Dizzy and Confused: A Step-by-Step Evaluation of the Clinician's Favorite Chief Complaint

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KEYWORDS

• Dizziness • Vertigo • Altered mental status • Evaluation

Few chief complaints can provoke more of a sense of fear and loathing in the emergency physician than that of dizziness. The differential diagnosis is extensive and the symptoms and signs are frequently vague and hard to define. Moreover, life-threatening illnesses may masquerade as benign conditions and tests ordered to screen for such illness are often insensitive.[1] Dizziness ranks among the most frequent complaints leading to outpatient evaluation, and is the single most common complaint among patients older than 75 years of age.[2,3] Evaluating patients with a complaint of dizziness presents a significant challenge to the emergency physician, especially in the setting of patients with altered mentation. However, a systematic approach will produce good patient care and avoid considerable consternation on the part of the physician.

DEFINITIONS

Dizziness is an imprecise complaint that encompasses many and varied diagnoses, including syncope, pre-syncope, lightheadedness, gait instability, nausea, anxiety, or generalized weakness. Vertigo is a type of dizziness and has been defined as, "the sensation of motion when no motion is occurring relative to earth's gravity".[4] Vertigo occurs with asymmetric provocation of the vestibular system and is often described as a sensation of spinning relative to the patients' environment or vice-versa.[5] Vertigo can be further divided into central and peripheral depending on what part of the

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vestibular system has triggered the patients' symptoms. The semicircular canals, otoliths, and vestibular nerve (cranial nerve eight) can all cause peripheral vertigo, whereas the vestibular nuclear complex, vestibulocerebellum, brainstem, spinal cord, and vestibular cortex are all potential causes of central vertigo. Even when patients describe their symptoms specifically as vertigo, it is advisable to clarify their complaint, because many patients are not familiar with the exact meaning of many medical terms.

EPIDEMIOLOGY

It is estimated that 7.5 million patients with dizziness are examined each year in ambulatory centers, and it is one of the most common chief complaints in the emergency department (ED).[5] The lifetime prevalence of vertigo in adults aged 18 to 79 years is 7.4%, with a clear increase in prevalence with age. With the advancing age of North America's population, dizzy and vertiginous patients will continue to be frequent visitors to our EDs.

APPROACH TO PATIENTS WITH DIZZINESS

Clarifying the nature of the complaint is critical because evaluation and management will differ dramatically depending on the history obtained. Generally, the approach to patients with dizziness begins with delineating whether or not patients have true vertigo. Such clarity will allow the physician to limit the scope of the workup and focus on conditions most relevant to the patients' presentation. Patients presenting with symptoms and signs of vertigo will need further evaluation to determine if it is central or peripheral in origin. To this end, a good history and physical examination are invaluable tools that will frequently lead clinicians to the correct diagnosis.[6,7] Alternatively, patients whose dizziness is not vertigo will need investigation into potential cardiovascular, infectious, toxicologic, or psychiatric etiologies of their symptoms.

HISTORY

Traditional teaching has recommended that the physician obtain clarification by asking patients to describe their symptoms without using the word dizziness. Although it is sometimes useful to get a good description with precise terms, patients' particular description can be unclear, inconsistent, or unreliable.[8] Therefore, while obtaining the history, the physician should encourage patients to focus on the timing, triggers, and progression of the symptoms.[8] Despite its limitations, a careful history is usually sufficient to distinguish between central and peripheral vertigo.[5] If the history is consistent with nonspecific dizziness, weakness, or gait instability, then evaluation should proceed in a manner most suitable to those complaints.

The onset of symptoms, whether abrupt or indolent, can be an important distinguishing feature. A slow indolent onset is characteristic of central vertigo. The duration of symptoms can be weeks to months and is characterized by the gradual progression of the overall disease process. Historically, much emphasis has been placed on whether the symptoms worsen with movement of the patients' head. However, most people experiencing dizziness or vertigo of any etiology will experience worsening symptoms with movement. The important distinction is whether the symptoms are *triggered* by head movement or merely worsened by it. Patients experiencing central vertigo may report worsening of their symptoms with head movement, but they should not report that symptoms are *triggered* by it. Finally, central vertigo is frequently associated with neurologic deficits, nystagmus, or visual field abnormalities.

