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The Practical Journal for Emergency Physicians

Emergency Medicine Reports®

Volume 26, Number 14 June 27, 2005

Dizziness

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Introduction

Few chief complaints cause more apprehension and dread for emergency physicians (EPs) than dizziness. It is a common condition seen in the emergency department (ED), is understood poorly, and has potentially malignant etiologies. Dizziness cannot be measured. It can mean different things to different patients and is often difficult to precisely characterize. Unfortunately, it is the ability to obtain a precise history and perform an exacting examination that allows a diagnosis to be made and appropriate treatment instituted. This article examines some of the different causes of dizziness, how they can be differentiated via history and physical examination, and their appropriate treatments and dispositions.

Epidemiology

Dizziness is a sensation of abnormal orientation in space.[1] It is the third most common outpatient complaint.[2] It accounted for about 1.5 million ED visits in 2001.[3] Patients with benign paroxysmal positional vertigo (BPPV) on average are seen by four physicians at a cost of more than $2000 per work-up.[4] Twenty percent of patients older than 60 years have experienced dizziness significant enough to affect their daily activities.[5,6] Drachman and Hart described four subtypes that still remain the basis for dizziness definition and classification today: vertigo, presyncopal lightheadedness, disequilibrium, and "other" dizziness.[7] A meta-analysis by Kroenke and colleagues found that the most common

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This publication does not receive commercial support.

etiologies for dizziness were peripheral vestibulopathies (44%), psychiatric disorders (16%), central vestibulopathies (11%), other causes (26%), and unknown causes (13%).[8] Causes of disequilibrium are rarely the primary cause of dizziness, though they are common contributory factors. Serious causes of dizziness include cerebrovascular disease (6%), arrhythmia (1.5%), and brain tumors (<1%). While a significant proportion of dizziness will resolve within a two-week time frame, dizziness that remains longer generally has a multifactorial etiology.[8]

Pathophysiology

The pathophysiology of dizziness is specific to the condition that accounts for the underlying cause. The maintenance of balance is a complex interaction whereby the central nervous system coordinates and integrates sensory input from vestibular, visual, and proprioceptive systems. Vestibular input is transmitted via the 8th cranial nerve. The labyrinth (inner ear) includes three semicircular canals that are perpendicular to each other and are oriented in three planes of space. Rotational acceleration causes endolymph within the semicircular canals to deform hair cells attached to the cupula generating signals that allow perception of movement and position.[9] This input helps form the impression of orientation of head and body and the perception of motion.

Sensory input that helps maintain balance is symmetric from the bilateral vestibular systems. If this input becomes asymmetric it causes vertigo.[10] In BPPV, canalithiasis is thought to be the most common mechanism and describes free-floating otoconia (i.e., calcium carbonate crystals) in a semicircular canal. These otoconia are displaced from the otolithic membrane in the utricle.[9] The right posterior semicircular canal is most commonly affected because it is the most gravity dependent portion of the vestibular labyrinth.[9,11] Head movements then cause the otoconia to move, which in turn causes a plunger effect on endolymph in the canal. This leads to hair cell stimulation, and the cupula is displaced.[10,12] The central nervous system (CNS) interprets these signals as angular acceleration of the head when none exists, producing vertigo and nystagmus.[10,13] Cupulolithiasis, where otoconia are adherent to the cupula of a semicircular canal, also can occur but is less common.[9] The cupula with the attached otoconia is thought to become heavy relative to the endolymph and deflects hair cells with position change causing vertigo and nystagmus.[13] Vertigo found in other conditions (e.g., migraine and stroke) is thought to be due to ischemia to labyrinth. [14] Tumors can cause vertigo through deformation of the labyrinth or vestibular structures. In Meniere's disease, vertigo is thought to occur from end organ deformation due to endolymphatic hydrops.[15] Presyncopal lightheadedness is caused by transient global hypoperfusion to the brain, which usually is due to cardiovascular-mediated abnormalities. Common causes include arrhythmias, orthostatic hypotension, autonomic dysfunction, cerebrovascular atherosclerotic disease, anemia, and dehydration.[10,16] Disequilibrium is thought to cause dizziness through a loss of visual, vestibular, and/or somatosensory input needed to maintain balance.[17]

History

The history obtained in the evaluation of a dizzy patient is the most important aspect of the EP's workup. In up to 76% of cases of dizziness, a diagnosis may be made from history alone.[18,19] History was most sensitive in diagnosing vertigo (87%), presyncope (74%), psychiatric disorders (55%), and least sensitive in disequilibrium (33%).[19] Unfortunatel,y it has been found that up to 40% of some patients' complaints are too indistinct to fit into a single category[18] Regardless, a meticulous history that attempts to precisely define symptoms and elicits inciting and modifying factors is paramount. It is important to have the patient describe exactly what he experiences when he complains of dizziness without biasing his answer. This often can be accomplished by asking the patient to describe his symptoms without using the word "dizzy." Even then it can be very difficult for the patient to put the symptoms into a meaningful description. Determining the exact sense of imbalance is important. A sense that the patient or his environment is moving (i.e., usually spinning) suggests vertigo. Presyncopal lightheadedness is described as a sense of impending faint and is due to transient diffuse cerebral hypoperfusion as may occur with orthostatic hypotension.

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Glen Harris
Specialty Editor: Shelly Morrow Mark
Marketing Manager: Nan Reeves

GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

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Disequilibrium is a loss of balance without head sensation often due to neuromuscular problems. The "Other" category of dizziness, frequently attributed to psychiatric illness, often is accompanied by other somatic complaints and/or signs of underlying psychiatric involvement.[20,21] Rapidity of onset and severity of symptoms are important to elicit. Generally, rapid severe symptoms suggest peripheral vertigo, though this also may occur with cerebellar bleeding/infarction. Mild symptoms with gradual onset are more suggestive of a central process (e.g. tumor). Associated symptoms are important to ascertain; benign conditions usually are not associated with such complaints as headache, altered mental status, focal weakness/numbness, speech difficulty, neck pain, or vision disturbance. Symptoms such as nausea/vomiting, diaphoresis, and pallor are not uncommon with vertigo.[22] Noting the duration of symptoms is also essential; an episodic nature is often suggestive of a benign peripheral process, whereas continuous symptoms are more likely due to a more malignant central one. Modifying or inciting factors are important to identify. Symptoms that are brought on by head movement suggest a vestibular process.[23] Medication history also is critical; prescribed medication toxicity has been found to cause dizziness in up to 10% of cases.[20] Past medical history is also important to obtain. Patients with benign vertigo may have had previous similar episodes. Knowing other medical conditions may give clues as to the etiology of a current episode (i.e., similar lightheadedness in a patient with a history of cardiac arrhythmia). Unfortunately, while these historical features often are useful and generally hold true, they are not foolproof in determining benign etiologies from more serious ones. The practitioner's clinical suspicion should be considered. Additionally, history from family members also is helpful to add to and corroborate the information obtained from the patient.

Physical Examination

In addition to a thorough history, a methodical physical examination is vital. Physical examination has been found helpful in confirming the diagnosis made by the patient's history but rarely changed it.[19] Potentially useful examination maneuvers include Dix- Hallpike testing, determination of orthostatic vital signs, gaze testing for nystagmus, and gait testing. It is difficult to limit the examination to certain body systems; there may be multiple systems that are potentially causative or at least involved in the patient's dizziness. Orthostatic vital signs may be important to check because they can indicate volume depletion as a factor. Specifics of the head examination include checking the ears for cerumen impaction and otitis media, which have been reported to cause vertigo. Tympanic membrane perforation or nystagmus elicited by pneumatic otoscopy may indicate perilymphatic fistula. [24] Cholesteatoma may be suggested by noting a white pearl-like mass behind the tympanic membrane. Detection of vertigo with impaired hearing to whispered voice may indicate sensorineural hearing loss from tumor with compression of the ipsilateral 8th nerve.[1] The eye examination is vital to differentiate central from peripheral processes. Peripheral vertigo may have either horizontal or rotatory nystagmus, which abates with visual fixation. With horizontal nystagmus, the slow component of eye movement is toward the affected ear with rapid movement back to the midline. Central vertigo can be horizontal or rotatory, but does not fatigue with visual fixation. Vertical, direction-changing or dysconjugate nystagmus, or nystagmus that is not suppressed with visual fixation is always abnormal and indicates a central process.[1] A few beats of horizontal nystagmus on extremes of lateral gaze is normal. One study found that about 10% of patients with BPPV will not have clinically detectable nystagmus.[25] A fundoscopic examination looking for papilledema may be important in assessing indirect signs of increased intracranial pressure as with an intracranial mass. Pupillary function and extraocular movement examination is also crucial. Third or sixth nerve palsy may indicate brainstem pathology or multiple sclerosis. [22] Visual acuity and visual field testing helps to determine if there is a sensory input deficit contributing to the patient's dizziness. More thorough cranial nerve examination is indicated based upon clinical findings. Testing for gait, dysmetria, and dysdiadochokinesia, and pronator drift should be performed for cerebellar dysfunction. Gait examination that shows a tendency to fall to the side of the lesion while walking is typical of peripheral vestibulopathy. Falling away from the lesion is usually from a central cause. [26] Cerebellar abnormalities suggest infarction or bleeding and are not found with peripheral vestibulopathies. Romberg testing should be done, but it actually tests proprioception rather than the cerebellum. Auscultation of bruits along the carotid artery may suggest atherosclerotic disease as the etiology of dizziness. Hyperventilation, if present, is nearly always associated with a psychiatric cause, usually an anxiety disorder.[2]

