common form of endolymphatic hydrops is Meniere’s disease. Patients may present with the classic triad of tinnitus, fluctuant sensorineural hearing loss, and vertigo. The vertigo attacks may last several minutes to an hour. It is not typical for these attacks to persist longer than several hours. As the disease progresses, attacks occur more frequently and are more severe. Although the disease starts unilaterally, almost half of patients develop bilateral auditory symptoms.

The predominant pathology of Meniere’s disease is dilation of the endolymphatic system caused by excess production of endolymph or decreased reabsorption. Because salt in the diet is thought to increase the endolymphatic volume, the cornerstone of medical treatment involves salt restriction and diuretics. Greater than 90% of patients respond well to medical management [18]. For patients in whom medical therapy is not effective, surgical options include endolymphatic sac decompression or shunting, vestibular nerve resection, or labyrinthectomy. Chemical ablation of the vestibular apparatus has also gained wide acceptance as a treatment modality. In these cases, gentamycin is injected transtympanically. Although a less aggressive approach than surgery, chemical ablation has a greater risk of hearing loss.

**Acoustic neuroma**

An acoustic neuroma is a tumor composed of the Schwann cells of the vestibular nerve. Although vertigo is the most common presenting symptom, it is often associated with unilateral hearing loss or tinnitus [19]. In
patients who have suspected acoustic neuroma, a gadolinium-enhanced MRI should be ordered. The MRI detects intracanalicular abnormalities with 100% sensitivity and is the “gold standard” for detecting this tumor. Because there is a potential for the tumor to expand intracranially, the patient should be reimaged regularly [20,21]. Treatment options include radiotherapy or surgical removal.

**Central vertigo**

Central vertigo manifests as marked vertigo, nausea, and vertical nystagmus. Neurologic symptoms such as headache or gait ataxia may also be present. In severe cases, patients may have depressed levels of consciousness. The cerebellum is often involved, and etiologies include multiple sclerosis, tumor, hemorrhage, and ischemia. Vascular injuries and infarcts of the central neurologic system can cause permanent debilitating disease. Because central processes have more serious consequences, aggressive work-up and treatment are recommended.

Even in patients who have mild symptoms, it is important to maintain a high level of clinical suspicion when advanced age, atrial fibrillation, hypertension, or previous cerebrovascular disease is present. Often, vertigo is the only presenting symptom in patients who have impending infarction. When a central etiology is suspected, the patient should be transferred immediately by ambulance to an emergency department for neurologic imaging. Evaluation by neurology and neurosurgery may be needed.

**Cerebellar hemorrhage**

In patients who have acute neurologic deficit, it is often difficult to distinguish intracranial hemorrhage from ischemic infarct. It is imperative not to administer anticoagulant medicine, including aspirin, until intracranial hemorrhage has been ruled out by imaging. Because the posterior fossa is a relatively small and nonexpandable space, hemorrhage can lead to rapid compression and compromise of vital medullary functions, obstructive hydrocephalus, or herniation of the medullary tonsils. Endotracheal intubation may be needed to protect the airway, control breathing, and allow therapeutic hyperventilation. Because neurosurgical consultation may be needed for surgical decompression by way of suboccipital craniectomy or ventriculostomy, all patients who have a presentation of central disease should be transferred only to centers that have neurosurgical capabilities.

**Brainstem ischemia**

Vertigo may occur from infarcts in the posterior fossa that contain vestibular pathways. The cerebellar circulation is complex, and it is often difficult to localize the area of ischemia without magnetic resonance angiography.
The anterior inferior cerebellar artery divides into several branches that perfuse the lateral cerebellum, the pons, and the labyrinth. Several types of anterior inferior cerebellar artery syndromes result in acute vertigo. Infarct of the anterior vestibular artery can present with peripheral symptoms only. Infarct of the common cochlear artery causes peripheral symptoms and hearing loss and tinnitus. Patients who have infarcts of the pontine branch can present with central signs such as dysarthria, facial palsy, sensory loss, Horner syndrome, and dysmetria [22].

The posterior inferior cerebellar artery perfuses part of the cerebellum and the dorsolateral medulla. Infarcts in the lateral medulla often damage the vestibular nucleus and cause vertigo. This condition is known as Wallenberg syndrome and characterized by crossed sensory signs, ipsilateral lateropulsion, ataxia, and Horner’s sign.

**Vertebrobasilar insufficiency**

Vertebrobasilar insufficiency occurs when there is narrowing of the arteries that supply the posterior brain (subclavian, vertebral, or basilar arteries). It is usually the result of hardening of the arteries (atherosclerosis) and occurs among patients older than 50 years. The narrowed arteries decrease the blood flow and, therefore, the oxygen to the vestibular center in the brain. Because the vestibular system is very sensitive to a lack of oxygen, difficulty with balance is often one of the first symptoms of vertebrobasilar insufficiency.

Transient ischemic attacks from vertebrobasilar ischemia provoke episodes of dizziness that are abrupt and usually last only a few minutes. They are frequently associated with other symptoms, most commonly visual disturbance, drop attacks, unsteadiness, or weakness. Changing or rapidly progressive symptoms should also raise suspicions of impending posterior circulation occlusion.

