EMERGENCY MEDICINE PRACTICE

AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

The Dizzy Patient: An Evidence-Based Diagnosis And Treatment Strategy

It's 3 a.m. in the ED—again. The patient in room 10 is 58 years old with no significant past medical history and a normal examination. But she's "dizzy." Do I get any blood work? Order a CT? She looks so healthy that this can't be something serious. Right?

WHETHER described as dizziness, lightheadedness, vertigo, or just "not feeling right," these are the all-too-common complaints that bring patients to the ED and emergency physicians to their knees. Upon picking up the chart of an elderly woman who is weak and dizzy, the emergency physician may long for a "simple" multiple trauma! The multitude of vague symptoms is matched only by the lengthy and sometimes arcane differential diagnoses. Since the underlying etiology may be life-threatening, the need for an organized and careful approach is essential. This issue of *Emergency Medicine Practice* will review the causes of "dizziness" and provide a basic framework for efficient and effective management.

Epidemiology

"Dizziness covers anything from severe aural vertigo to a housewife feeling nervous in the supermarket."
—Henry George Miller, 1968

When attempting to determine the prevalence of dizziness among ED patients, it is important to first determine what a particular patient means when he or she complains of being dizzy. Dizziness, while potentially representing true vertigo, may signify a host of other complaints including lightheadedness, near-syncope, weakness, disequilibrium, or an ill-defined disturbance in mentation.

Vertigo is characterized by a subjective sensation of rotation of the subject or of objects around the subject. It is often accompanied by a feeling of imbal-

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CME Objectives

Upon completing this article, you should be able to:

1. describe the most common causes of dizziness and how they may present;
2. discuss diagnostic modalities for patients with dizziness;
3. identify the treatment options for the most common causes of dizziness; and
4. identify those patients with life-threatening causes of dizziness.

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ance, nausea, and / or vomiting. Dizziness is a disorder of spatial orientation. Patients become uncertain of their position or motion.[1] When sensory organs distort information, or when different senses provide conflicting information, dizziness may occur. It also results from slow or incomplete integration of these signals by the brain. Near-syncope usually refers to a feeling of almost passing out. While the word "lightheaded" can mean almost anything to a patient, to a physician it is the ill-defined "catch-all" catagory of dizziness.

Although this may not be their primary complaint, as many as 25% of ED patients have some element of dizziness as part of their presentation.[2] Not often seen in children, the incidence of dizziness increases with advancing age. In elderly patients, that proportion can approach 40%, and *it is the most common complaint in patients over 75.*[3,4] A diagnosis can be made in the ED in about 80% of patients with dizziness. About half of patients whose chief complaint is dizziness have vestibular causes (both peripheral and central). Near-syncope, often due to hypovolemia, also accounts for a significant proportion.[5]

Vertigo and dizziness are not always benign—particularly in the elderly. Vertebrobasilar insufficiency (VBI) may present with isolated vertigo in up to one-fourth of patients.[6] In one study of cerebellar infarction, the case fatality rate was 23%, the highest for any area of brain infarction.[7] Even in the absence of a clear cerebrovascular cause, dizziness takes its toll in disability and injury, especially in falls sustained by elderly patients.[8] The ability to perform daily activities can be severely hampered by dizziness or unsteady gait.

The literature regarding the etiology of dizziness varies wildly. In one interesting review of dizziness among the elderly, vestibular disease was identified as a primary or contributing cause in 4%-64% of cases of dizziness, depending upon the study.[3] The authors note a wide disparity in other etiologies: Cerebrovascular causes were identified in 0%-70% of cases, psychiatric causes in 0%-40%, and cervical spondylosis in 0%-66%. In some studies, no diagnosis could be made in 8%-22% of cases, whereas multiple diagnoses were assigned in 0%-85% of cases.[3]

Differential Diagnosis

More than 60 disorders may result in the complaint of dizziness,[9] and approximately 12% of patients have multiple causes for their dizziness.[10] (See **Table 1**.) Fortunately, most patients with dizziness can be classified as having one of the following syndromes:[11]

- Near-syncope secondary to decreased blood flow to the brain (such as orthostatic hypotension, cardiac pre-syncope)
- True vertigo
- Disequilibrium (a sensation of imbalance when standing or walking—often secondary to multiple sensory deficits)
- Vague lightheadedness other than vertigo, pre-syncope, or disequilibrium. Patients may complain of "heavy-headedness" or "wooziness." This type of dizziness may occur with psychiatric disorders, hyperventilation syndrome, encephalopathies, and multisensory dizziness, as well as many other conditions not causing the first three types.[9]

Near-Syncope

The final common mechanism in near-syncopal dizziness is cerebral hypoperfusion. This may be due to a decrease in cardiac output, hypovolemia, or a failure of vasomotor tone.

Cardiovascular Causes

Decreased cerebral perfusion leading to these symptoms can occur as the result of a number of underlying disorders, including impaired cardiac output due to arrhythmia or structural cardiovascular disease, vasovagal episodes, hypovolemia, or orthostatic hypotension. In studies of syncope, vasovagal or vasodepressor syncope accounts for about 40% of all cases.[12-14]

The differential diagnosis for dizziness due to cardiovascular causes is essentially the same as that for syncope. Often, patients will relate a history of syncopal or near-syncopal events prior to the current episode. Although dizziness due to cardiac causes without syncope has not been extensively studied, true cardiac syncope is a serious entity whose one-year mortality rate has been estimated to be between 18% and 33%.[12-14] In a recent meta-analysis of 12 studies, near-syncope was the cause of dizziness in 6% of

Table 1. Differential Diagnosis Of Dizziness.

Cardiovascular Causes
Arrhythmias (fast or slow rate)
Orthostatic hypotension
Hypovolemia or anemia
Myocardial ischemia
Structural cardiac or valvular disease
Hypoxia
Vasovagal episode (also neurologic)

Neurologic-Otologic Causes
Peripheral vestibular causes:
- Benign paroxysmal positional vertigo
- Vestibular neuritis
- Ménière's disease

Central vestibular causes:
- CVA
- Vertebrobasilar ischemia
- Cerebellopontine angle mass
- Multiple sclerosis
- Basilar artery migraine

Other
Drug effects:
- Aminoglycosides
- Anticonvulsants
- Antihypertensives
- Hypoglycemics
- Antipsychotics
- Sedative/hypnotics
- Alcohol

Psychiatric (hyperventilation, anxiety)
Thyroid disorders

patients.[15] In one ED-based study, however, 16% of patients with dizziness were found to be near-syncopal.[5] It should be noted that near-syncope is much more likely to be due to volume depletion rather than to a primary cardiac cause.

Of the cardiovascular etiologies for dizziness, impaired cardiac output due to arrhythmia is probably the most concerning and the most life-threatening. Both tachyarrhythmias and bradyarrhythmias can lead to significant cerebral hypoperfusion and symptoms of dizziness. In Stokes-Adams attacks, high-degree atrioventricular block is associated with either syncope or near-syncope. If ventricular fibrillation is the cause, however, it is unlikely that dizziness will be the presenting complaint!

Structural cardiovascular disease, including valvular disease, cardiomyopathy, or other extracardiac vascular disease, can also lead to decrease in cardiac output and cerebral hypoperfusion.

Volume Depletion And Vasomotor Instability

Volume depletion is an important cause of dizziness secondary to near-syncope. It may be responsible for 75% of near-syncopal events.[15] Occult gastrointestinal bleeding can go undetected for months before finally presenting with signs of volume depletion and significant anemia. Certain medications, particularly in the elderly, may lead to orthostatic hypotension, including antihypertensives, anti-Parkinsonian drugs, neuroleptics, and anticholinergics. Poor conditioning and autonomic insufficiency are other causes of orthostatic hypotension in the elderly. Also note that elderly patients may become dizzy with postural change without blood pressure changes.[16,17] A recent study describes decreased cerebral oxygenation during postural change in the elderly.[18]

Vertiginous Disorders

Vertiginous disorders are generally separated into peripheral (semicircular canals and vestibular nerve) and central (brainstem and cerebellum). Among the more common of these are benign paroxysmal positional vertigo, Ménière's disease, and vestibular neuritis.[19] Conflicting signals from the visual, proprioceptive, and vestibular systems result in the sensation of vertigo. Asymmetric input to the vestibular nuclei via afferent nerves from the otolith organs and the semicircular canals of the inner ear can also trigger symptoms.[20]

Peripheral causes of vertigo are the most common[19]—they account for up to 85% of patients with vertigo, with central causes being present only 15% of the time.[21] In addition to a spinning-room sensation, patients with peripheral causes of vertigo often describe nausea, vomiting, and diaphoresis. Although these symptoms do not tend to be present in other causes of dizziness, rapid postural changes with associated orthostatic hypotension may elicit nausea, vomiting, and diaphoresis.[22] A vertiginous component in the latter case, however, will be notably absent.

Benign Paroxysmal Positional Vertigo

Of the causes of peripheral vertigo, benign paroxysmal positional vertigo (BPPV) is the most common, accounting for 16% of patients with dizziness.[15] The pathogenesis of this condition is thought to be due to the accumulation of free-floating particulate debris (specifically calcium carbonate crystals) within the endolymph of the posterior semicircular canal.[23] Intraoperative observation of this phenomenon supports this conclusion. The exact nature of the relationship between this particulate matter and the onset of vertigo is unclear. However, unilateral changes in endolymphatic pressure may be responsible, leading to asymmetric input to the vestibular nuclei.[23] A history of prior head trauma is linked to the onset of BPPV, presumably due to dislodged endolymphatic debris. This condition appears to be more common in women than men by a 2:1 ratio, usually occurring between the ages of 60 and 70.[20]

This syndrome, which typically lasts less than 30 seconds, is characterized by a rapid onset of vertigo symptoms after a change in head position. Often, patients will complain of the acute onset of symptoms after rolling over in bed, gazing upwards, or bending forward.[21] Torsional nystagmus, nausea, and/or vomiting are also prominent features.[20,22,24] Patients may experience disequilibrium on standing and walking, and they will often obtain relief upon lying still with their eyes closed.