In contrast, peripheral vertigo is characterized by the sudden onset of symptoms that are often severe. Symptoms are episodic and typically last from seconds to

minutes with symptom-free intervals between attacks. Patients can frequently trigger attacks by movement of their heads, but can alleviate symptoms by lying still. Unlike central vertigo, peripheral vertigo is not associated with focal neurologic deficits or visual field abnormalities. Peripheral vertigo can be accompanied by a sensation of fullness in the affected ear. In some cases, hearing loss or tinnitus is reported. Careful history may localize the auditory symptoms to one ear, and usually the symptomatic ear is ipsilateral to the vestibular damage.[9] During an acute vertiginous attack, nausea and vomiting are quite common.

Inquiries into the patients' past medical history may be beneficial in determining the cause of the symptoms, especially if there is a history of cancer. Such a finding could heighten one's suspicion of a central nervous system (CNS) metastasis being the cause. Prior ear surgery, vestibular dysfunction, or recent trauma would also be pertinent history.

A social history can be illuminating, especially a history of significant alcohol intake. Chronic alcohol abuse can trigger peripheral neuropathy and resulting gait instability or foot drop that patients often describe as dizziness. Additionally, chronic alcohol abuse should raise the suspicion of vitamin deficiencies and Wernicke's encephalopathy, which classically presents with ophthalmoplegia, ataxia, and confusion. Review of patients' medications is also important because prescription medications can cause dizziness through direct ototoxicity or through a lowering of blood pressure, which may lead to diminished perfusion of the CNS. Though increasingly rare, tabes dorsalis (syphilitic myelopathy) can develop in untreated tertiary syphilis and cause gait instability secondary to proprioceptive abnormalities. Therefore, it is prudent to ask about prior exposure to sexually transmitted diseases. Finally, psychiatric illnesses, especially depression or anxiety, can manifest as almost any complaint, including dizziness, and should be considered as a potential cause of patients' symptoms.

PHYSICAL EXAMINATION

After obtaining a thorough history, the physical examination can often be used to confirm the suspected diagnosis. Patients' vital signs should not be overlooked because they may suggest infectious etiologies or cardiovascular instability as potential causes of dizziness and altered mentation.

Particular attention should be focused on the eye examination, where extraocular motion must be thoroughly tested. The presence of nystagmus is notable. If present, one must determine whether the nystagmus is vertical or horizontal, whether or not it is fatigable, and if it is triggered by a particular head movement. Nystagmus is characteristically vertical and indefatigable in central vertigo. Additionally, visual fields and conjugate gaze should be tested. Internuclear ophthalmoplegia, classically seen as unilateral nystagmus elicited by conjugate lateral gaze, can frequently be found in patients with CNS infarction or multiple sclerosis.[10] The ears should be examined for evidence of trauma or middle ear infection. If the history suggests hearing loss or tinnitus, a full auditory evaluation is prudent. The examination continues with the cranial nerves, with tests for symmetry and function. A deficit in ipsilateral cranial nerves VII and VIII is strongly suggestive of a central lesion.

Extremity strength, sensation, and deep tendon reflexes should be assessed. Gait should likewise be tested in patients who are able to stand. Pronator drift, finger-to-nose, and Romberg tests are useful for evaluating proprioceptive or cerebellar disorders. However, if patients are markedly symptomatic, these maneuvers may prove difficult and have questionable utility. Focal neurologic deficits discovered on the neurologic examination may be indicative of ischemic stroke or other central

pathology. The cardiovascular system should be examined for carotid bruits or an irregular heart rhythm, which would raise the possibility of an embolic event.