Another helpful test in differentiating peripheral from central vertigo is the Dix-Hallpike maneuver. This often erroneously is referred to as the Barany or Nylen-Barany test. This is the classic test for BPPV due to canalithiasis in the posterior semicircular canal, which is the most commonly affected canal.[27] The maneuver begins with the patient seated in the upright position (*Figure 1*). The examiner supports the patient's head and turns it 45 degrees to one side. The patient then quickly assumes the supine position with the head hyperextended approximately 45 degrees over the edge of the bed. The patient should keep his eyes open even if he feels dizzy and should look straight ahead. The procedure then is repeated with the head turned 45 degrees in the opposite direction.[28] The ear in the downward position when nystagmus and vertigo are elicited is the affected ear.[13] Nystagmus from a peripheral vestibulopathy is either horizontal or rotatory, has latency of onset of several seconds, extinguishes with fixation, and lasts fewer than 30 seconds.[23,29] The nystagmus also may reverse direction when the patient assumes the sitting position.[13] Nystagmus that has immediate onset, is vertical, or does not fatigue is suggestive of a central disorder. In a meta-analysis by Hoffman, the Dix-Hallpike maneuver was positive in 50-88% of those patients with BPPV.[29-32] If initial testing in patients with symptoms suggesting BPPV fails to elicit nystagmus, lying supine and repeating the test will confirm the diagnosis up to 20% of the time. If symptoms of BPPV are present and Dix-Hallpike testing is negative, a side-lying variation of the Dix-

Hallpike should be performed to test for horizontal canal BPPV, which occurs less than 1% of the time.[33] Dix-Hallpike testing may have limited utility in obese patients and those without normal range of motion in the neck, trunk, and hips. An alternative side-lying technique has been described that was shown to have equivalent findings to the traditional technique.[34]

Laboratory Testing

Laboratory testing adds little to the evaluation of dizziness. In a meta-analysis by Hoffman, lab abnormalities that explained the dizziness were found in only 3 of 4538 patients.[29] A study by Stewart and colleagues found blood laboratory studies to have low utility and were determined not to be cost effective in the routine work-up of vertigo.[35] Hemoglobin and hematocrit levels determination may be indicated if anemia is suspected. Chemistry studies to evaluate for pre-renal azotemia or adrenal insufficiency also may be indicated if these conditions are suspected. Blood glucose level determination also may be helpful occasionally. Other laboratory studies are not routinely indicated.[2] Routine use of electrocardiogram (ECG), blood testing, and magnetic resonance imaging (MRI) is not recommended due to the frequency of abnormalities seen in symptom-free controls.[36]

Cardiovascular Testing

Electrocardiograms rarely are helpful in the evaluation of the dizzy patient. Two studies found no electrocardiogram changes that were diagnostic in dizzy patients.[37,38] An ED series reported significant electrocardiogram findings in six of 125 patients, five of whom had a known history of arrhythmia.[39] Ambulatory electrocardiographic monitoring was normal in 70% of dizzy patients, half of whom had symptoms. Paroxysmal atrial fibrillation was the most common abnormality found, but no patients had symptoms during episodes of the arrhythmia.[36] Noninvasive carotid studies found hemodynamically significant lesions in 21 of 101 patients referred with dizziness but were normal in the seven patients with complaints of isolated vertigo.[40]

Imaging

Routine imaging of the dizzy patient is not required. Computerized tomography (CT) scans frequently are not helpful; they are not the ideal study to evaluate for intracranial mass lesions, and cannot diagnose multiple sclerosis. Additionally, CT is not ideal for imaging the posterior fossa (e.g., cerebellum, pons, medulla), which is often the area of the brain with abnormalities causing central vertigo. In the evaluation of vertigo/dizziness thought to be due to a central process, MRI is usually the imaging modality of choice.[41] When MRI is used as a general screening tool for undifferentiated vertigo, it is not cost effective. If applied more selectively in patients with symptoms that suggest a central cause, the cost effectiveness likely increases.[35]

Figure 1. Dix-Hallpike Maneuver

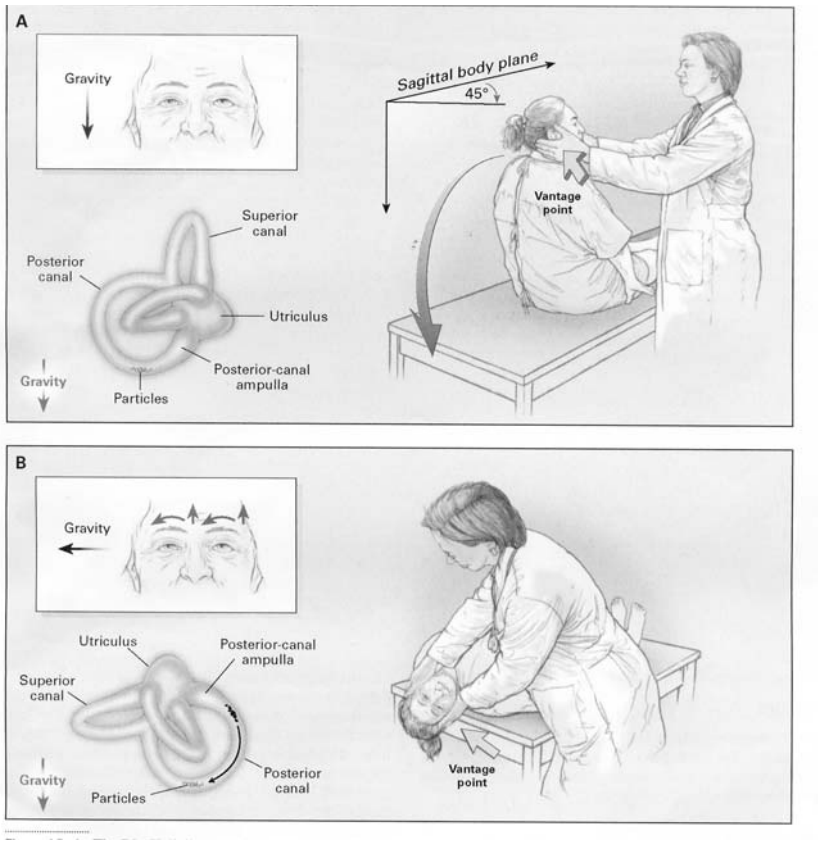

Figure 1. **A**.The patient assumes a sitting position far enough back on the stretcher so that when she lies back her head will extend over the end of the stretcher. The head is turned 45 degrees to one side and is supported by the examiner. **B.** The patient rapidly assumes the supine position with the head extended over the bed 45 degrees and turned 45 degrees to the side. The examiner should continue to support the patient's head. Reproduction of vertigo and nystagmus is diagnostic of BPPV. The maneuver is repeated with head turned to the opposite side if necessary.

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The Subtypes of Dizziness

Vertigo. Vertigo is a subtype of dizziness with patients describing the illusion that they (subjective vertigo) or their environment (objective vertigo) is spinning.[13] Although there are a large number of causes, an immediate concern for the EP is to distinguish between relatively benign peripheral causes and potentially life-threatening central causes (*Figure 2*). Central versus peripheral causes will be discussed further.

Peripheral Vertigo. *BPPV*. First described by Barany in 1921, BPPV is characterized by brief, self-limited (paroxysmal) episodes of vertigo provoked by typical position changes.[42] It is most common between the 5th-7th decades. There is a female predominance 1.6:1.[13] The most common cause of BPPV is primary or idiopathic and accounts for 50-70% of cases. The most

Figure 2. Differentiating Peripheral Versus Central Vertigo

	PERIPHERAL	CENTRAL
Onset:	Abrupt	Gradual
Intensity:	Moderate-intense	Mild
Duration:	Usually seconds-minutes	Weeks-months, but can last longer
Temporal pattern	Episodic	Chronic
Nystagmus:	Latency	No latency
Nystagmus-Direction:	Horizontal, rotatory Vertical	Horizontal, rotatory Dyscongugate
Nystagmus:	Fatigues	Does not fatigue
Changes in Head:	Worsens vertigo	No effect on vertigo
Associated neurologic:	None	Often present
Associated hearing:	May be present	Absent

common secondary cause is trauma accounting for up to 17% of cases.[9] Symptoms include sudden onset of vertigo, nystagmus, nausea, and a tendency to fall to the side of the lesion without cochlear or other neurologic symptoms.[26] Symptoms are usually acute onset, short in duration (i.e., lasting < 30 seconds), severe, and brought on by certain head positions and movements. Common movements include rolling over in bed, looking up, and bending forward.[28] Although usually a benign condition, patients are at risk for falls and other traumatic injuries (i.e., motor vehicle crash while driving). History, eye findings, and response to the Dix-Hallpike maneuver are usually all that is needed to make the diagnosis. Nystagmus usually has a latency of several seconds, is horizontal or rotatory, and fatigues with visual fixation. Vertigo does tend to recur with rates of about 10-15% per year.[43]