Ménière's Disease

In addition to BPPV, peripheral vertigo may also be caused by other vestibular disorders, including Ménière's disease and vestibular neuritis/labyrinthitis. Pathologic findings in Ménière's disease include increased endolymph volume with distension of the endolymphatic system (endolymphatic hydrops). Presumably, the deleterious effects of this distension on hair cells lead to abnormal vestibular input with accompanying hearing loss.[20] On formal audiometry, unilateral low-frequency hearing loss is the most common finding.

Ménière's disease, like BPPV, may be associated with nausea, vomiting, and vertigo, but the duration of symptoms in Ménière's disease tends to be hours rather than seconds, as in BPPV.[25,26] It is significantly less common than BPPV, affecting just 5% of patients with dizziness in a recent critical review of the literature.[15] Its onset is most frequently in the fifth decade of life. Ménière's disease also differs from BPPV, as it is associated with a sensation of "fullness" in the affected ear, fluctuating sensorineural hearing loss, and tinnitus. Disequilibrium may also be present.

Vestibular Neuritis/Labyrinthitis

Vestibular neuritis is the most common term for an acute unilateral loss of peripheral vestibular function associated with vertigo, nausea, vomiting, spontaneous nystagmus, and disequilibrium.[25,27] The time course of symptoms tends to be over a period of days, with symptoms generally peaking during the first day, then gradually improving over the next few days. In a recent review of acute vestibular syndromes, Hotson and Baloh state that in otherwise healthy patients, this constellation of symptoms is generally due to a viral infection.[25] They concede, however, that fewer than half of affected patients have a preceding viral infection and that evidence for an inflammatory or infectious

etiology to this syndrome is not compelling. The term "neurolabyrinthitis" is used when acute vestibular symptoms are accompanied by hearing loss.[25] In their meta-analysis of 12 studies on dizziness, Kroenke et al report that vestibular neuritis or labyrinthitis affected 9% of patients presenting with dizziness.[15]

Post-traumatic Vestibular Syndromes

A rare cause of peripheral vertigo is perilymphatic fistula. This usually post-traumatic finding involves an abnormal connection between the middle and inner ear. It can be caused by a direct blow to the ear, a forceful Valsalva maneuver, or acute external pressure changes (as in scuba diving or descent in an airplane). Treatment consists of conservative therapy in the majority of patients, with surgical patching reserved for those with recurrent symptoms. Acute traumatic tympanic membrane rupture can produce immediate vertigo, nausea, and / or vomiting associated with hearing loss—a particularly disconcerting event when it occurs 100 feet below the surface. A similar constellation of symptoms is seen in patients with fractures through the petrous portion of the temporal bone.[28]

Central Vestibular Causes

Although peripheral causes of vertigo make up an estimated 85% of patients with vertigo, central causes include disorders with significant potential morbidity. These include vertebrobasilar insufficiency, basilar artery migraine, and infarcts or hemorrhage of the cerebellum and brainstem. Like patients with peripheral vertigo, patients with central vertigo tend to complain of severe imbalance. However, unlike their "peripheral" counterparts, they have little nausea. Rarely will they have any auditory symptoms.They have difficulty compensating for their vertigo, which is often accompanied by other neurologic complaints.[29,30] Symptoms associated with brainstem ischemia include diplopia, ataxia, dysarthria, and facial weakness.[31] The symptoms are often insidious in their progression.[22]

Infarction or hemorrhage in the inferior cerebellum may represent a neurosurgical emergency due to the potential for rapidly increasing mass effect in the posterior fossa. Blood supply to both the vestibular nuclei in the brainstem as well as to the cerebellum comes from the vertebrobasilar system. Insults to this area may present with prominent vertigo symptoms.[32] (See **Table 2** on page 5.)

Other Neurologic Causes

In addition to vertebrobasilar insufficiency and cerebellar ischemia / hemorrhage, multiple sclerosis (MS), basilar artery migraine, and cerebellopontine angle (CPA) tumor are less frequent causes of vertigo. Vertigo is the initial manifestation of multiple sclerosis in 10% of patients and is present at some point in up to one-third of those with MS.[33] Basilar artery migraine can produce severe occipital headache, vertigo, and temporal lobe seizures.[31] Those with CPA tumors (usually acoustic neuromas) often complain of vertigo accompanied by hearing loss. In these patients, the slow hearing loss tends to be more prominent than vertigo, as the brain compensates for prolonged asymmetric vestibular input.[25,31]

Other Medical Causes Of Dizziness

In addition to cardiovascular, vestibular, and CNS causes of dizziness, several other clinical entities may require consideration. Among these are drug toxicity, hypoglycemia, anemia, and hypothyroidism.[22] Aminoglycosides produce vertigo and disequilibrium through direct ototoxic effects on vestibular hair cells, as can cisplatin and certain other chemotherapeutic agents.[29] Symptoms tend to be bilateral, often leading to disequilibrium rather than vertigo. Oscillopsia, the subjective sensation of oscillation of vision, occurs with bilateral vestibular dysfunction. It may be idiopathic or due to otologic or neurologic disease (such as cerebellar degeneration), autoimmune conditions, toxins, or neoplasms.[34]

Anticonvulsant toxicity, particularly involving phenytoin or carbamazepine, may cause CNS depression, nystagmus, and ataxia as well as dizziness. Phenytoin may also produce mild nystagmus at therapeutic concentrations.[35] Benzodiazepines, barbiturates, alcohol, and other CNS depressants may create a globally depressed mental status that may present as nonspecific dizziness.[29]

Symptomatic hypoglycemia may also result in nonspecific dizziness or lightheadedness associated with fatigue, palpitations, and nausea. More severe hypoglycemia can be associated with altered mental status, lethargy, seizures, and diaphoresis. In Kroenke et al's meta-analysis of patients presenting with dizziness, 13% had dizziness thought to be metabolic in nature, including drug reactions, anemia, hypoglycemia, or thyroid disease.[15]

Disequilibrium

Disequilibrium is a feeling of imbalance when standing or walking and may be due to numerous causes. Sensory deficits may result in this condition even in the absence of vestibular dysfunction. Patients usually deny any abnormal head sensation and may refer to their condition as "dizziness in the feet."[9] Such patients may have impaired motor control. Physical examination may reveal decreased visual acuity, peripheral neuropathy (loss of proprioception), and abnormal vestibular function.

Vague Lightheadedness

This category of dizziness provides the greatest diagnostic frustration to the physician. It may have medical or psychiatric etiologies. Psychiatric causes of dizziness, including anxiety and hyperventilation syndromes, may account for up to 16% of patients presenting with dizziness.[15] It may be more frequent in patients with major depression, anxiety, or somatization disorders. Hyperventilation may be associated with lightheadedness, perioral and peripheral numbness, tingling, and a sensation of intense anxiety. Patients with a history of similar episodes or previously diagnosed panic disorder may become hypervigilant to the onset of dizziness symptoms and precipitate recurrent attacks.[31]

Psychogenic dizziness is a diagnosis of exclusion. Take care to ascertain whether an underlying organic disorder is

present, as patients with significant medical problems may also present with hyperventilation. Remember, psychiatric illness provides little protection against medical disease.

Pre-hospital Care

There is scant literature regarding the pre-hospital care of the dizzy patient. Management will depend primarily upon the clinical picture, especially the vital signs. The dizzy patient with tachycardia and hypotension may require intravenous fluids and rapid transport.

ED Evaluation

As with any ED patient, the initial approach to the dizzy patient should include the ABCs (airway, breathing, and circulation). Patients with any evidence of hemodynamic compromise or altered mental status should be urgently triaged and rapidly stabilized by the emergency physician. The next important task is to understand what type of event has occurred. Sometimes the answer is obvious, such as the young person who faints in a hot, crowded room or a diabetic whose glucose is very low. Frequently, though, the understanding of the event requires further questioning.

"Study history, study history. In history lies all the secrets."
—Winston Churchill

History

The presence (or absence) of key elements of the patient's history will be essential in helping the emergency physician pinpoint the cause of the patient's symptoms. Two office-based studies found that the etiology of dizziness could be made using history alone in 69%-72% of patients.[36,37] (See **Table 3** on page 6.)

Description Of Symptoms

It is important to understand what the *patient* means by the word "dizzy." No offense, but what you mean by this word is immaterial. In some cultures, the word "dizzy" really

Table 2. Peripheral Vertigo vs. Central Vertigo.

Finding	Peripheral	Central
Nausea and v omiting	Severe	Moderate
Nystagmus		
• Type	Horizontal-rotary or rotary; never vertical	Any direction (may be purely vertical)
• Direction (refers to fast component)	Unidirectional (fast component in same direction regardless of direction of gaze)	May be bidirectional (changes direction depending upon direction of gaze)
• Fixation	Suppressed with fixation	Not suppressed with fixation
• Fatigable	Nystagmus is less intense upon repeated testing of lateral gaze	Nystagmus remains just as prominent despite numerous trials of lateral gaze
• Intensity	Increases when the gaze is in the direction of the fast phase, and decreases when the gaze is away from the fast phase (Alexander's law)	May have no change in intensity based on gaze
Gait		
• Imbalance	Mild	Severe (often unable to walk)
• Falling or veering	To side opposite fast component	Falls or veers to either side
Romberg testing	Falls to the side opposite to the direction of the fast phase of nystagmus	Falls to either side
Hearing loss	Common	Rare
Associated neurologic sympt oms	None	Common (cranial-nerve signs, motor weakness, prominent dysmetria, sensory changes, or abnormal reflexes)
Dix-Hallpike maneuv er		
• Nystagmus	Rotatory nystagmus toward the dependent ear; diminishes within 50 seconds of onset	Nystagmus of varying patterns; may persist
• Latency	Symptoms begin 1-5 seconds or longer after maneuver	Symptoms may begin immediately upon maneuver
• Extinction	Extinction of nystagmus and vertigo after several trials	No extinction

means "sick" or "not well." Because the word "dizziness" is a catch-all term, use open-ended questions such as "Can you describe this dizziness?" as a means of obtaining further details.[22] Avoid leading questions, and allow the patient to describe the events in his or her own terms.