Though no bedside examination maneuver is diagnostic, the head thrust maneuver deserves special mention. It assesses patients' vestibular ocular reflex (VOR) and can accurately distinguish cerebellar stroke from benign peripheral conditions, such as vestibular neuritis. The head thrust maneuver is performed by placing the examiner's hands on each side of the patients' head. Quick head movements are then made 20 to 30 degrees to the patients' left and right while attempting to focus on a fixed object (usually the examiner's nose). The response is abnormal if the patients' eyes move with their head and then snap back to the examiner's nose in a single corrective movement. Such an abnormal response is suggestive of a disruption of the VOR and implicates a peripheral cause of the vertigo, such as vestibular neuritis. A normal response to this maneuver is for patients' eyes to remain fixed on the object despite the head movements and suggests a normal intact VOR. Patients with cerebellar infarction give a normal response because the VOR bypasses the cerebellum. Therefore, in acutely vertiginous patients, a normal response is strongly suggestive of cerebellar stroke even in the presence of normal neuroimaging. In cases of suspected vestibular neuritis, an abnormal head thrust test, horizontal nystagmus, and the absence of vertical ocular misalignment can exclude 91% of acute strokes.[1]

Table 1 summarizes the characteristics of central and peripheral vertigo. Although each characteristic generally pertains to one or the other type of vertigo, no characteristic is diagnostic or exclusionary for either type. A thorough history should touch on each of these characteristics and more often than not, a clear trend will become apparent and the differential diagnosis will be narrowed considerably.[6] Although most conditions causing peripheral vertigo are benign and do not require emergent treatment or stabilization in the ED, patients with altered mentation and vertigo are not typical of this disease process and require careful evaluation.

VERTIGO

Vertigo is a symptom that generally has a benign underlying cause. However, in patients presenting with altered mental status, vertigo should never be assumed to have a trivial cause. The type of vertigo, along with other neurological deficits or signs, can be important clues for physicians investigating patients' illness.

A 62-year-old man with hypertension and diabetes presents complaining of dizziness and gait instability. He has no history of these symptoms and notes that they are present

Table 1
Characteristics of central and peripheral vertigo

Characteristic	Central	Peripheral
Onset	Slow	Sudden
Frequency	Constant, progressive	Episodic, recurrent
Duration	Weeks to months	Seconds to minutes
Changes in head position triggers?	No, symptoms may worsen but are not triggered by motion	Yes, symptoms triggered by motion and alleviated by being motionless
Nystagmus	Vertical	Horizontal
Associated symptoms	Neurologic or visual deficits	Tinnitus, nausea
Fatigable	No	Yes

at rest or with movements of his head. His wife reports he has fallen at home several times and is concerned. On examination he is noted to have indefatigable horizontal nystagmus, a normal head thrust maneuver, and a positive Romberg sign.

CEREBELLAR STROKE

Although many strokes located in the anterior or middle circulations do not present a diagnostic dilemma, diagnosing an acute cerebellar infarction can be quite challenging. The most common symptoms, dizziness, vertigo, and gait instability, are nonspecific and frequently have benign underlying causes.[1] Moreover, CT of the brain frequently does not detect acute infarctions in the posterior fossa. Misdiagnosis can be problematic when and if clinicians fail to recognize this limitation.[1] Once again, clinicians must rely on a thorough history and physical examination to help guide the evaluation and ultimate disposition of patients.[1,11]

Approximately 20,000 new cerebellar strokes are diagnosed in the United States each year.[1] In nine studies of consecutive subjects with ischemic strokes, cerebellar infarction accounted for approximately 3% of strokes. General risk factors for ischemic stroke, including hypertension, diabetes, tobacco usage, dyslipidemia, atrial fibrillation, and history of ischemic stroke or transient ischemic attack (TIA), likewise apply to cerebellar stroke.

Patients often present with nonspecific symptoms, such as dizziness, nausea, vomiting, unsteady gait, or headache, when acute infarction is limited to the cerebellum. Neurologic signs, such as dysarthria, ataxia, or nystagmus, may be subtle or absent. Although dizziness or vertigo occurs in nearly three quarters of patients with cerebellar infarction, they are also common complaints in many benign disorders. Indeed, an isolated complaint of dizziness is a poor predictor of stroke of any kind. In a retrospective study of 1660 subjects presenting to the ED with a chief complaint of dizziness, only 3% had a stroke or TIA; and of subjects presenting with an isolated complaint of dizziness, less than 1% were found to have had an ischemic stroke or TIA.[12]

Signs of cerebellar stroke can include limb or truncal ataxia, dysarthria, and nystagmus. Though confusion can be present, it has been noted in only about one third of patients with cerebellar stroke.[1] Coma is even less likely to be present, and usually indicates basilar artery occlusion or secondary complications, such as brainstem compression or herniation. In patients experiencing vertigo, the VOR can be assessed rapidly at the bedside by using the head thrust maneuver (described earlier). A normal response is strongly suggestive of cerebellar stroke, whereas an abnormal response implicates a peripheral cause of vertigo. No one feature is diagnostic, and it is incumbent upon the clinician to interpret each feature in the context of the entire clinical encounter.