Canalith Repositioning Procedures

The recommended treatment for BPPV is a canalith repositioning procedure (CRP). There are several CRPs described in the literature; the most studied and used in the United States was first described by Epley in 1992.[9,44] It is a series of position changes that attempts to restore the free-floating otoliths in the posterior semicircular canal to their proper place in the utricle. The maneuvers are effective, safe, inexpensive, and have few side effects or relative contraindications. The Vestibular Disorders Association recommends the Epley maneuver as firstline treatment for BPPV.[42] The Epley maneuver is performed as follows (*Figure 3*):

1. The patient starts in the sitting position.
2. Then, he assumes the supine position with head extended over the edge of a table (stretcher) at a 45-degree angle with the head turned toward the affected side.
3. Once the vertigo and nystagmus have ceased, the head is then turned opposite the affected labyrinth 45 degrees from the midline.
4. The patient's body then is turned in that direction assuming the lateral decubitus position with the head slightly angled and looking at the floor.
5. The patient then is returned to the sitting position.
6. The chin is tilted down to touch the chest.[28]

This maneuver may need to be performed more than once. Another CRP, the Semont maneuver, involves having the patient assume a side-lying position with the affected ear down for four minutes, then quickly turning over so that the other ear is down for four minutes, then returning to the sitting position.[45,46]

A meta-analysis that includes a Cochrane review found that those patients treated with CRP were more likely to demonstrate symptom resolution at the time of first follow-up, though there were few methodologically sound studies from which to extract these data.[12] Multiple studies have shown that the Epley maneuver provides both objective (i.e, positive Dix-Hallpike maneuver to negative maneuver) and subjective improvement in treating BPPV.[45,47-52] It has also been shown that generalists and EPs have been able to effectively use the Epley maneuver in the treatment of BPPV.[53,31] In many patients, BPPV will resolve spontaneously; at one month as many as 23% of patients still will be symptomatic.[54] Tirelli found that if patients have symptoms of BPPV, but nystagmus cannot be elicited with Dix-Hallpike testing, CRPs still were 60% effective in relieving symptoms.[25] Steenerson found equivalent efficacy with Epley and Semont techniques in resolving BPPV with 94% and 98% success rates, respectively.[44] The Epley maneuver required fewer treatment sessions than Semont (2.98 vs. 4.34, respectively) and had fewer recurrences at six months (12% Epley vs. 21.8% Semont).[44,12] Success rates of relieving BPPV with single treatment sessions for the Epley maneuver are reported from 44-88%.[45,48,55-58] The only factor shown to be associated with need for multiple treatments was BPPV not located in a single posterior semicircular canal.[58] The Epley maneuver also is thought to be easier to perform in older patients.[45,48] Some advocate having the patient remain upright for one or two days after CRP, sleep upright, and avoid bending over, though studies have not shown post-CRP position instructions to make a difference.[13,59,60] CRP maneuvers can be taught to patients who can do them at home, either for continued treatment of their BPPV that is not resolved completely in the ED or for recurrences. In this case, the Epley maneuver was shown to be more effective in relieving BPPV than the Semont maneuver.[61] There is no evidence to show that these maneuvers affect later recurrences of BPPV.[62]

Vestibular sedatives, benzodiazepines, and antihistamines (e,g., meclizine) are thought to improve symptoms through blocking cholinergic transmission in the vestibular nuclei. Although some physicians recommend them, there are little data to support their efficacy, and they should be considered only as adjunctive therapy to those with BPPV with severe symptoms (primarily nausea).[63] CRP maneuvers are considered definitive firstline treatment.[13,61] Because it has antiemetic properties, meclizine may be an effective adjunct to CRP treatment of BPPV.[64] Also, these medications may impair vestibular compensation and habituation and cause sedation with increased fall risk.[65] Treatment of associated nausea may require use of antiemetics. Medications (e.g., promethazine and metoclopromide) are useful for their effects on nausea and due to anticholin-

ergic and antidopaminergic properties, which may help reduce vertigo symptoms.[10] Patients with BPPV usually are treated as outpatients, provided they have adequate support, do not have intractable vomiting, and their vertigo is not so severe as to put them at risk for falls. They should be encouraged not to drive or perform any other similarly dangerous activity until their symptoms have resolved completely. Outpatient otolaryngology or neurology referral is indicated.

Meniere's Disease

The classic triad of tinnitus, fluctuating hearing loss, and vertigo characterizes this condition. Attacks can be severe with vomiting and aural pressure/fullness. Unilateral sensorineural hearing loss suggests Meniere's disease. Usually with spontaneous recovery in hours to days, the disease becomes progressively more frequent and severe. Hearing abnormalities may not resolve between episodes. Meniere's disease is caused by excessive production or reduced resorption of endolymph.[24] An autoimmune mechanism also may be involved. The mechanism of vertigo in Meniere's disease is not understood well but does not appear to be due to cupulolithiasis or canalithiasis.[26] Treatment is generally effective and includes salt restriction, smoking cessation, caffeine avoidance, and diuretics to reduce endolymphatic fluid. A histamine derivative, betahistine, also has been shown to be effective by reducing asymmetrical vestibular function and increasing vestibular blood flow.[10,65] Vestibular suppressants (e.g., meclizine) and central suppressants (e.g., valium) may be effective in reducing symptoms but not correcting the underlying pathophysiology.[24,64] As with BPPV, patients with Meniere's disease usually can be treated as outpatients with otolaryngology or neurology follow-up.

Figure 3. Epley Canalith Repositioning Procedure

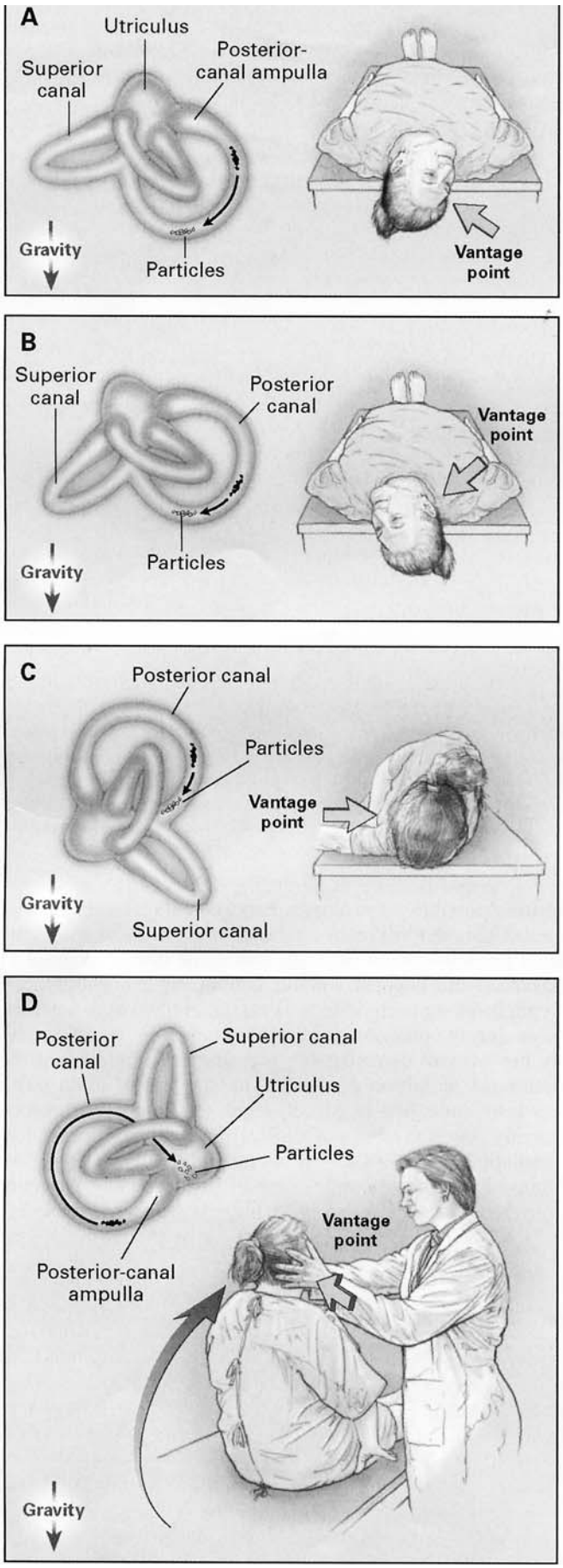

Figure 3. Performing the Epley Canalith Repositioning Maneuver. **A.** In similar fashion to performing the Dix-Hallpike maneuver, the patient assumes a sitting position far enough back on the stretcher so that when he lies back his head will extend over the end of the stretcher. The head is turned 45 degrees to the side that brought on vertigo and nystagmus when performing the Dix-Hallpike maneuver. **B.** As with the Dix-Hallpike maneuver, the patient is rapidly brought back to the supine position with his head extended 45 degrees over the edge of the stretcher and turned 45 degrees to the affected labyrinth (in this case the right). The patient remains in this position for about 30 seconds or until vertigo and nystagmus have resolved. Examiner should support the patient's head throughout the procedure. **C.** The head is then turned 45 degrees past the midline in the opposite direction. **D.** Again, after about 30 seconds or resolution of vertigo and nystagmus, the patient assumes the lateral decubitus position with the head looking down toward the floor. **E**. After 30 seconds or resolution of symptoms, the patient assumes the sitting position. **F**. The patient then places his chin to his chest. This procedure may be repeated as necessary.