- **Vertigo:** *Webster's Dictionary* defines vertigo as a disturbance "in which the external world seems to revolve around the individual or in which the individual seems to revolve in space." Determine whether there is a sensation of movement—either that the patient or the room is moving. The illusion of movement or spinning is highly associated with vestibular causes.
- **Weakness:** If the complaint is "weakness," determine what the patient means by this. Does the patient mean global fatigue, lack of energy, or inability to ambulate? Did the patient suffer focal weakness, such as an isolated extremity paresis?
- **Syncope:** There are wide variations in regional dialects and the descriptions of dizziness. Be prepared to hear a range of terms, like "fixin' to faint" to describe near-syncope, and "falling out" to describe syncope.

If the complaint is, "I passed out," the emergency physician needs to ascertain whether this was true syncope (did the patient hit the floor?) or whether this was a near-syncopal event. Ask whether a prodrome was present, such as palpitations, diaphoresis, nausea, visual changes or headache, abdominal pain, or other triggering event. If there was true loss of consciousness, were there any associated symptoms (witnessed seizure, loss of bladder / bowel control, head or neck trauma)?

What Were The Circumstances Of The Event?

Determine whether the symptoms occurred during a change in position. Dizziness that occurred only upon standing may reflect vertigo, decreased cerebral perfusion, or disequilibrium. If the patient's symptoms are consistently present after arising from a supine or sitting position, orthostatic hypotension (and underlying causes of hypovolemia) may be the culprit. Symptoms that occur with head turning, lying down, or rolling over in bed are more consistent with vertigo.[11]

Ask the patient whether symptoms are induced by exercise, related to environmental factors (e.g., recent head trauma or heat exposure), or whether they only occur in specific situations (e.g., induced by anxiety or emotional stressors). Determine any recent change in medication. A new antihypertensive medication, anxiolytic, anticonvulsant, or neuroleptic may induce a poorly defined feeling of disequilibrium, lightheadedness, or altered sensorium.[31,38] A new antihypertensive agent may cause profound orthostasis. Drug use—especially alcohol or benzodiazepines—may also produce this type of sensation.

If the patient presents in the early fall wearing a red vest and one leather glove, the diagnosis is obvious. "Bow hunter's stroke" consists of repeated vertebrobasilar ischemic attacks induced by head rotation 45° to the left, as when shooting an arrow.[39] Other causes include chiropractic manipulation, yoga, and cervical trauma. Even a vigorous shampoo in hyperextended neck position can produce cervical vertigo[40]—providing a compelling argument for washing your own hair.

Table 3. The Dizzy Patient: Essential Elements Of The History.

- Is true vertigo present?
- What is the pattern of onset?
- What is the duration of the symptoms?
- Have there been auditory symptoms?
- Are there associated neurologic symptoms?
- Are there other associated symptoms?
- What is the patient's past medical history?
- What medications is the patient taking?

How Old Is The Patient?

The patient's age can narrow the differential. Young people are more likely to have vestibular disorders, hyperventilation, multiple sclerosis, and panic attacks. While the elderly are also susceptible to these conditions, they are more likely than the young to suffer stroke, multisensory dizziness, and neurodegenerative diseases including Parkinsonism.[9]

Associated Symptoms

The associated symptoms may narrow the differential to an offending organ. Generalized symptoms such as nausea and vomiting are nonspecific. These complaints, along with ataxia, may occur with both benign and serious causes of vertigo.

- **Neurologic:** The posterior fossa is a small place. Insults to this area are likely to cause "neighborhood" problems, not just isolated vertigo. The five "Ds" of posterior circulation problems are: dizziness, diplopia, dysarthria, dysphagia, and dystaxia. Ask posterior fossa questions such as: "Did you have difficulty speaking or double vision?" Altered mental status in the presence of dizziness is ominous, as are thunderclap headaches and focal neurologic deficits.[22] Any of these may presage a neurologic disaster. Fever and headache with dizziness raises the possibility of infection, including mastoiditis, suppurative labyrinthitis, or meningitis.
- **Ear Complaints:** Some patients with cochlear disease may complain of fullness in their ear. Unilateral hearing loss or tinnitus (a ringing or roaring sensation in the ear) points to a vestibular cause, while progressive hearing loss may represent a mass effect, such as by a cerebellopontine angle tumor. Vertigo associated with acute hearing loss, with or without tinnitus, suggests Ménière's disease, impacted cerumen, or eardrum rupture.
- **Cardiovascular Symptoms:** Palpitations, chest pain, and / or dyspnea may accompany dizziness and can implicate a cardiac cause. If the patient gives a history of near-syncope or syncope related to exertion, consider a structural heart disease, such as aortic stenosis or hypertrophic cardiomyopathy.
- **Orthostatic Symptoms:** Patients with orthostatic

symptoms may complain of dim vision ("gray out") or roaring in their ears upon standing or sitting up. While this may occur with arrhythmias, most symptoms are related to volume depletion. Ask about a history of vomiting, diarrhea, or tarry stools. Vasovagal symptoms can account for near-syncopal and syncopal symptoms as well. These episodes are often brought on by emotional stress, pain, hot, crowded settings, or prolonged standing, as in soldiers standing at attention for extended periods. Onset while standing or sitting and a prodrome of lightheadedness, diaphoresis, nausea, and / or vomiting are typical. Patients recover when placed in a supine position.

Time Course

Having the patient define the approximate time of symptom onset is helpful in determining the probable etiology. A patient with a lengthy time course of generalized weakness that's accompanied by intermittent palpitations and near-syncope paints a markedly different picture than one with acute onset of vertigo, nausea, and vomiting lasting only seconds.

Vertigo is typically episodic—worse at onset, then gradually resolving. The time course for various causes of vertigo is helpful for diagnosis: A time course of seconds implies BPPV; minutes, cerebrovascular ischemia (e.g., VBI or TIA); hours, Ménière's disease; and days, vestibular neuritis / labyrinthitis.[22]

Near-syncope or lightheadedness due to orthostatic hypotension or hypovolemia tends to resolve on return to the supine or sitting position but may persist if the patient remains erect for an extended period. If symptoms are secondary to arrhythmia, the onset is typically abrupt, and the time course may vary from only seconds to hours or even days in duration.

Past Medical History

The past is a great predictor of the future. Ask whether the patient has ever had a similar event in the past; if so, how often? (And better yet, what was the diagnosis?) The past medical history may have bearing on the current complaint in other ways. Patients with a history of seizures may be suffering from phenytoin or carbamazepine toxicity. A history of valvular disease, valve replacement, or atrial fibrillation increases the possibility of emboli-related symptoms.

If you detect florid nystagmus on physical examination, ask about a prior history of nystagmus. Congenital and sensory-deficit blindness is not uncommon. Nystagmus in an albino is most likely congenital.[41]

Physical Examination

"Ignorance more frequently
begets confidence than does knowledge."
—Charles Darwin, The Descent of Man, 1871

Vital Signs

Consider the following in assessing the vital signs in patients who are dizzy:

- **Blood Pressure:** The presence of hypertension in patients complaining of dizziness—especially in patients with a long-standing history of hypertension—should raise the question of vertebrobasilar insufficiency and / or cerebellar infarction / hemorrhage. Vertigo accompanied by hypertension is more worrisome, as vertebrobasilar disorders may present with isolated vertigo.[29] Hypotension suggests symptoms that may be related to decreased cerebral perfusion.
- **Heart Rate:** Significant tachycardia (usually > 150 bpm) or bradycardia (< 40 bpm) may impair cardiac output and cerebral perfusion, causing dizziness, near-syncope, or syncope.[22]
- **Respiratory Rate:** Hyperventilation, usually related to anxiety, may lead to dizziness, near-syncope, or syncope. It should be noted, however, that some patients with recurrent vertigo may develop anxiety, panic attacks, and hyperventilation in response to repeated episodes of peripheral vertigo.[42]
- **Temperature:** Fever alone, as previously mentioned, not only may produce a sensation of dizziness but also may accompany CNS or other infections.

General Appearance

In the first few moments of the patient encounter, assess his or her general appearance. Patients who appear pale or dehydrated may have an orthostatic component to their complaint. The patient who is dizzy and diaphoretic may harbor serious cardiac pathology.

Targeted Examination

In addition to the vital signs, good head and neck, cardiovascular, and neurologic exams are essential parts of the evaluation.

Eye Exam

Check for the presence of nystagmus. When first testing for nystagmus, it may be helpful to ask patients to look to their right and then to their left, *without* having them track the examiner's finger. Following the finger causes ocular fixation, which may extinguish the nystagmus. Note the *direction* of the nystagmus—that is, the direction of the fast component. In cases of vestibular disease, the fast component points away from the side of the lesion.[25] *In peripheral vertigo, spontaneous nystagmus continues in only one direction even when the direction of gaze changes.* Also note the nature of nystagmus—horizontal, rotary, horizontal-rotary, vertical, or vertical-rotary. Peripheral vestibular nystagmus is typically horizontal-rotary with a slow and fast component. It typically extinguishes with repeated testing or ocular fixation.[25,31]

In central disorders, such as infarction or hemorrhage of the brainstem or the cerebellum, *the spontaneous nystagmus may change its direction whenever there is a change in the direction of gaze (gaze-evoked nystagmus).*[25] Occasionally, in cerebellar stroke, nystagmus occurs only when the patient is gazing in one direction, thus mimicking peripheral disease.