Neuroimaging is crucial in the evaluation of suspected cerebellar stroke. Head CT is the most readily available form of neuroimaging for emergency physicians, but has significant limitations. It is uncommon to visualize acute stokes in the posterior cranial fossa with CT. Therefore, in patients with suspected cerebellar or brainstem stroke, CT scan should not be used to exclude this diagnosis.[1,11,13] One study suggests that misdiagnosis occurs more frequently in junior physicians who place too much emphasis on the role of CT in excluding acute stroke.[14] MRI has greater sensitivity than CT, especially when diffusion weighted imaging is used, but is not readily available to many emergency physicians. Given these constraints, CT scan is the preferred initial screening test.[11,13]

In patients with suspected acute brainstem or cerebellar stroke, admission and neurologic consultation is warranted for definitive neuroimaging and treatment, even in the

presence of negative head CT scan. Antiplatelet agents should be withheld until neuroimaging has been completed and the physician is satisfied that no hemorrhage is present.

An 80-year-old women presents by ambulance to the emergency department with report of sudden onset left-sided facial weakness, disorientation, and right-sided extremity weakness. Family members note abnormal slurred speech and disorientation. On examination, the patient is noted to have nystagmus and ataxic limb movements.

VERTEBROBASILAR STROKE

Vertebrobasilar strokes usually cause more neurologic abnormalities than isolated cerebellar infarctions because a larger area of the brain is affected by occlusions of the posterior circulation. Headache, dizziness, vertigo, or confusion are all possible presenting complaints.[15] Physical examination findings include, but are not limited to, pupillary abnormalities, abnormal ocular movements, facial palsy, and hemiplegia or quadriplegia.[15]

Evaluation and workup should follow the same basic guidelines outlined previously with respect to history and physical examination, followed by appropriate neuroimaging and admission for suspected acute infarction. Again, physicians should be cognizant of the limitations of CT for diagnosis of acute infarctions in the posterior cranial fossa.

An infrequent but notable link between cervical manipulative therapy (CMT) in chiropractic practice and vertebral artery dissection has been demonstrated.[16,17] History of antecedent CMT in the face of neurologic findings should raise suspicion for this entity. Diagnosis can be made with CT angiography, conventional angiography, or MRI angiography.

A 75-year-old man presents with gradual onset of disorientation, worsening dizziness, and falling over the past several months. His family noted these symptoms were subtle at first and thought they were caused by the patient's advancing age. However, the family notes increasing difficulty walking and a broad based shuffling gait. He has a history of being treated for urinary incontinence and recently had a Foley catheter placed.

IDIOPATHIC ADULT HYDROCEPHALUS SYNDROME (NORMAL PRESSURE HYDROCEPHALUS)

Normal pressure hydrocephalus (NPH) is a syndrome of gait dysfunction and enlarged cerebral ventricles in the absence of another cause. Contrary to its traditional name, however, intracranial pressure is not always normal and a more accurate name is idiopathic adult hydrocephalus syndrome.[18] The more familiar term, NPH, is used in the following discussion. NPH usually presents in the sixth or seventh decade of life, and although the incidence increases with age it is still considered a rare diagnosis.[18,19]

The classic triad of abnormal gait, dementia, and urinary incontinence is well described. However, because NPH generally occurs in the elderly population, these findings are not uncommon in patients with other illnesses. Patients will frequently use the terms dizziness or weakness to describe gait instability or inability to ambulate normally. Abnormal gait is characteristically the first and most prominent finding and occurs in 89% of people diagnosed with NPH.[19] The gait is typically wide based with reduced step height and stride length, along with reduced velocity and imbalance or unsteadiness. With time, gait impairment progresses so that patients find it difficult to initiate walking. This condition is described as a ''magnetic gait'' because patients' feet seem stuck to the floor as if magnetized.[18,19] Other conditions common in this age group, such as peripheral neuropathy, parkinsonism, and lumbar canal stenosis, can

also cause gait abnormalities and should be considered when evaluating patients with a complaint of dizziness or abnormal gait.