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Vestibular Neuronitis (Acute Labyrinthitis)

The terms *labyrinthitis* and *vestibular neuronitis* are used interchangeably by some physicians. Others differentiate vestibular neuronitis as affecting only the vestibular fibers of the 8th nerve and not causing hearing loss. Labyrintitis affects both the vestibular and cochlear portions of the 8th nerve and usually is associated with hearing loss.[64] Here, they both will be referred to as vestibular neuronitis because the pathophysiology and treatment are the same. Most commonly thought to be caused by viral infections (without much support) that affect the vestibular nuclei, symptoms usually follow an upper respiratory infection within two weeks.[24,66] Other viruses (e.g., measles, mumps and rubella) also have been implicated. Otic herpes zoster that also affects CN VII and VIII is known as Ramsay Hunt syndrome.[24] Bacterial infections such as otitis media also are thought to cause vestibular neuronitis.[10] This condition is characterized by single or recurrent sudden episodes of vertigo that are severe for several days then subside during the course of a few weeks. Frequently it is associated with nausea, vomiting, and hearing loss. Also, the patient with vestibular neuronitis will have a positive (i.e., abnormal) head impulse (head thrust) test. In a patient with right-sided vestibular neuronitis, if the head is turned rapidly 15 degrees to the right, the patient is unable to maintain visual fixation on a distant object and must make voluntary rapid eye movements (saccades) back to the target, indicating loss of the vestibulooccular reflex. If this reflex is preserved, vestibular neuronitis is not present and symptoms likely are due to a cerebellar infarction.[66,67]Treatment centers on bed rest and pharmacologic suppression of symptoms with vestibular sedatives and antiemetics.[24,65] Medication usage should be withdrawn after a few days, if possible, and activity encouraged facilitating central compensation.[64] Viral testing is not indicated because causation of a vestibular syndrome cannot be proven.[67] Prednisone is helpful in the first ten days of treatment and may accelerate the process of central compensation.[65,68] Treatment with antivirals is indicated only for Ramsay Hunt syndrome.[68] Patients with vestibular neuronitis usually can be treated as outpatients with otolaryngology or neurology referral.

Traumatic Vertigo

Mild traumatic brain injury is the second most common neurologic disorder. The mechanism by which vertigo is caused is unclear but may be due to labyrinthine concussion with massive dislodging of otoconia.[69] This conditon can occur with blunt or whiplash-type trauma. Vertigo or nonspecific dizziness may be part of a postconcussive syndrome. Temporal bone fracture also must be ruled out, especially if there is hearing loss. Perilymphatic fistulas may occur after trauma and cause vertigo.[70] The mechanism is thought to be due to disruption of the lining membranes of the labyrinth at the oval or round windows.[15] Perilymphatic fistula may result from trauma such as straining with weight lifting, barotraumas from diving and flying, or forceful coughing or nose blowing.[65] An opening in either the round or oval window allows pressure changes to be transmitted to the vestibular apparatus. Diagnosis is made by inducing vertigo with pneumatic otoscopy. This condition usually heals spontaneously. Noncontrast CT scans generally are indicated at the time of traumatic injury to rule out intracranial bleeding or skull fracture.[10] Bed rest and avoiding head positions and other activities (e.g., sneezing, cough, straining) that elicit symptoms are helpful.[65] Surgery to repair the fistula occasionally is required.[71] Otolaryngology or neurology referral for follow-up examination and treatment is indicated

Other Causes of Peripheral Vertigo

Cerumen impaction and external auditory canal foreign bodies can cause vertigo. Usually, symptoms will resolve with removal of the offending material.[10]

Central Causes of Vertigo

Central vertigo is caused by dysfunction of the vestibular portion of the 8th nerve, vestibular nuclei in the brainstem, and their central connections.[72] It is uncommon in ED settings, accounting for less than 5% of vertigo cases.[64]

Cerebrovascular Disorders. Cerebrovascular disorders account for the largest proportion of causes of central vertigo. Vertigo rarely accompanies strokes/transient ischemic attacks of the anterior circulation, which supplies the bilateral cerebral hemispheres, internal carotids, and middle and anterior cerebral arteries. Vertigo is present in more than 75% of strokes/transient ischemic attacks involving the posterior circulation, which supplies the brainstem, cerebellum, and peripheral vestibular apparatus, basilar, vertebral, anterior and posterior inferior cerebellar arteries.[73] Posterior circulation etiologies for vertigo also are accompanied by other neurologic symptoms, usually dysarthria, numbness, diplopia, or hemiparesis.

These findings, though, may be very subtle and not even obvious to the patient.[10,73] A crossed defect (e.g., sensory/motor defect on one side of the face and the opposite side of the body) is characteristic of a brainstem lesion. Transient blindness, diplopia, or hemianopia may occur with occipital lobe ischemia. Ataxia, imbalance, and disequilibrium may occur with cerebellar ischemia.[72] Isolated, transient vertigo can occasionally occur.[68] This condition may occur with vertebral artery atherosclerosis, where certain head positions (i.e., turning to the side) may occlude the vessel and impair brainstem blood flow inducing transient ischemia. This condition is referred to as vertebrobasilar insufficiency (VBI). VBI usually is associated with other neurologic signs or symptoms that resolve. Cerebellar infarction is the primary serious condition to rule out in the patient with suspected vestibular neuronitis.[66] This is especially important because about one third of cerebellar infarctions will develop life-threatening posterior fossa edema requiring neurosurgical decompression. Also, the cause of cerebellar infarction is usually from a cardioembolic source and requires anticoagulation.[74] Examination findings that indicate cerebellar infarction include vertical or dysconjugate nystagmus that is not suppressed with visual fixation and the inability to stand without support.[66,72] Additionally, the patient has a normal head impulse (head thrust) test result differentiating this condition from vestibular neuronitis (*See sec-*

tion on vestibular neuronitis.). Dissection of the vertebral or basilar arteries most commonly is associated with vertigo and head or neck pain. Vertigo associated with head or neck pain is a dissection until proven otherwise.[75] Dissections have been well described after relatively minor trauma (e.g., motor vehicle crashes and chiropractic manipulation). MRI usually is required to radiographically diagnose posterior circulation cerebrovascular accidents. MR angiogram is the imaging modality of choice for suspected dissection and other posterior circulation abnormalities. Treatment of VBI usually requires antiplatelet agents and neurology consultation.[65] Treatment of dissection involves neurology/neurosurgical consultation and heparinization.

Migraine. Vertigo occurs in up to 33% and nonspecific dizziness occurs in up to 72% of patients with migraine.[14] Vertigo may be part of the aura before headache, a migraine equivalent, or may be unrelated to headache.[66] It is thought to be due to vascular damage to the labyrinth.[76] It can be spontaneous or positional.[77] Usually migraine- associated vertigo repeatedly is accompanied by migraine symptoms (e.g., photophobia).[78] Basilar artery migraine has symptoms similar to vertebrobasilar insufficiency (VBI) as described below.[10] Other causes of vertigo also must be ruled out. Vertigo in patients with migraines often can be treated with medicines used to treat migraines (e.g., ergots and triptans).[66] Tricyclic antidepressants and beta blockers also have been found to be effective.[14] Vestibular suppressants and referral for neurology follow-up and vestibular rehabilitation therapy is indicated.

Tumor. Cerebellopontine angle tumors (usually acoustic neuromas) rarely present with isolated vertigo. Headache often is present. Decreased hearing or tinnitus usually is present from compression of the cochlear portion of the 8th nerve. Disequilibrium usually is more commonly present than vertigo.[71,72] Posterior fossa tumors may present with symptoms of central vertigo and also have oscillopsia (i.e., a visual illusion of objects bouncing or jiggling).[72] Diagnosis is confirmed with MRI, and neurosurgical consultation is required.