Vertebrobasilar insufficiency should be considered in any patient of advanced age who has new-onset vertigo without an obvious cause [6]. These patients should be evaluated by and admitted to the neurology service. Magnetic resonance arteriography can be performed to assess posterior circulation vessels and transcranial Doppler may detect decreased flow in the basilar artery. Treatment includes reduction of risk factors for cerebrovascular disease and antiplatelet therapy. Warfarin is used when there is significant vertebral or basilar artery stenosis [23]. Fig. 1 summarizes the management and disposition for patients who have central or peripheral vertigo.

**Dizziness**

In patients who complain of dizziness without clear vertiginous symptoms, the differential remains broad. Many patients complain of disequilibrium and imbalance, whereas others note light-headedness and other
Presyncope symptoms. Often, dizziness can be a multisensory disorder due to any combination of peripheral neuropathy, visual impairment, and musculoskeletal disease.

**Proprioceptive abnormalities**

Many diseases directly affect the proprioceptive sensory fibers. Chronic alcoholism is among the diseases manifesting with symptoms of dizziness and imbalance. These patients may have deterioration of the vestibulospinal pathways. These symptoms are usually not reversible and the patient must be counseled on safety and fall risk. Chronic alcoholism can also lead to

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**Fig. 1. Management of vertigo.**
compromised vitamin absorption from the gastrointestinal tract, leading to peripheral neurologic changes.

Syphilis is another rare but important cause of dizziness. In tertiary syphilis (tabes dorsalis), there is a deterioration of the posterior columns. These patients have compromised proprioception and often complain of difficulty walking in the dark.

Cerebral anoxia

A number of conditions can lead to poor blood flow to the central nervous system. These patients do not classically complain of light-headedness while sitting or lying down. Their symptoms can usually be reproduced with standing. Anemia may produce cerebral anoxia and can result from any number of causes. Iron deficiency, malignancy, vitamin deficiency, and chronic blood loss are some examples. Patients who have significant atherosclerosis may also complain of positional symptoms. In these patients, other neurologic symptoms including weakness and syncope may be present.

Orthostatic hypotension is often seen in patients who complain of dizziness when arising to a standing position. The symptoms are generally transient and resolve spontaneously. In these cases, medication-induced hypotension must be ruled out. Often, no underlying cause is found for the autonomic response.

Metabolic disorders

Many patients who have thyroid dysfunction can present with dizziness as an initial complaint. Many patients also have dizziness associated with pregnancy or menstruation. It has been well documented that acute changes in hormone levels commonly lead to symptoms of light-headedness. Hypoglycemia can also cause dizziness. In patients who do not have an obvious diagnosis, a finger-stick blood glucose should be performed [24].

Migraines

The mechanism of dizziness or vertigo from migraines is unknown. Migraine is a vascular disease characterized by periodic, unilateral headaches. These headaches are often preceded for a variable time by associated neurologic symptoms called the aura. In individuals who have migraine, dizziness and vertigo can occur as part of the aura or separately. Spells usually last approximately an hour but can last several hours or days in patients who have severe symptoms. Most patients who have migraines have a long history of recurring symptoms.

The management of migraine is divided into two categories: symptomatic and preventive treatments. Acute attacks can be treated with various nonopioid analgesics. Preventive treatment is most frequently accomplished with amitryptiline, β-blockers, calcium channel blockers, and acetazolamide.
Acetazolamide has been particularly effective in treating patients who have vestibular symptoms associated with migraine.

Presyncope

Presyncopal patients complain of feeling faint and light-headed without losing consciousness. Sometimes nausea, dizziness, diaphoresis, and a sense of warmth accompany a feeling of faintness. Patients who have a history of unexplained fainting or recurring presyncope often need an inpatient evaluation to investigate cardiac causes of their symptoms. The work-up and treatment of these patients is discussed more thoroughly in articles on syncope.

Psychogenic

Psychogenic dizziness often occurs in patients who have chronic anxiety. The complaints are often vague, numerous, and out of proportion to the physical findings. In other patients, panic attacks manifest as sudden intense fear or discomfort and reach a crescendo within 10 minutes. They are frequently associated with brief episodes of dizziness, nausea, shortness of breath, chest tightness, paresthesias, and diaphoresis. Physical examination findings in patients who have psychogenic disorders are often dramatic. They include excessive slowness or hesitation, exaggerated sway on standing, and sudden buckling of the knees.

Selective serotonin reuptake inhibitors are the mainstay of treatment for panic disorders and chronic anxiety. Counseling and behavior modification are also frequently helpful.

Summary

Dizziness and vertigo present in patients of all ages. Particularly in older patients, dizziness is associated with a variety of cardiovascular, neurosensory, and psychiatric conditions and with the use of multiple medications [25]. For the patient, the symptoms can be debilitating. In patients older than 60 years, 20% have experienced dizziness severe enough to affect their daily activities [26]. Appropriate diagnosis and treatment can significantly improve quality of life. Most causes of dizziness are benign, but early recognition of serious or life-threatening disease is important. Management of these patients includes referral for neuroimaging and further evaluation in an emergency department.

References