Other clues to central vertigo include spontaneous, non-fatigable vertical nystagmus.[43] Vertical nystagmus is

due to a central neurologic cause until proven otherwise.

Certain medications can produce nystagmus, including phenytoin, alcohol, ketamine, and phencyclidine. Vertical nystagmus is rarely seen as a drug-induced phenomenon except in the case of phencyclidine intoxication. The presence of nystagmus at extreme end-gaze is not pathologic and is seen in up to 60% of normal people.[44]

Ear Examination

Examine the tympanic membranes and external auditory canals (EAC) for the presence of infection, tympanic membrane rupture, impacted cerumen, or foreign body.[45] The presence of vesicles within the EAC, in association with ipsilateral facial nerve palsy, should suggest herpes zoster oticus (Ramsay Hunt's syndrome).[20] While acute otitis media rarely causes vertigo,[42] it may on occasion be the culprit. Replication of vertigo with pneumatic otoscopy is diagnostic of post-traumatic labyrinthitis.

Test the patient's hearing. This can be done with a tuning fork (if one can be found in the ED), rubbing your fingers together, or even activating your beeper by each of the patient's ears. The presence of recent unilateral hearing loss in the setting of vestibular symptoms suggests Ménière's disease. Acoustic neuromas, due to their slow growth, typically present with a gradual decline in hearing and are rarely accompanied by symptoms of vestibulopathy.[42,46]

Cardiovascular Exam

Auscultate the carotid arteries for the presence of bruits. High-grade carotid stenosis may produce cerebral hypoperfusion with resultant near-syncope or syncope, especially if it's associated with exertion, even if only mild. Carotid disease, however, is unlikely to cause true vertigo.

The presence of the midsystolic murmur of aortic stenosis in the patient complaining of near-syncope or syncope may be diagnostic.

Neurologic Exam

Begin with a thorough cranial nerve exam, including evaluation of cranial nerves and cerebellar function using finger-to-nose and rapid alternating movement tests. Involvement of other cranial nerves in addition to the vestibulocochlear nerve strongly suggests central disease. Whenever possible, evaluate the patient's ability to ambulate. Patients with peripheral vertigo are typically able to walk without assistance, although they tend to veer to one side. The opposite tends to be true in patients with acute cerebellar infarction or hemorrhage, where walking without falling may be impossible.[25] Romberg testing in patients with cerebellar infarction may be variable. Patients with peripheral vestibular disorders, however, tend to lean or fall to the opposite direction of the fast component of nystagmus (i.e., toward the lesion).[25]

The presence of altered mental status, headache, or focal neurologic deficits should prompt further investigation, but a non-focal neurologic exam in a vertiginous patient with significant cerebrovascular disease risk factors does not exclude potentially serious pathology such as vertibrobasilar insufficiency.

Diagnostic Testing

There is little evidence-based literature on the evaluation of dizziness—either in ED or office-based settings. Practice patterns vary widely, leading to multiple competing theories regarding which tests are most valuable. In this section, we examine the clinical utility of various diagnostic tools in the management of dizzy patients, including provocative maneuvers, laboratory tests, imaging studies, and more sophisticated neurologic testing.

Not every test is appropriate for all patients with dizziness, however. Dividing dizzy patients into those with clearly defined vertigo and those without will focus the diagnostic evaluation.

Bedside Maneuvers

Orthostatic Vital Signs

Orthostatic hypotension, usually defined as a drop in systolic blood pressure of 20 mmHg or more within two minutes of standing upright, may account for near-syncope or syncope.[5,15,22] Some researchers believe that the change in *mean* blood pressure (not the systolic pressure), either immediately or at two minutes, is more reflective of true orthostasis, as this measure better correlates with cerebral perfusion.[3]

Recognize that orthostatic changes are not especially sensitive. Several studies have found that elderly patients become dizzy with postural change in the absence of orthostatic vital signs changes, which suggests that orthostatic vital signs in the evaluation of dizziness are only moderately sensitive at best.[16,17] They aren't necessarily specific, either. Dizziness with postural changes is common in the elderly.[47] Orthostatic hypotension occurs in 20%-30% of elderly patients not confined to nursing homes.[48] For these reasons, the finding of orthostatic hypotension should not dissuade the emergency physician from pursuing other causes of syncope.

In one meta-analysis of 12 studies, orthostatic changes were found in only 5% of dizzy patients.[19] In an ED-based study included in this meta-analysis, however, 16% of patients were found to be near-syncopal.[5]

Despite the drawbacks of orthostatic vital signs, they may be useful in the near-syncopal patient. If the patient is significantly orthostatic (and symptomatic), look for causes such as volume depletion (including active internal bleeding), drug effects, and other etiologies such as deconditioning and autonomic insufficiency.

Dix-Hallpike Maneuver

The diagnosis of BPPV is generally made from the history. However, certain findings on the physical exam will confirm the diagnosis. These findings were first described by Dix and Hallpike in 1952.[49] They include:

1. nystagmus, as described;
2. provocative head position;
3. brief latency to symptoms after change in position;

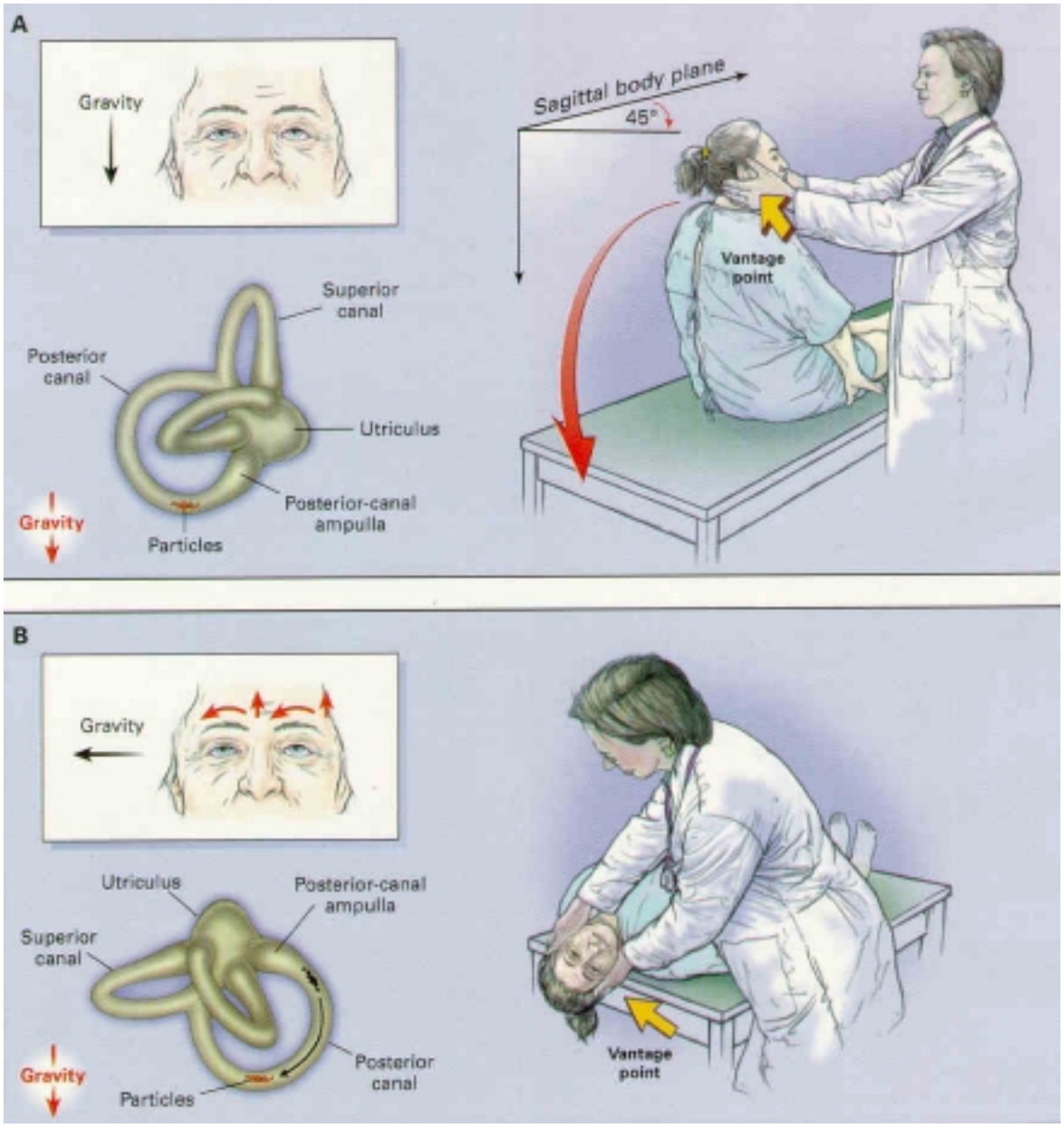
4. short duration of attack;
5. fatigability of nystagmus on repeat testing; and
6. reversal of nystagmus on returning to an upright position.