Cerebellar Hemorrhage. Cerebellar hemorrhage usually presents with acute ataxia, ipsilateral gaze palsy, and ipsilateral cranial nerve VII palsy. The vertigo that accompanies this condition is usually of a sense of front-to-back or side-to-side motion rather than spinning.[10] Other accompanying symptoms include dizziness, repeated vomiting, dysmetrias, dysarthria, and inability to stand or walk. Rarely is this condition associated with hemiparesis or hemiplegia. Cerebellar edema may cause herniation of the tonsils into the foramen magnum with brainstem compression. Rapid recognition and neurosurgical decompression is often life-saving.[10,71,79]

Multiple Sclerosis. This condition is caused by CNS demyelinization. Patients present with vertigo that is quite variable in severity and duration, ataxia, severe nystagmus, and often other ocular symptoms such as neuritis. Diagnosis is confirmed by MRI. Neurology referral is required.[10]

Wallenberg Lateral Medullary Syndrome. This syndrome is caused by an infarction of the lateral medulla from occlusion of the intracranial vertebral artery and also the posterior inferior cerebellar artery. Symptoms begin acutely with vertigo, ataxia, nausea, and nystagmus. There is loss of ipsilateral facial pain and temperature sensation. Additionally, Horner's syndrome and paresis of pharyngeal and laryngeal muscles resulting in dysphagia and dysphonia are present. Loss of pain and temperature sensation also occurs on the contralateral side of the body. MRI and urgent neurology referral are indicated.[10,79]

Systemic Causes of Vertigo. Drugs are commonly found to cause vertigo. These include anticonvulsants, hypnotics, antihypertensives, furosemide, analgesics, tranquilizers, and aminoglycoside antibiotics.[72] Symptoms usually will abate with discontinuing the offending drug, though aminoglycosides can result in permanent effects.[71] Acute and chronic alcohol use may also cause vertigo. Endocrine disorders (e.g., hypothyroidism and diabetes mellitus) though more likely to cause disequilibrium, also can cause vertigo.[72]

Psychiatric Dizziness

Dizziness is a common complaint with certain psychiatric disorders and is the second most common symptom reported by patients with panic disorder.[80] It is often difficult to determine the cause of psychiatric dizziness because it is hard to know if the underlying psychiatric disorder is causing the dizziness or if the dizziness (usually vertigo) is causing the psychiatric symptoms. Often these spells, if psychogenic, can be reproduced by having the patient hyperventilate.[71,80] Frequently, psychiatric vertigo is associated with situational or environmental fears similar to agoraphobia.[80] Other features of patients with psychogenic dizziness include moment- to-moment fluctuations in symptoms; excessive slowness or hesitation; exaggerated sway with Romberg testing (often distractible); extreme caution with restricted steps (e.g., walking on ice); or sudden buckling of the knees usually without falling.[68] It has been proposed that psychiatric dizziness "should occur exclusively in combination with other symptoms as part of a recognized psychiatric symptom cluster, and this symptom cluster is not itself related to vestibular dysfunction."[80] Treatment for the underlying psychiatric disorder often improves the dizziness. Because this condition is usually related to anxiety and panic disorders, benzodiazepines are the mainstay of treatment.[64]

Disequilibrium

This can be a very difficult complaint to assess and treat. There are usually multiple factors involved, some of which are amenable to treatment, and some are not. Frequently, it is seen in the elderly population with multiple age-related issues contributing to the disequilibrium. Patients often complain of ill-defined dizziness and gait unsteadiness.[36] It often is associated with age-related decreases in vision and hearing, neuropathy, impaired proprioceptive input, and central compensatory mechanisms. Frequently, there is muscle mass decrease, decreased range of motion, and increased reflex time involved.[68] Cervical spondylosis may be associated with dizziness related to disturbance in postural control.[81] Frequently, there is some degree of postural instability. Anxiety is commonly a contributing factor but less likely in elderly patients to be the primary cause.[36,37] Many med-

ications prescribed can contribute to these symptoms (e.g., beta blockers, diuretics, sedatives). Central vascular and cardiovascular causes —especially carotid sinus hypersensitivity— are common.[36,82] Parkinson's disease must be ruled out. Thorough history and physical examination is required, but a diagnosis can be made in most cases.[82] Falls are a major risk for those patients with disequilibrium and occur four times as often as in controls.[17] Treatment is based upon the specific etiology that is found. Referrals for balance training and vestibular rehabilitation may be helpful.[36]

Presyncope

Presyncope was found as the cause in up to 16% of patients presenting with dizziness to EDs.[39] It is an impending feeling that one is about to pass out and is due to transient cerebral hypoperfusion. Occasionally, this is the prodrome to an actual syncopal event. Syncope often may be avoided if the patient assumes the supine position or the underlying etiology resolves (e.g., paroxysmal arrhythmia). The causes of presyncope are the same as for the causes of syncope. The most common broad categories of presyncope include neurocardiogenic (e.g., vasovagal), postural (e.g., orthostatic) hypotension, and cardiac causes (e.g., arrhythmias and valvular disease).[10] Frequently, etiologies for this symptom are not found. Neurocardiogenic causes account for about 50% of cases and may be precipitated by fear, pain, or emotional stress. There is an inappropriate vasodilation with relative bradycardia that leads to hypotension, lightheadedness, tunnel vision, diaphoresis, and diffuse weakness. It usually can be averted by assuming the supine position and elevating the legs.[83] Postural hypotension is defined variably. In general, there is a drop ($\geq$ 20 Torr) in systolic blood pressure and a drop ($\geq$ 10 Torr) in diastolic blood pressure within 3 minutes of standing. Reproduction of symptoms of lightheadedness is also important.[16] Many conditions can cause that including dehydration, anemia, medications (e.g., diuretics, beta blockers), and autonomic dysfunction (e.g., diabetes mellitus, Parkinson's disease). Frequently, there are multiple factors contributing to the individual patient's condition. Treatment of this condition requires finding a specific etiology and possible referral for tilt table testing.[10]

Conclusion

Dizziness can be a very challenging complaint for the EP to evaluate. In general it is due to benign, self-limited conditions. Unfortunately, there can be significant morbidity and disability even with these benign conditions. Frequently, the cause of dizziness can be a serious and life-threatening condition that requires prompt recognition and treatment for a favorable outcome. It is essential that the EP understand the pathophysiology of the different causes of dizziness. It is also imperative that the EP be able to take an accurate history and perform a thorough physical examination; these are the investigative tools that will allow the condition to be appropriately diagnosed and treated.

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Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

CME Questions

131. Peripheral vertigo is characterized by all but which of the following?
   A. Severe, acute onset of symptoms
   B. Nystagmus that fatigues
   C. Vertical nystagmus
   D. Falling to the same side of the lesion when ambulating

132. The cause of dizziness most often is found through which of the following means?
   A. History obtained from the patient
   B. Physical examination
   C. Intracranial imaging
   D. Electrocardiogram

133. Cerebellar infarction is associated with which of the following?
   A. Tinnitus
   B. Acute need for neurosurgical evaluation
   C. Falling to the side of the lesion when walking
   D. Positive (abnormal) Head Thrust (Head Impulse) test

134. Head or neck pain after a minor whiplash injury with vertigo is most often associated with what condition?
   A. Cerebellar hemorrhage
   B. Subdural hematoma
   C. BPPV
   D. Vertebral artery dissection

135. Vestibular neuronitis is treated with:
   A. Epley maneuvers.
   B. Semont maneuvers.
   C. a short course of vestibular suppressants
   D. bed rest.
   E. Both C and D.

136. Laboratory evaluation for the patient with suspected BPPV includes:
   A. H and H to rule out anemia.
   B. ECG to rule out dysrhythmia as etiology of symptoms.
   C. chemistry panel to rule out electrolyte disturbance.
   D. Laboratory studies are not indicated for suspected BPPV.

137. The treatment of choice for BPPV is?
   A. vestibular rehabilitation exercises
   B. Semont maneuvers
   C. Epley maneuvers
   D. meclizine and antiemetics

138. Vertebrobasilar insufficiency (VBI):
   A. is associated with severe occipital headache.
   B. presents with transient posterior circulation symptoms that resolve.
   C. must be associated with syncope to make the diagnosis.
   D. is associated with bilateral hearing loss.

139. Which one of the following statements about benign paroxysmal positional vertigo (BPPV) is true?
   A. It must be associated with reproducible nystagmus to make the diagnosis.
   B. If not corrected with a trial of Epley maneuvers, the diagnosis of BPPV is incorrect.
   C. If symptoms are not elicited with Dix-Hallpike maneuver, the diagnosis of BPPV is incorrect.
   D. It usually is associated with canalith of the posterior semicircular canal with free-floating otoconia in the right posterior semicircular canal.

140. Which of the following statements is *incorrect*?
   A. Rotatory nystagmus always indicates a peripheral cause of vertigo.
   B. Vertical nystagmus is always indicative of a central cause of vertigo.
   C. A few fine beats of nystagmus on extreme lateral gaze is normal.
   D. Changes of head position has no effect on central vertigo.

CME Answer Key

131. C
132. A
133. B
134. D
135. E
136. D
137. C
138. B
139. D
140. A

In Future Issues: **Alcohol withdrawal**

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Dizziness

Dix-Hallpike Maneuver

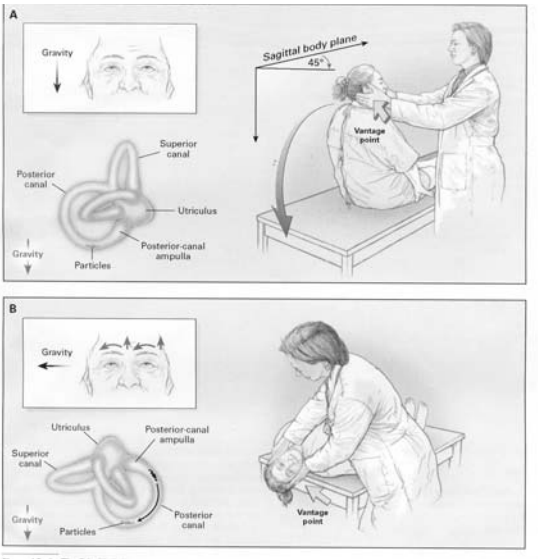

Figure. **A**.The patient assumes a sitting position far enough back on the stretcher so that when she lies back her head will extend over the end of the stretcher. The head is turned 45 degrees to one side and is supported by the examiner. **B.** The patient rapidly assumes the supine position with the head extended over the bed 45 degrees and turned 45 degrees to the side. The examiner should continue to support the patient's head. Reproduction of vertigo and nystagmus is diagnostic of BPPV. The maneuver is repeated with head turned to the opposite side if necessary.

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Differentiating Peripheral Versus Central Vertigo

	PERIPHERAL	CENTRAL
Onset:	Abrupt	Gradual
Intensity:	Moderate-intense	Mild
Duration:	Usually seconds-minutes	Weeks-months, but can last longer
Temporal pattern	Episodic	Chronic
Nystagmus:	Latency	No latency
Nystagmus-Direction:	Horizontal, rotatory Vertical	Horizontal, rotatory Dyscongugate
Nystagmus:	Fatigues	Does not fatigue
Changes in Head:	Worsens vertigo	No effect on vertigo
Associated neurologic:	None	Often present
Associated hearing:	May be present	Absent

Epley Canalith Repositioning Procedure

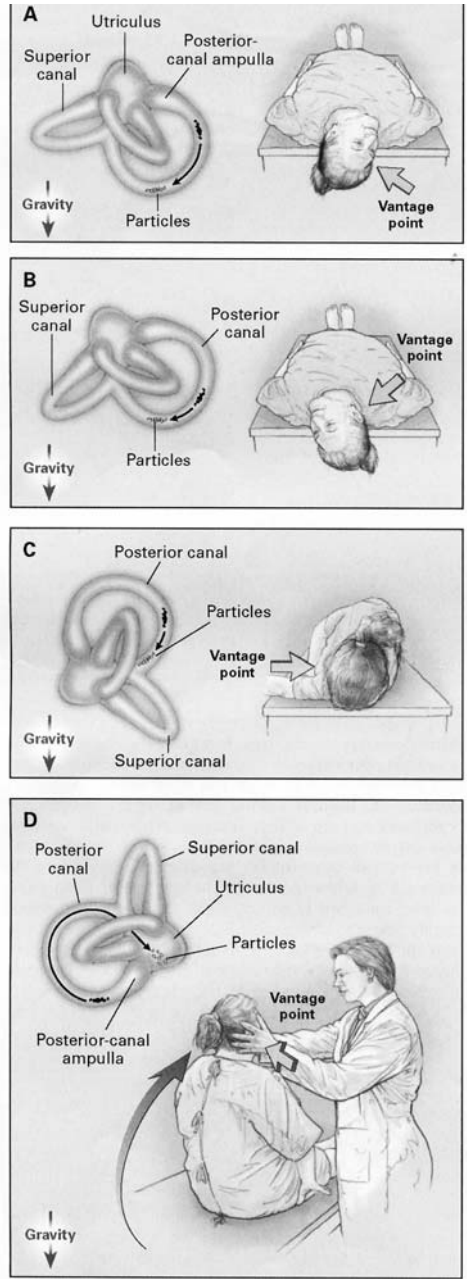

Figure. Performing the Epley Canalith Repositioning Maneuver. **A.** In similar fashion to performing the Dix-Hallpike maneuver, the patient assumes a sitting position far enough back on the stretcher so that when he lies back his head will extend over the end of the stretcher. The head is turned 45 degrees to the side that brought on vertigo and nystagmus when performing the Dix-Hallpike maneuver. **B.** As with the Dix-Hallpike maneuver, the patient is rapidly brought back to the supine position with his head extended 45 degrees over the edge of the stretcher and turned 45 degrees to the affected labyrinth (in this case the right). The patient remains in this position for about 30 seconds or until vertigo and nystagmus have resolved. Examiner should support the patient's head throughout the procedure. **C.** The head is then turned 45 degrees past the midline in the opposite direction. **D.** Again, after about 30 seconds or resolution of vertigo and nystagmus, the patient assumes the lateral decubitus position with the head looking down toward the floor. **E**. After 30 seconds or resolution of symptoms, the patient assumes the sitting position. **F**. The patient then places his chin to his chest. This procedure may be repeated as necessary.

Reprinted from: Furman C: Benign paroxysmal positional vertigo. New Engl J Med *1999:341:1590.*

Supplement to *Emergency Medicine Reports*, June 27, 2005: "Dizziness." *Author:* **Robert Frank, MD,** Clinical Instructor, Mercy Hospital of Pittsburgh**.** *Peer Reviewers:* **Steven M. Winograd, MD, FACEP,** Attending Physician, Emergency Department, Adena Regional Medical Center, Chillicothe, OH; and **Andrew D. Perron, MD, FACEP, FACSM,** Residency Program Director, Maine Medical Center, **Falmouth, ME.** *Emergency Medicine Reports' "Rapid Access Guidelines."* **Editor-in-Chief:** Gideon Bosker, MD. **Vice President and Group Publisher:** Brenda Mooney. **Editorial Group Head:** Glen Harris. **Specialty Editor:** Shelly Morrow Mark. For customer service, call: **1-800-688-2421.**

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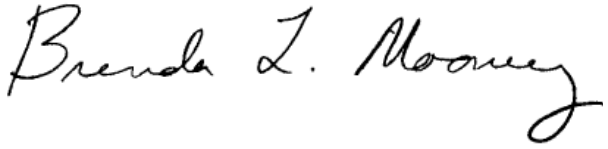

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Emergency Medicine Specialty Reports

Supplement S05179 **June 2005**

Informed Consent for Emergency Procedures

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Informed consent for medical treatment is an essential aspect of emergency medical care. The informed consent process is intended to assure the protection of the rights of patients. Numerous barriers to the informed consent process may exist among emergency patients, including impaired decisional capacity, impaired cognition, language barriers, illiteracy, insufficient time and communication, and numerous others. Because of the inherent vulnerability of ED patients, particular attention should be paid to addressing barriers to adequate informed consent, and steps should be taken to ensure adequate delivery of information, understanding of the proposed intervention and its risks and benefits, and voluntariness of the informed consent.

—The Editor

What Is Informed Consent?

The principle of informed consent is a fundamental principle of American ethics, recognized in 1914 when the New York State Supreme Court held that adults of sound mind have the right to make medical decisions.[1] The term "informed consent" requires both disclosure of information to the patient before the patient agrees to the proposed intervention, and voluntariness on the part of the patient to consent. Similarly, patients with decisional capacity also voluntarily may refuse a proposed treatment or procedure. Although physicians have the training and experience to make diagnoses and recommend treatment, it is accepted that the patient is most capable of deciding whether the proposed interventions are compatible with his/her value system and goals.

Informed consent is a fundamental right of patient autonomy in medical decision making.[2-9] Many emergency department (ED) procedures, such as intravenous lines and blood drawing, are considered routine, and are performed after general consent to treatment, agreed orally or in writing. Other more invasive procedures should be performed following a discussion with the patient regarding the procedure, purpose, risks, benefits, and alternatives to the proposed procedure or intervention.[10-12] Certain important exceptions exist to obtaining informed consent and will be discussed below.

In reality, compliance with informed consent discussions and documentation of informed consent, varies widely.[13] The American Medical Association (AMA) has a general policy that states, "Health care professionals should inform patients or their surrogates of their clinical impression or diagnosis; alternative treatments and consequences of treatments, including the consequence of no treatment; and

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This publication does not receive commercial support.

recommendations for treatment."[14] The American College of Emergency Physicians (ACEP) Code of Ethics States, "Emergency physicians shall communicate truthfully with patients and secure their informed consent for treatment, unless the urgency of the patient's conditions demands an immediate response."[15]

There are three basic types of informed consent. The first is express consent, when a patient specifically agrees to an intervention. The second type is implied consent, which is consent that is implied by the patient's conduct, for example, when a patient willingly holds his arm out to have blood drawn. Thirdly, consent implied in law refers to emergency treatment given to save life or preserve health, for patients unable to give consent in life-threatening situations.

In most cases, providing medical care to adult patients requires their consent. However, there are several important exceptions to the duty to obtain informed consent. In emergent situations, when the patient in unable to provide informed consent, and a surrogate decisionmaker is not available, if immediate treatment is indicated to prevent death or serious harm, appropriate medical interventions should be performed, under the presumption that a reasonable person would desire such. Other notable exceptions to the duty to obtain informed consent include: patients who waive their right to consent, patients who are unable to consent, certain public health or legal requirements, and the rarely used therapeutic privilege, in circumstances in which it is judged that the information would be so traumatic as to impair the patient's ability to consent.

The process of informed consent includes three important elements: determination of decisional capacity, delivery of information, and voluntary consent from the patient.