They developed a provocative maneuver to elicit symptoms. This test, the Dix-Hallpike maneuver (sometimes called the Nylen-Bárány, Bárány, Nylen, or Hallpike maneuver), involves moving the patient rapidly from sitting to a position of head hanging with one ear downward, and then repeating the test with the other ear. (See **Figure 1**.) When positive, the patient will, after a 1- to 5-second latency period (or longer), complain of a sensation of

Continued on page 12

Figure 1. The Dix-Hallpike Test.

This figure depicts the Dix-Hallpike test of a patient with benign paroxysmal positional vertigo affecting the right ear. In Panel A, the examiner stands at the patient's right side and rotates the patient's head 45° to the right to align the right posterior semicircular canal with the sagittal plane of the body. In Panel B, the examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The arrows in the inset depict the direction of nystagmus in patients with typical benign paroxysmal positional vertigo. The presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown.



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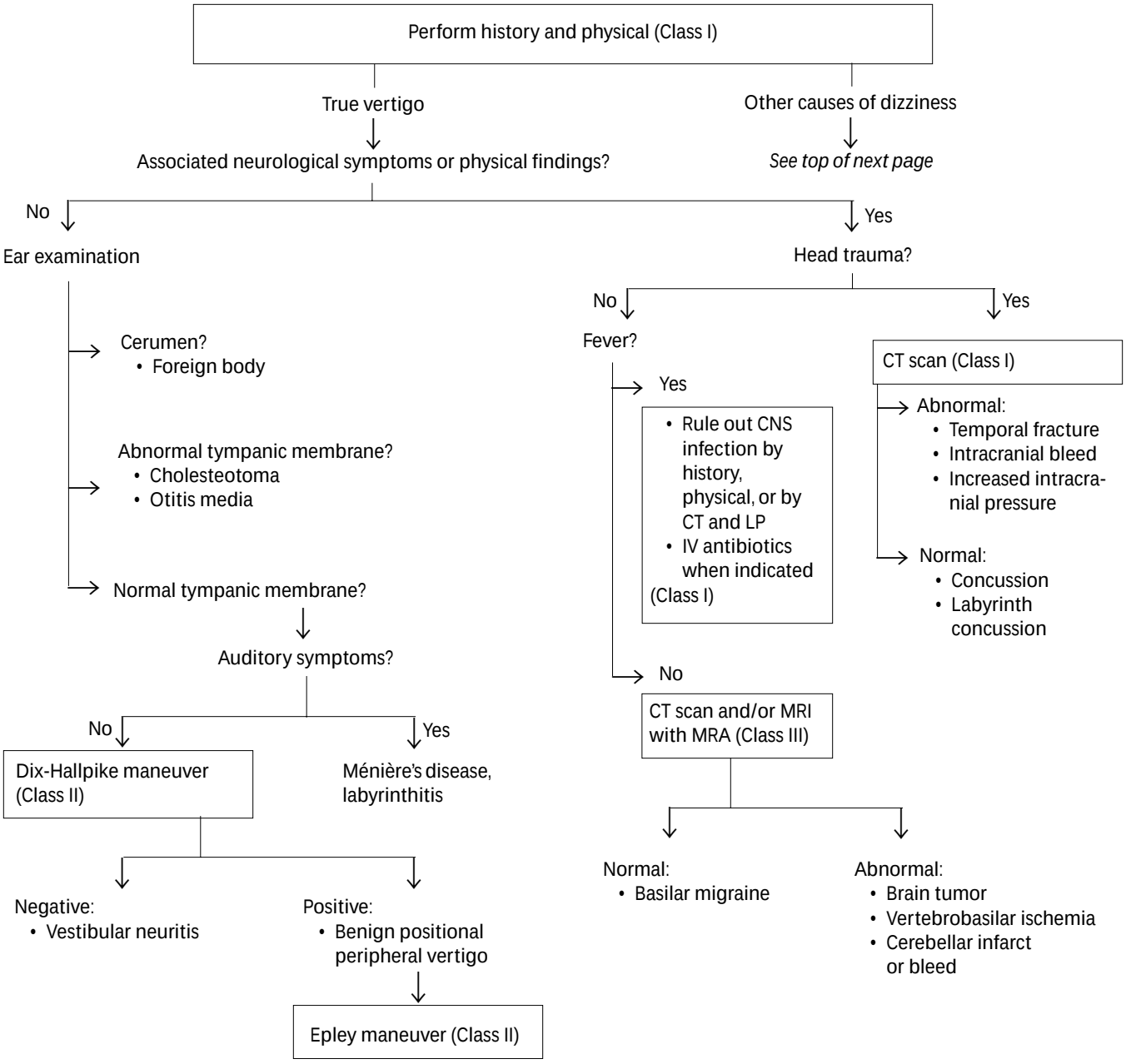
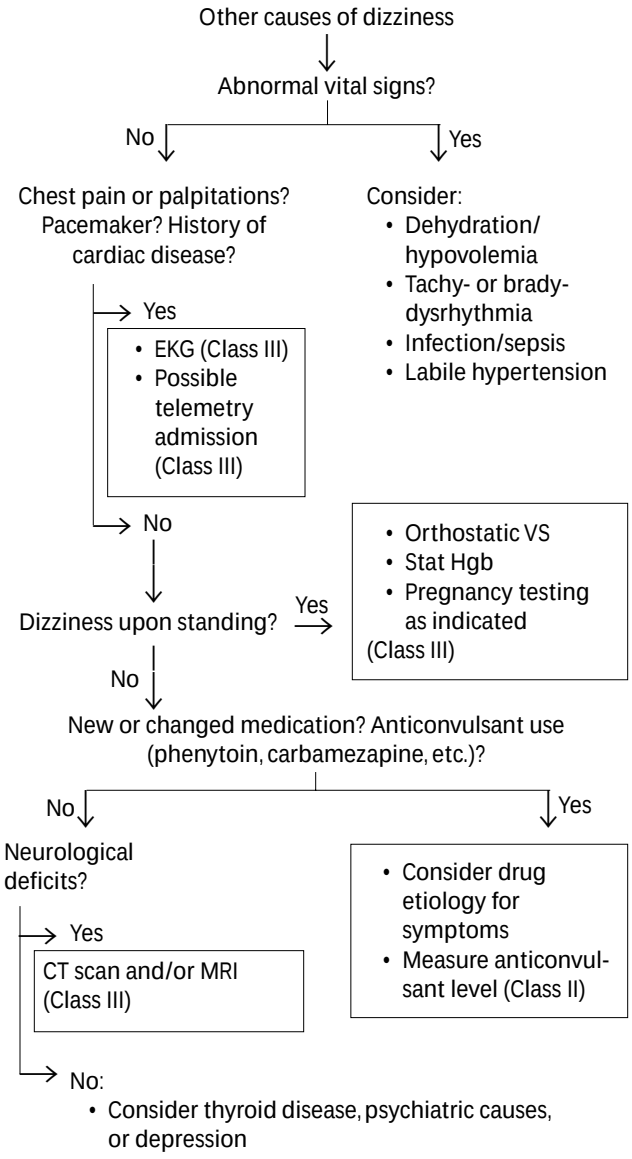
Clinical Pathway: Evaluation Of The Dizzy Patient



The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Clinical Pathway: Evaluation Of The Dizzy Patient (continued)



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Cost-Effective Strategies For Managing Dizzy Patients

1. The history is free.

A careful history can save a lot of diagnostic frustration. If the patient provides you with a rambling, incoherent narrative, direct the questions more narrowly. When did the symptoms start? What exactly did you feel? Was there chest pain, shortness of breath, or palpitations? Look for the key words that will suggest one diagnosis over another. If a patient says, "I was so dizzy, I thought I was going to fall," let that statement lead the discussion to the specific areas of interest: near-syncope, vertigo, disequilibrium, confusion, or anxiety.

2. Use your physical exam skills.

In general, patients with dizziness look fairly benign, and they usually are. If we see a patient as "alert and oriented, nonfocal, physical exam unremarkable," the desperate among us immediately proceed to "lab 'em up," hoping that some abnormal value will pop up. Snap out of it. Instead, learn to use your physical exam and diagnostic maneuvers to eliminate possible causes of dizziness. The Dix-Hallpike maneuver, in conjunction with a meticulous neurological examination, will be more fruitful than indiscriminate neuroimaging.

3. The ECG is your friend.

Nearly as cheap as the paper on which it's printed, the ECG provides a lot of information in a readily available, cost-effective package. True, most of the time, it will be normal, but for picking the patients with unrecognized rhythm disturbances out of the rest, the ECG is an excellent tool that supplies easily accessible, inexpensive data. But be sure to recognize its limitations as well: It only provides 12 seconds of information, and its greatest yield will be in patients with a history of cardiac disease or prior arrhythmia. It is unlikely to be helpful in the patient who complains of vertigo.

4. Save neuroimaging for those who need it.

The yield for CT scanning in dizzy patients with a nonfocal neurologic examination is very low. In the absence of significant risk factors for ischemic cerebrovascular disease or focal neurologic findings, the patient's symptoms are unlikely to be clarified by CT scanning or MRI. Gearing your evaluation toward peripheral vestibular disorders or non-vestibular complaints such as hemodynamic or metabolic abnormalities will save you and the patient time and money. A Dilantin level is a lot less expensive than a CT scan, and more apropos in the patient with a concomitant seizure disorder. On the other hand, if the patient's condition warrants neuroimaging, then order the test. ▲

Continued from page 9

rotational vertigo, accompanied by nystagmus. The nystagmus tends to be vertical and rotatory, "the upper pole of the eye beating toward the dependent ear and the vertical nystagmus beating toward the forehead."[23] While vertical nystagmus is "bad" (indicating a central lesion) if it occurs during gaze testing, vertical nystagmus is "okay"(indicating a peripheral lesion) if it occurs in the context of the Dix-Hallpike maneuver.