Emergency Medicine Specialty Reports is published by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Glen Harris
Specialty Editor: Shelly Morrow Mark
Marketing Manager: Nan Webb
GST Registration No.: R128870672

Periodical postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Specialty Reports***, P.O. Box 740059, Atlanta, GA 30374.

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Who Should Obtain Informed Consent?

The physician who will be performing the procedure should obtain informed consent from the patient.[16,17] Similarly, the physician involved has the ultimate responsibility for the discussion of the risks, benefits, and alternatives. The process of informed consent should not be delegated to others who will not be in attendance, or who may be unfamiliar with the intervention and its risks, benefits, and alternatives (e.g., residents, secretaries,)

How Should Decisional Capacity Be Assessed?

Decisional capacity refers to a patient's ability to make an authentic choice. Capacity includes cognitive and affective functions, including attention, intellect, memory, judgment, insight, language, emotion, and calculation. Decisional capacity includes the following four abilities on the part of the patient:

- Receive information;
- Process and understand information;
- Deliberate; and
- Make and articulate a choice.

Decisional capacity should be assessed on some level in all patients. Capacity is essential to the processes of informed consent and informed refusal of treatment.[18-20]

The assessment of medical decision-making capacity is an important skill for emergency physicians. In many cases, this can be accomplished by routine interactions with the patient who is alert, conversational, and appropriate. In such cases, physicians are able to determine a patient's capacity to participate in the informed consent process. In some cases, however, the determination of adequate decisional capacity can be challenging.

Decisional capacity may vary over time, as a function of host and environmental factors over time. Capacity is a dynamic, task-specific, and changing attribute, and may be viewed as a spectrum, pertaining to the particular healthcare decision at hand. Some impairments of capacity may be temporary, such as acute alcohol intoxication.

A stepwise approach may be used in the determination of decision-making capacity.[21] Essential elements of a suggested stepwise approach are summarized in Table 1. Standardized tests may be valuable in the determination of capacity.[22-29] One standardized test easily administered in the ED is the Mini-Mental Status Examination. (*See Table 2.*)[30] Typically, a score of 25-30 is acceptable for establishing appropriate mental status, 20-25 is borderline, and below 20 indicates serious impairment of mental status.

The formulation by the President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research is another standardized test that is well recognized and useful.[31]

Impaired decision making capacity may result from impairment in any of the four essential elements: impaired ability to receive, process, and understand information, deliberate, or to communicate a decision.

Numerous threats and barriers to capacity exist, many of which are reversible, or may change over time.[32,33] Although a detailed review of causes of altered mental status is beyond the

Table 1. Stepwise Approach to Determination of Capacity

1. Ensure ability to communicate.
2. Correct reversible environmental, metabolic, mental, and physical challenges to capacity.
3. Utilize standardized tests of competency, when appropriate.
4. Survey patient goals and values using open-ended questions about the choices (including risks and benefits), alternatives (including the option not to treat), and consequences.
5. Communicate with the patient and his or her health care advocates, if present.
6. Document essential elements of capacity or its impairment in the medical record.

scope of this document, some examples of clinical settings of impaired capacity may include patients with dementia,[34,35] intoxicated patients, psychiatric patients, patients of other cultures or languages, patients with physical communication impairments, severe pain, organic disease states,[36,37] and numerous other clinical settings. Every effort should be made to correct reversible etiologies of diminished capacity. Emergency physicians must be aware of potential barriers to capacity and must make an individual assessment of capacity in each case.[38-40] Even individuals who have some impairment of capacity may demonstrate sufficient understanding of the decision at hand to make an appropriate informed choice.

What Procedures Require Informed Consent?

There is tremendous variability among emergency physicians regarding the type of consent (verbal, written, or none) for many emergency procedures, including lumbar puncture, endotracheal intubation, arthrocentesis, venipuncture, and others.[41] In general, risks and benefits of all medical interventions should be discussed with patients. There is no national standard that dictates which procedures require written consent. Interventions with high risk, uncertain necessity, or other such factors may dictate written documentation of the discussion of the procedure, risks, benefits, and alternatives. Previous authors have suggested that specific written consent be obtained for a variety of procedures and interventions, including invasive procedures, HIV testing, radiographic procedures, treatment with controlled substances, and others.[42,43]

What Information Should Be Disclosed?

How much information should be presented to patients? Should every possible complication be detailed? Or might this frighten a patient, perhaps unnecessarily? The balance of the right amount of information to be delivered can be challenging to discern. Certainly sufficient information to weigh the risks and benefits should be presented. However, an overwhelming amount of information may be confusing or even intimidating to patients.

The "reasonable person" standard of disclosure counsels that sufficient information should be disclosed that would be "material" to a reasonable person to make a decision in that particular case. Needless to say, a judgment about what information is considered "material" can be very subjective. "Material" risks are those considered significant for decision making by a reasonable person. Although most states favor the "reasonable person" disclosure standard, some states utilize the "professional standard," which indicates that the physician should disclose what other physicians in the community would disclose in similar circumstances. Risks that are considered very remote or those that are commonly known, need not be disclosed in every case.

Should Status as a Resident or Medical Student Be Disclosed?

In most teaching institutions, general consent to treatment includes basic information about being treated by residents, interns, and students under appropriate supervision. However, many patients are unaware of the training status of many health care professionals.[44,45] General ethical principles dictate that patients should be informed about the identity and training level of health care providers participating in their care. However, disclosing details about previous training and experience is less clear. The issues of patient autonomy regarding health care and necessity to train health care professionals must be balanced.

Many patients are willing to be treated by students and residents, if this information is disclosed.[46] Many patients are reluctant to have students perform a procedure for the first time on them, and many believe that they should be informed if the resident or student is performing a procedure for the first time.[47,48]

What About Patients Who Refuse to Consent?

Just as all competent patients have the right to participate in the informed consent processs prior to medical interventions, patients also have the right to refuse medical care. Refusal of care may occur at several levels, including general treatment, hospital admission, or specific tests or therapies. As with informed consent, informed refusal is a process, not merely a signature on a form documenting that the patient is leaving against medical advice, or an AMA form. The process should similarly consist of determination of decisional capacity, delivery of information, including risks of refusing treatment, and documentation of the process. When a patient refused medical treatment, great care should be taken that the patient understands the consequences, and that the physician remains willing to treat the patient.

In some circumstances, patients should be treated appropriately even without informed consent, or even if they specifically refuse care. Examples of treatment without consent include patients who lack decisional capacity, patients with public health risks, and patients who pose an immediate threat to the health or well-being of themselves or others. Medical treatments necessary to prevent potential loss of life, limb, or impaired quality of life should be undertaken in such circumstances.

Table 2. Mini-Mental Status Examination

	Score	Maximum Score
ORIENTATION		
What is the (year) (season) (date) (day) (month)?	____	5
Where are we? (state) (county) (town) (hospital) (floor)	____	5
REGISTRATION		
Name three objects and ask patient to repeat.	____	3
ATTENTION AND CALCULATION		
Serial 7s (one point for each correct up to 5) Option: Spell "world" backward.	____	5
RECALL		
Ask for the 3 objects repeated above.	____	3
LANGUAGE		
Name a pencil and watch. (2 points)		
Repeat "no ifs, ands, or buts." (1 point)		
Follow a 3-stage command. (3 points)		
Read and follow the command "Close your eyes." (1 point)		
Write a sentence. (1 point)		
Copy a design. (1 point)	____	9

When caring for patients, the safety of staff and patients is a primary objective.[49,50] As a result, some patients may need to be physically restrained without their consent to assure safety of the environment. The use of physical restraints should be limited to cases in which the safety of the patient, other patients, or staff is threatened, such as for patients who are violent or suicidal patients who pose an immediate elopement risk. Restraints should be used in the least restrictive manner possible, and for the least amount of time necessary to achieve the desired goals. According to policy set by the American College of Emergency Physicians (ACEP), "restraints should be individualized and afford as much dignity to the patient as the situation allows" and "any restraints should be humanely and professionally administered." Additionally, this policy states that protocols should ensure appropriate observation, treatment, assessments, and documentation.[51] Restraints should never be used for competent patients who refuse medical therapy.[52]

Though every recommendation a patient chooses to refuse should be accompanied by an informed refusal discussion, every refusal need not be accompanied by an AMA form. When the dangers to the health of the patient from refusal are great, and a reasonable patient would follow the course of action the physician recommends (e.g., treatment and hospitalization for a probable myocardial infarction), but this particular patient who is decisional is not following the physician's recommendation, an AMA form can act as an important documentation of the refusal to the physician and the information that the patient understood before refusing treatment.

What About Minors?

Unlike adults, who generally are deemed capable of making their own health care decisions, minors generally do not have this right legally recognized. Under most circumstances, parents have the right to consent or refuse medical care for their minor children. Many states allow adolescent minors some limited autonomy, often specifically in the areas of contraception, treatment for sexually transmitted disease or substance abuse without parental permission. State laws vary widely, and emergency physicians should be aware of the state laws where they practice. In many states, "emancipated minors" are considered competent to make their own healthcare decisions. Emancipated minors often are defined as minors who are married or living independently and supporting themselves, in the military, or who are pregnant or have children. In addition, case law in some states on the "mature minor" now recognizes that as the adolescent's age increases toward maturity, he or she can have a progressively greater part in the decision-making process for important medical decisions.