The vertigo and nystagmus resolve within 50 seconds of onset.[50] Nystagmus may recur after the patient returns to the seated position, but this time its direction is reversed. If the nystagmus induced by the Dix-Hallpike maneuver does not fit this description, the patient may have central vertigo.

The Dix-Hallpike maneuver was positive in 7%-44% of patients complaining of dizziness. In patients with BPPV, however, the sensitivity of the maneuver increased to 50%-88%, suggesting that this maneuver is likely to be useful only in patients who complain of vertigo.[19] Repetition of the maneuver leads to a reduction in the intensity of vestibular symptoms.[23,27] The patient who displayed such prominent vertigo and nystagmus after the initial maneuver may have a much less impressive response when you return with your colleagues to demonstrate your findings!

Tests For Perilymphatic Fistula (Fistula Tests)

Replication of the patient's symptoms on pneumatic otoscopy (Hennebert sign), combined with the typical history is diagnostic of perilymphatic fistula. The physician can also forcefully press on the tragus of the patient's ear, thus generating a pressure wave that ultimately impacts the inner ear. Patients with a fistula will develop vertigo, nausea, and nystagmus with this maneuver. However, otosyphilis and Ménière's disease can produce a false-positive fistula test.[51,52]

Head Shake

Nystagmus after rapid head-shaking is often seen in patients with vestibulopathy.[53]

Hyperventilation

The hyperventilation challenge is sometimes used when the physician suspects a psychiatric etiology for dizziness. The physician asks the patient to hyperventilate for two minutes to see if this exactly reproduces the presenting complaint. The physician can initially hyperventilate along with the patient to encourage the desired rate and depth of breathing.[54]

The utility of this test remains unclear. In a prospective study of 100 ambulatory patients with a chief complaint of dizziness, hyperventilation reproduced the offending symptoms in 21; however, the final diagnosis was hyperventilation syndrome in only one.[36]

Other Maneuvers

Several other maneuvers may occasionally be helpful in the patient with vertigo. Carotid sinus massage that produces transient asystole and near-syncope may indicate cardioinhibitory disease. However, it is not clear that a positive test confirms the etiology of dizziness. Barany rotation involves quickly spinning a patient in a chair with his or her head tilted down 30˚. Reproduction of the patient's symptoms may indicate that he or she is suffering from some type of vertigo. This test may be dangerous on an ED stool (potentially adding a traumatic injury to an insult), and its value in the emergency setting remains untested.

Laboratory Testing

Routine laboratory testing, including complete blood count, electrolytes, glucose, and creatinine levels, rarely helps in the evaluation of dizziness.[19] In patients with symptoms consistent with hypoglycemia, or those with evidence of significant anemia or occult blood loss, fingerstick glucose or hematocrit levels may be useful. The utility of routine pregnancy testing in female patients with dizziness has not been explicitly studied but should be considered.

Cardiovascular Testing

When evaluating the dizzy patient who complains of near-syncope, the ECG can be your best friend (although that probably means you don't get out enough). Even though a single 12-lead ECG in the ED is unlikely to provide a definitive diagnosis, a small proportion of dizzy patients (1%-5%) will have an arrhythmia causing their symptoms.[15] It is unlikely to be helpful in patients with true vertigo, but it may provide some answers in the patient with near-syncope. Given its accessibility, it is reasonable to obtain an ECG on dizzy patients who are elderly or have known cardiovascular disease. For patients with suspected rhythm disturbances, continuous ECG monitoring in the ED might be helpful, but its diagnostic yield is not well-studied.

When evaluating the ECG, look for rapid or slow rates as well as for prolongation of the QT interval. A wide QRS complex with a slurred upstroke in association with a short PR interval may indicate Wolff-Parkinson-White syndrome. Myocardial ischemia can also manifest itself as near-syncope or syncope, especially if it is associated with arrhythmia. Certain high-risk patients with an abnormal ECG or worrisome history may require more extensive cardiovascular testing, such as Holter monitoring, carotid Dopplers, or echocardiography, on an outpatient basis.

Neuroimaging

Certain high-risk patients who present with dizziness clearly require urgent neuroimaging based on the likelihood of a treatable insult such as ischemic stroke or hemorrhage. Such patients include those with a thunderclap or severe headache and those with hard neurological findings—particularly those referable to the brainstem or cerebellum. These findings may include motor deficits, particularly crossed hemiplegia (ipsilateral cranial nerve, contralateral extremity motor); dysarthria or dysphagia (with asymmetric movement of the palate); inability to walk; bidirectional or vertical nystagmus; and signs of cerebellar dysfunction. Gait ataxia alone (particularly when the patient consistently veers to the side opposite the fast phase of the nystagmus) is not an indication for neuroimaging.

The low-risk patient with no or few cerebrovascular

risk factors and physical examination compatible with peripheral vertigo generally does not require neuroimaging. The problem arises when the patient has several risk factors for cerebrovascular disease and possibly an equivocal physical examination. One ED-based study examined 24 vertiginous patients with risk factors for stroke who presented with symptoms of greater than 48 hours' duration and no neurologic abnormality apart from nystagmus. In this group of elderly patients who received neuroimaging, one-quarter were found to have inferior cerebellar infarction.[55] Although isolated vestibular nuclei ischemia can mimic peripheral vertigo, a cerebrovascular event affecting this area often presents with other neurologic symptoms as well, including diplopia, dysarthria, and/or focal motor or sensory deficits.[25] Remember, the brainstem is a small neighborhood; any "ruckus" is bound to be noticed on a good neurologic exam.

Other retrospective studies evaluating patients with prolonged vertigo symptoms with no other neurologic deficits have found evidence of vertebrobasilar insufficiency by MR angiography, with the greatest incidence in patients in their eighth decade of life.[55-59] However, there is considerable debate as to whether it is important to make the diagnosis of vertebrobasilar ischemia (VBI) in the ED. While symptoms of VBI may be the first warning signs of a future brainstem stroke, it is less clear that urgent diagnosis improves outcomes. There is no convincing evidence that admission for a vertebrobasilar TIA will have any effect on preventing future stroke. In addition, despite this habit among some neurologists, there are no data to support the use of heparin in the treatment of posterior circulation TIAs. (See also the July 1999 issue of *Emergency Medicine Practice*, "Code Stroke: A State-Of-The-Art Strategy For Rapid Assessment And Treatment.") The only proven intervention useful in the acute treatment of VBI is placing the patient on aspirin (or other antiplatelet agent)—an intercession that can be performed without an MR angiogram.

Two studies that evaluated neuroimaging in patients with dizziness studied selected populations referred for suspicion of CNS processes. These patients had either a clinical examination suggestive of a central process or were referred to a tertiary care center for high-resolution MR of the inner ear and cerebellopontine angle (CPA). Both studies found a high incidence of abnormalities, including vertebrobasilar disease, schwannomas, and one meningioma in this highly selected population.[60,61] In an unselected ED population, however, routine neuroimaging—either CT or MRI—is unlikely to be cost-effective. In a probability study of patients with dizziness and otologic symptoms being evaluated for CPA mass, one group of investigators concluded that 2500 imaging studies would have to be done to detect one CPA mass.[62]

If neuroimaging is deemed necessary for the evaluation of the dizzy patient, the choice of CT or MRI will be governed by the clinical entity being sought and by availability. For suspicion of vertebrobasilar disease and cerebellar ischemia, magnetic resonance brain imaging and angiography are the most desirable tests.[25] Computed tomography is more sensitive for hemorrhage, but MRI is more likely to detect subtle brainstem or inferior cerebellar infarction. CT is also more available than MRI. While MR angiography is useful to diagnose vertebrobasilar occlusive disease, the optimal timing of this study remains in question.

It is important to recognize the limitations of CT scanning for the evaluation of TIA, infarction, and the posterior fossa. Even with a negative head CT, the patient with vertigo and a worrisome neurologic examination needs an urgent neurologic or neurosurgical consultation.

Specialized Neurologic Testing

Beyond the information provided by physical exam, routine laboratory testing, or even neuroimaging, more specific neurologic tests have been used to evaluate patients complaining of dizziness. Among these, audiometry and electronystagmography (ENG) are the tests most commonly used as diagnostic adjuncts during the outpatient work-up of dizziness.

In dizzy patients with unilateral hearing loss, particularly those with progressively worsening hearing, formal audiometry is routinely recommended on an outpatient basis. Evidence of hearing loss will be found in the majority of patients with Ménière's disease and in those with acoustic neuromas.[26,46] However, as there is generally no role for audiometry in the ED, scheduling this intervention is usually left to the consultant.

ENG is a test of vestibular function that uses electrodes to monitor nystagmus that appears spontaneously or when induced by lateral gaze, positional change, or caloric testing.[19] Specific abnormalities in ENG testing may indicate peripheral or central vestibular disorders. Although several studies have shown its effectiveness in detecting vestibular abnormalities, the value of ENG in differentiating between peripheral or central causes is questionable.[37,63] The American Academy of Neurology recently concluded that the evidence for ENG was Class III—offering no clear benefit.[64] As with audiometry, ENG is likely to be impractical in the ED (although it has been studied in this setting[5]).

ED Therapy

Epley Maneuver

After confirming the diagnosis, therapy for BPPV is centered on repositioning the inner ear crystals to prevent recurrence of symptoms. This can be achieved via a bedside maneuver introduced by Epley in 1992.[65] The canalith repositioning maneuver, often called the Epley maneuver, involves a series of head-positioning maneuvers that allow the floating particulate matter in the posterior semicircular canal to pass into the utricle, thereby eliminating abnormal vestibular input and improving symptoms. This maneuver is described in the accompanying diagram. (See **Figure 2** on page 14.) Repetition of the procedure is recommended for patients not experiencing relief after the initial maneuver. Epley reported an 80% success rate after a single treatment session and a 100% success rate after more than one session.[65] Other attempts to replicate Epley's success rate have measured success rates ranging from 44% to 88%.[66-68] A

randomized trial of the Epley maneuver vs. untreated controls showed 89% success in the treatment arm and 23% in the control arm.[69] Presumably, this procedure can also be performed in an ED setting, although no ED success rates have been reported. Vestibular rehabilitation exercises consisting of repetitive side-to-side head movements performed while lying down should be provided as part of the discharge instructions for patients with BPPV. They have been shown to be very effective in reducing the need for return ED visits for recurrent symptoms.[70-72] However, even with treatment, Epley reported a 30% recurrence rate over a 30-month period.[65]

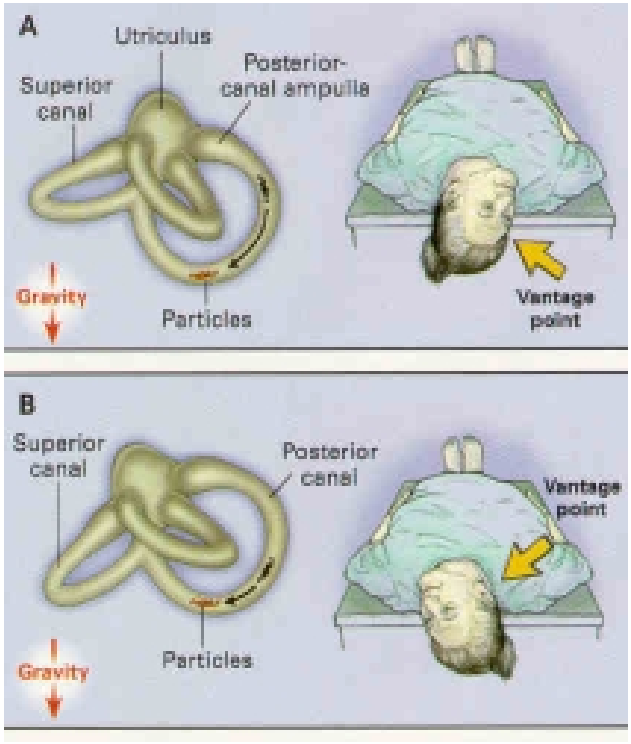
Vestibular Suppressants

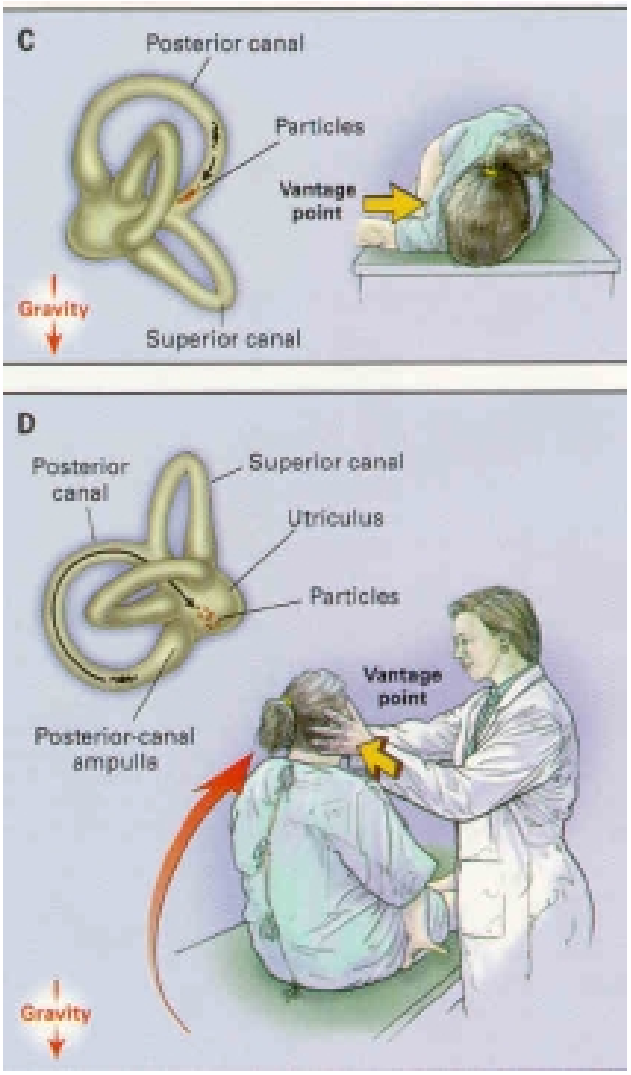
Vestibular suppressants (see **Table 4** on page 15) are also effective for peripheral vestibular symptoms from a variety of causes, such as BPPV, vestibular neuritis, or labyrinthitis. Meclizine, an H_1-antagonist antihistamine, is the most commonly used medication for peripheral vertigo and can significantly reduce symptoms.[73] Other antihistamines, including dimenhydrinate and diphenhydramine, have also been used. Their efficacy is likely mediated by their anticholinergic activity. These agents inhibit muscarinic acetylcholine receptors involved in feedback from the brainstem to the vestibular labyrinth, rather than via direct H_1-receptor blockade.[74] In patients with prominent nausea or vomiting, antiemetics such as promethazine or prochlorperazine are effective, although extrapyramidal effects or dystonia can occur.[20]

Benzodiazepines are also useful in the treatment

Figure 2. The Epley Maneuver.

This figure depicts the bedside maneuver for the treatment of a patient with benign paroxysmal positional vertigo affecting the right ear. The presumed position of the debris within the labyrinth during the maneuver is shown in each panel. The maneuver is a three-step procedure. First, a Dix-Hallpike test is performed with the patient's head rotated 45° toward the right ear and the neck slightly extended with the chin pointed slightly upward. This position results in the patient's head hanging to the right (Panel A). Once the vertigo and nystagmus provoked by the Dix-Hallpike test cease, the patient's head is rotated about the rostral-caudal body axis until the left ear is down (Panel B). Then the head and body are further rotated until the head is face down (Panel C). The vertex of the head is kept tilted downward throughout the rotation. The maneuver usually provokes brief vertigo. The patient should be kept in the final, face-down position for about 10-15 seconds. With the head kept turned toward the left shoulder, the patient is brought into the seated position (Panel D). Once the patient is upright, the head is tilted so that the chin is pointed slightly downward.





Used with permission from: Furman JM, Cass SP. Benign paroxysmal positional vertigo. *N Engl J Med* 1999 Nov 18;341(21):1590-1596. Figure 3.

of vertigo.[75] Their mechanism of action, however, is through generalized inhibition of neural activity, as they have no specific effect on the vestibular system.[74] As the natural course of BPPV consists of intermittent, brief, symptomatic episodes separated by asymptomatic periods, improvement with vestibular suppressant therapy is not necessarily diagnostic.[23]

In a recent review of BPPV, the authors did not encourage the use of vestibular suppressants, suggesting that, while they may suppress the intensity of symptoms, they do not reduce the frequency of attacks of recurrent vertigo. Side effects of these medications include lethargy, sleepiness, and worsening of balance.[23] Although vestibular suppressants can be helpful in the acute setting, long-term use of these medications may impair vestibular compensation.[76] For patients with severe symptoms accompanied by disequilibrium despite therapy (particularly the elderly at risk for falling), admission may be necessary. In discharge instructions for patients with BPPV, it is important to mention that their symptoms may be recurrent.

Therapy for Ménière's disease consists of vestibular suppressants, as listed above. Although a low-salt diet and diuretics have also been advocated,[26] there is no convincing evidence for their utility.[77]

In patients with post-traumatic vestibular syndromes, otolaryngology referral is appropriate, and in the setting of skull fracture, further trauma evaluation is mandated as well. In the absence of infection, antibiotic therapy (either topical or oral) is unnecessary.

If patients complain of intractable and incapacitating vestibular symptoms, inner-ear surgery may ultimately be required, including operations on the endolymphatic sac or even labyrinthectomy (a destructive ablation of the semicircular canals and vestibule). The vast majority of patients with vestibular disorders, however, can be managed with conservative therapy including vestibular rehabilitation and medication.[31]

Other Therapies

The comprehensive treatment of patients with suspected stroke or TIA is beyond the scope of this article. Those with possible TIAs may benefit from antiplatelet agents such as aspirin. Other agents such as clopidogrel or dipyridamole may be used if the patient has contraindications to aspirin, or they may be added to the aspirin therapy if the patient is already on aspirin.[29,78] Despite the occasionally catastrophic outcome, optimal therapy for patients with symptomatic intracranial vertebral artery or basilar stenosis remains unclear. Patients with stroke may require more invasive therapy, including cerebral angiography, angioplasty, or stenting.

Treatment for cardiovascular causes of dizziness should be directed at the underlying disorder, whether arrhythmia, ischemia, or structural heart disease. Hypovolemia or symptomatic anemia should be corrected as necessary.

For patients in whom drug-induced ototoxicity is suspected, the offending agent should be stopped immediately and the patient referred for audiometry and otolaryngology follow-up. For other patients with potentially drug-related symptoms, such as a sensation of intoxication or disequilibrium, a careful re-evaluation of the patient's medication regimen is appropriate.

Relaxation techniques can alleviate symptoms in hyperventilating patients and reverse respiratory alkalosis by allowing low pCO_2 levels to return to normal. Anxiolytics in conjunction with psychiatric referral or consultation may be necessary for patients with severe or refractory symptoms.