Many pediatric and adolescent patients present to the ED without parent or guardian to consent for treatment (approximately 2-3% of pediatric ED patients).[53] In such circumstances, "Appropriate medical care for the pediatric patient with an urgent or emergent conditions should never be withheld or delayed because of problems with obtaining consent."(This statement is endorsed by The American College of Emergency Physicians, American College of Surgeons, Emergency Nurses Association, and National Association of EMS Physicians).[54] In addition, the Emergency Medical Treatment and Active Labor Act (EMTALA), federal law, requires that a screening examination for an emergency medical condition be performed for all patients presenting to emergency departments, including minors, with or without parental consent.

Parents may in some cases refuse care for their minor children. In some circumstances, the state allows medical personnel to deliver emergency medical care to children, even if parents refuse care, because of the state's interest in the well-being of the child. In such circumstances, physicians can and should treat emergency medical conditions of minors, even if the parents object.[55,56] In general, a court order should be obtained, if it would not inappropriately delay treatment.

Should Religious and Moral Issues Be Considered?

Some patients may refuse medical interventions because of religious or moral reasons. One example is the Jehovah's Witness patient who refuses blood transfusions. If considered to have

appropriate decisional capacity, adult patients can agree to or refuse treatment based on religious or moral considerations or other personal reasons, even if it results in adverse outcomes, including death.

What About Mandatory Reporting?

Certain clinical situations warrant the disclosure of protected health information (PHI) without specific consent from patients. Situations in which the disclosure of PHI is permissible include when required by law (court order, statute, or regulation), FDA reporting, certain communicable diseases, and certain work-related illnesses or injuries. Additionally, release of PHI may be required or permitted by law in cases of suspected neglect, abuse, or domestic violence.

According to the Health Insurance Portability and Accountability Act of 1996 (HIPAA), disclosure to law enforcement officials without consent may be permissible in some circumstances, including in response to court orders, warrants, or subpoenas, to assist in the identification or location of a suspect, fugitive, witness, or missing person, or when responding to a law enforcement official's request for information about a crime victim. Disclosure also is permissible when a person's death may be the result of criminal activity, when PHI may be evidence of a crime that occurred on hospital property, or when necessary to inform officials about the nature of a crime, the location of victims, or the perpetrator.[57]

Physicians have a duty to warn individuals or groups when information is available that indicates that there is a significant risk posed by a patient or by information divulged by a patient, without specific consent from the patient. This duty to warn has been upheld in multiple legal cases by the U.S. courts. National policies including those written by the AMA, ACEP, and language in HIPAA also recognize that disclosure of PHI may be appropriate in cases in which there is a potential threat to the public or to an individual.[58-62]

Individual states regulate the reporting of certain conditions as reportable to public health officials. Although there is some variation in conditions that mandate reporting, certain conditions commonly are recognized as reportable conditions. Examples of statewide reportable conditions include traffic crashes, penetrating trauma, residential fires, occupational injuries, suicide prevention, falls, poisoning, violence, and drowning. The Centers for Disease Control and Prevention (CDC) maintains a Public Health Information Network, which contains data regarding national reportable conditions.

What About Informed Consent for Research?

Informed consent should be obtained from potential research subjects prior to participation in medical research. In some cases, verbal consent is sufficient, but in most cases, written informed consent is appropriate. Informed consent documents for research should be worded appropriately, understandable, and should include an explanation of the purposes of research, duration of participation, description of the study, risks, benefits, alternates, confidentiality, compensation, and information about voluntary participation.[63-68] Informed consent documents and discussions should be written and delivered at a reading level appropriate to the potential subject. This may require some individual adaptation.

There exist many unanswered questions about the ideal informed consent process. Several recent studies demonstrate that research subjects' understanding of detailed informed consent is poor.[69-71] Several recent reports indicate that a majority of patients prefer detailed information compared with abbreviated information.[72-74] Another study demonstrated improved information retention with a short form compared with a more detailed form.[75] Although many informed consent documents are written at an inappropriately high reading level,[76-78] careful attention to the written informed consent document and its linguistics can improve its readability.[79]

The same process of informed consent should be utilized in research settings that is used in clinical settings. Decisional capacity should be assessed, information should be delivered, and the patient should voluntarily agree to participate. A detailed discussion with the potential subject, including feedback from the subject regarding his or her understanding of risks and benefits, may elucidate the level of understanding and provide opportunity for additional education.

Coercion of potential research subjects, either overt or masked, is unethical. Although most researchers agree with this in principle, many continue to coerce subjects in subtle ways. Examples of inappropriate coercion include excessive monetary incentives, failure to inform the subject of voluntariness of participation, repeated questioning, inappropriate representation of the study benefits, withholding of care prior to consent, and numerous others. Any form of coercion must be avoided.

How Should Informed Consent Be Documented?

Some erroneously believe that a patient signature on an informed consent document is sufficient. Informed consent is a process, with necessary elements, including the discussion between the physician and patient, delivery of important information, including a description of the intervention, risks, benefits, and alternatives, and the patient's agreement to the proposed intervention. The piece of paper with signatures does not comprise informed consent and is not a substitute for the discussion. However, such signature does provide evidence of the patient's indication that the informed consent process did take place. Additionally, documentation of the informed discussion, information delivered, patient's decisional capacity, and the patient's consent to the intervention all should be documented in the medical record.

Conclusions

Informed consent for procedures in emergency medicine is an essential element of the protection of human subjects' rights, yet it remains a complex and challenging process. Decisional capacity should be evaluated, and if appropriate, the physician should communicate with the patient regarding the proposed interven-

tion, risks, benefits, and alternatives, and the patient's goals and values of therapy. If the patient voluntarily agrees, the intervention may be pursued. The patient encounter and important aspects of the communication should be documented in the medical record.

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Physician CME Questions

To participate in this CME program, read the article and then answer the questions below. Review any material related to missed questions. ***In order to receive credit for this activity, you must complete the evaluation form enclosed with this issue.*** *When your completed form has been received, a certificate will be mailed to you.*

1. Which principle of bioethics best relates to the principle of informed consent?
   A. Autonomy
   B. Beneficence

C. Nonmaleficence
D. Justice

2. Who should obtain informed consent from the patient?
   A. Nurse
   B. Primary care physician
   C. Physician performing the procedure
   D. Secretary
   E. Resident

3. Which of the following conditions allows a physician to treat the patient despite his or her refusal?
   A. Possibility of bad outcome
   B. Suicidality
   C. Life-threatening medical condition
   D. Psychiatric diagnosis

4. How much information should be disclosed when obtaining informed consent?
   A. All information available in the medical literature
   B. Only information requested by the patient
   C. Only information requested by the family
   D. Information desired by a reasonable person

5. If a patient has decisional capacity and refuses life-saving medical intervention, what is the best course of action?
   A. Allow the patient to refuse care
   B. Restrain the patient to administer life-saving medical care
   C. Obtain consent from the next of kin
   D. Obtain consent from two physicians for emergency care

6. What is the most essential element of informed consent?
   A. Signature on the informed consent document
   B. Discussion with the physician and patient
   C. Documentation in the medical record
   D. Witness' signature on the informed consent document

7. What is implied consent?
   A. A patient signs the general consent for treatment.
   B. A patient signs a specific informed consent document.
   C. A patient indicates by actions that he/she agrees with the intervention.
   D. A surrogate agrees to the intervention in place of the patient.

8. What is decisional capacity?
   A. The patient's ability to make an authentic choice
   B. The patient's legal competence
   C. The surrogate's ability to consent in place of the patient satisfaction
   D. The patient's mental capacity

9. Which of the following should be included in the written consent for research?
   A. Relevant articles from the medical literature
   B. Names of other study participants
   C. Preliminary results of the research project
   D. Purpose of the research project

10. Which of the following statements is true regarding the use of physical restraints?
   A. Informed consent from the patient is required prior to use.
   B. Restraints may be required for the protection of staff.
   C. Restraints should be used for as long a period as possible.
   D. Restraints may be used to restrain competent patients who refuse medical care.

CME Answer Key

1. A
2. C
3. B
4. D
5. A
6. B
7. C
8. A
9. D
10. B

Emergency Medicine Specialty Reports

CME Objectives

At the conclusion of this activitiy, participants should be able to:

- Discuss current laws relating to informed consent
- Explain medical ethical standards relating to providing informed consent
- Share processes involved in providing informed consent.

In Future Issues:

Evaluation of Elder Abuse

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The objectives of this issue of *Emergency Medicine Specialty Reports* are to:

1. Discuss current laws relating to informed consent
2. Explain medical ethical standards relating to providing informed consent
3. Share processes involved in providing informed consent.

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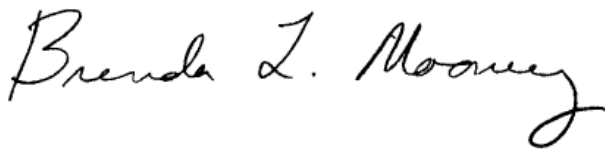

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