Special Considerations

Elderly Patients

Elderly patients make up a significant portion of patients who present with dizziness and, unfortunately, are at greater risk for the complications of dizziness as well. They suffer from functional disability caused by unsteady gait and fear of falling—not to mention increased frequency of falling and its sequelae.[8,79,80] Elderly patients are more likely to present with central causes of vertigo, such as ischemic cerebrovascular disease, and are more likely to be debilitated by peripheral vertigo symptoms. However, even among the elderly, life-threatening causes of dizziness are rare.[36]

Children

Children do not often complain of dizziness, but, as several recent reviews of dizziness in children have shown, they do present with a similar array of vestibular and non-vestibular problems.[81,82] Ear infections are more common in this age group and may progress to suppurative labyrinthitis or mastoiditis, leading to prominent vestibular symptoms. A chronic effusion ("glue ear") may lead to vestibular

Table 4. Vestibular Suppressant Therapy.

Class	Medication	Dosage
Antihistamines	Meclizine	25 mg PO q8-12h
	Diphenhydramine	25-50 mg PO q6h
Anticholinergics	Scopolamine	0.5 mg transdermal patch q3-4d (behind ear)
Phenothiazines	Promethazine	25-50 mg PO/PR q6h
Benzodiazepines	Diazepam	2 mg PO q8-12h
	Clonazepam	0.5 mg PO q12h

dysfunction in children.[83] This is the exception, however, as most complaints tend to be relatively benign and self-limited. Acute cerebellar ataxia is a condition primarily seen in children less than 6 years of age following a viral infection (varicella being a prime offender in the unimmunized child). Toxic ingestions may be another cause of ataxia in children.[84] An evaluation similar to that for adults, with emphasis on a careful history and physical, will likely narrow the differential significantly.

Important questions to ask include:

- Was there a fever?
- Did the child recently have chickenpox?
- Is vomiting or diarrhea present?
- Is there a history of head or neck trauma?
- Is there a family history of seizures or migraines?
- Are the symptoms related to exercise?
- Are there social stressors in the home or at school?

In younger children, signs of infection or volume depletion may be important clues to the diagnosis.

Treatment of dizziness in children, as in adults, depends on the likely source, including vestibular suppressants and antiemetics for children with vertigo. Rarely, a child will have such severe vestibular symptoms that he or she will need admission. Hypovolemia and fever warrant further investigation, as does near-syncope or syncope, including ECG to rule out long QT syndrome. Follow-up with the child's pediatrician is usually adequate.

Summary

Dizziness is often an acute event that sends patients careening to the nearest ED. There, they hope to find rapid relief from an array of distressing symptoms. By allowing dizzy patients to give an open-ended explanation of their

Ten Excuses That Don't Work In Court

1. "I thought the meclizine would help…not cause her to fall and break her hip."

Meclizine may be beneficial in cases of vestibular disease with vertigo, but unfortunately it can make patients with the "weak and dizzies" weaker and dizzier. Consider the pros and cons of anti-vertiginous medications, especially in the elderly and those on other psychoactive medications.

2. "I know it was vertical nystagmus, but there were no other neurological findings so I assumed it was peripheral vertigo."

Too bad for the patient that the physician didn't recognize there were significant risk factors for cerebrovascular disease and that vertical nystagmus indicates a central process until proven otherwise. The patient's posterior circulation stroke progressed to the locked-in syndrome.

3. "I thought it was obvious that the patient shouldn't drive."

Nothing is obvious. When the patient turned to change lanes, his benign positional vertigo was no longer so benign.

4. "The vertigo had subsided, so I thought it was okay for him to walk to the bathroom."

Supervise the activity of ED patients with vertigo to ensure that they do not fall and add injury to their problem.

5. "But the ECG was normal, so who would suspect an arrhythmia?"

This patient had syncope and had a pacemaker. Remember that the ECG is just a snapshot, and continuous monitoring is necessary to identify intermittent disturbances. The pacemaker may need to be checked with either a magnet or a computerized inquiry system.

6. "The patient was too young to worry about a stroke. By the way, what is a vertebral artery dissection?"

Strokes can occur at any age. Carotid and vertebral artery dissections are a potential cause of stroke in patients under the age of 40; vertigo is often the initial presentation in vertebral artery dissection due to posterior circulation compromise.

7. "I didn't know that the patient had decreased hearing."

How could you know if you didn't check? He ultimately went deaf. Testing hearing is a critical component of assessing vertigo in that it can help in localizing the underlying etiology.

8. "The CT was normal, so I thought it was safe to send the patient home."

This patient had poked himself in the eye on finger-nose testing. That indicates cerebellar dysfunction. Unfortunately, head CTs are notoriously insensitive for detecting posterior fossa lesions; thus, clinical judgment must be used in patient dispositions.

9. "Her boyfriend said that she's always dizzy. Besides, her exam was normal except for shoulder pain and a slightly tender belly, so I didn't see any reason to do laboratory testing."

She might "always be dizzy," but this time her ectopic pregnancy made it worse. Evaluating the dizzy patient requires an organized, systematic approach that helps avoid legal pitfalls.

10. "The patient came from the psychiatric hospital, so I assumed that he was crazy."

Even crazy people get medical illnesses. In this case, the patient was on valproic acid as a mood stabilizer, and his unsteadiness was due to drug toxicity. Sending him back on his normal dose did not help. ▲

symptoms, then asking a few clarifying questions, the emergency physician can often come to a rapid and confident diagnosis. Ask about onset of symptoms, time course, and associated symptoms—whether neurologic, cardiovascular, or psychological.

The physical exam should target abnormal vital signs, the presence of nystagmus (horizontal or vertical, fatiguing or non-fatigable), hearing loss, and neurological deficits. Three inexpensive tests that have good potential diagnostic yield include the ECG, bedside postural testing, and the Dix-Hallpike maneuver. Remember that meeting strict orthostatic criteria is not important if symptoms are reproduced.

With the help of the Clinical Pathway, "Evaluation Of The Dizzy Patient" (on page 10), patients who have associated neurological symptoms or deficits can proceed to rapid, cost-effective diagnostic imaging and definitive therapy. Equally important, patients whose symptoms suggest peripheral vestibular symptoms are best served by diagnostic and therapeutic bedside maneuvers that can produce rapid results without the need for unnecessary testing or expense.

A simple, streamlined approach to the evaluation of dizziness will serve both you and your patients well. No longer will the thought of one more weak and dizzy patient fill your heart with dread. With a new approach using a few simple questions, some quick, easy maneuvers, and the right diagnostic tests, both you and your patients will be walking the straight and narrow in no time. ▲

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in the paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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Physician CME Questions

33. Medications that can lead to dizziness include all of the following *except:*
a. levofloxacin.
b. phenytoin.
c. haldol.
d. gentamicin.
e. lorazepam.

34. What percentage of ED patients typically present with dizziness?
a. 5%
b. 10%
c. 25%
d. 40%

35. The item most likely to reveal the cause of dizziness is the:
a. history.
b. physical exam.
c. ECG.
d. CT scan.
e. MRI.

36. Nystagmus due to peripheral vertigo is characterized in all of the following ways *except:*
a. horizontal-rotatory.
b. non-fatigable.
c. suppressed with ocular fixation.
d. often associated with nausea and vomiting.

37. The most common cardiovascular abnormality leading to dizziness is:
a. tachycardia.
b. bradycardia.
c. hypovolemia.
d. ischemic heart disease.

38. Which of the following is *not* a therapy for BPPV?
a. Vestibular rehabilitation exercises
b. The Epley maneuver
c. The Dix-Hallpike maneuver
d. Vestibular suppressants

39. Which of the following is *not* a typical finding in Ménière's disease?
a. Hearing loss
b. Vertigo
c. Tinnitus
d. Vertical nystagmus

40. A patient complaining of diplopia, vertigo, and facial numbness likely has an infarct in the:
a. internal capsule.
b. brainstem.
c. frontal lobe.
d. basal ganglia.

41. For a young male with near-syncope, appropriate ED testing would include:
a. CBC, CT scan, glucose, orthostatics.
b. electrolytes, orthostatics, ECG.
c. Dix-Hallpike maneuver, glucose, orthostatics.
d. Hemoglobin, orthostatics, ECG.

42. Benign positional peripheral vertigo (BPPV) is thought to be due to:
a. abnormal build-up of endolymph fluid.
b. abnormal movement of otolithic debris in the middle ear.
c. ischemia of the vertebrobasilar artery system.
d. abnormal connection between the middle and inner ear.

43. Patients with vestibular neuritis/labyrinthitis usually have symptoms that last:
a. seconds.
b. minutes.
c. hours.
d. days.

44. Vestibular suppressants for the treatment of peripheral vertigo include all of the following *except:*
a. diphenhydramine.
b. prednisone.
c. meclizine.
d. promethazine.
e. diazepam.

45. An elderly man with a history of prior stroke, CABG, and hypertension presents with vertigo. The initial head CT is negative. Appropriate evaluation may include all of the following *except:*
a. ECG.
b. MRI with MRA.
c. reassurance and discharge without follow-up.
d. aspirin or antiplatelet agent.
e. consultation with the patient's primary physician or a neurologist.

46. All of the following are central causes of dizziness *except:*
a. vertebrobasilar insufficiency.
b. Ménière's disease.
c. cerebellar infarction.
d. neoplasm.
e. multiple sclerosis.

47. A 24-year-old woman presents with hypotension, pallor, and dizziness. She says, "I think I'm going to pass out." Appropriate initial evaluation includes all of the following *except:*

a. large-bore intravenous access.
b. orthostatics and gait testing.
c. CBC, type, and crossmatch.
d. pregnancy test.
e. taking the patient's history.

48. Elderly patients with dizziness are *not* at increased risk for which of the following?

a. migraine headaches.
b. long bone fractures.
c. decreased mobility.
d. difficulty with daily activities.
e. falls.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives an alpha-numerical score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA* 1992;268(16):2289-2295.

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