An estimated 45% to 90% of patients with NPH experience urinary incontinence.[19] Urinary frequency and urgency are the earliest manifestations of NPH, and are caused by stretching of the periventricular nerve fibers that control the urinary bladder. This stretching leads to partial loss of bladder contraction inhibition and progressive urge incontinence.[19] Other causes of urinary incontinence should be considered, such as prostatic hypertrophy, stress incontinence, neuropathy, or anticholinergic effects of medications.

Up to 77% of patients with NPH develop dementia, which is described as subcortical.[19] Typical findings are memory impairment, with decreased attention, alertness, and speed of mental processing. Cortical deficits of aphasia, apraxia, and agnosia are typically absent in NPH and may point to an alternative diagnosis,[19] such as depression with pseudodementia, Alzheimer's disease, or vascular dementia.

The diagnosis of NPH can be made by the astute examiner with the aid of either CT or MRI of the brain. Neuroimaging reveals ventriculomegaly, which is distinct from hydrocephalus ex vacuo, a common normal finding in elderly patients. Patients admitted with suspected NPH benefit from neurologic and especially neurosurgical consultation for assessment of potential shunt placement. NPH has variable resolution with shunt placement, but is one of the few reversible causes of dementia in those patients who do respond to treatment.

A 25-year-old woman presents to the emergency department with 2 years of intermittent motor and sensory complaints. In her most recent episode, she experienced frank vertigo. She has been seen by several physicians for complaints ranging from foot drop and dizziness to paresthesias and generalized fatigue. Several laboratory and diagnostic tests have been run and her primary care physician suggested she start treatment for anxiety and stress. On examination, she is noted to have internuclear ophthalmoplegia.

MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is a chronic inflammatory disorder affecting the brain, spinal cord, and optic nerve that typically presents in young adults aged between 20 to 45 years.[20–22] By definition, MS is characterized as having multiple episodes of inflammation disseminated in time and space.[20,21] Because MS can affect nearly any part of the CNS, presenting symptoms are highly variable and are often confused with other disease processes.[21]

Vertigo is the initial symptom in approximately 5% of patients diagnosed with MS and affects 50% of patients with MS at some point during the course of their disease.[5] Other prominent presenting symptoms are sensory complaints, such as frank numbness, or paresthesias. Sensory symptoms are typically vague and patients will often have a normal sensory examination. The discordance between the history and physical examination can lead to falsely attributing patients' symptoms to anxiety or emotional distress.[21]

Motor symptoms typically involve weakness or spasticity, but can also involve complaints of diminished hand dexterity or gait instability. Gait abnormalities can develop from weakness, dizziness, incoordination, sensory loss, or a combination of these problems.[20] Finally, fatigue is reported in up to 90% of patients with MS and may refer specifically to exertional muscle fatigue or general lassitude.[21]

MRI is an important tool that assists with the diagnosis of MS. Images of the brain can demonstrate multiple T2 signal abnormalities in the cerebral white matter.

Although other modalities, such as spinal fluid analysis, serologic testing, and evoked potentials, can be helpful to the neurologist, they are not available during a single ED encounter. Because MRI is not routinely available in many EDs, it is recommended that the clinician evaluate patients for other disease processes that could mimic MS and arrange follow-up for definitive testing.

A 35-year-old man presents with complaints of gradually worsening vertigo for the last several months. Although his symptoms wax and wane in intensity, they have recently worsened and he notes no symptom-free intervals. He also notes a painful occipital headache. His wife notes hearing loss specifically to the right ear. On physical examination, signs of cerebellar ataxia are noted when testing the patient's gait and coordination.

SPACE OCCUPYING LESIONS

Dizziness may be one of a variety of signs and symptoms of a CNS tumor, depending on the size and location of the tumor within the brain. Typically, the onset of symptoms is insidious, gradually worsening with tumor growth as adjacent structures are compromised. However, it can be more acute if the tumor undergoes acute infarction or bleeding.

Tumors of the cerebellopontine angle and posterior fossa are characterized by the accumulation and augmentation of neurologic signs and symptoms over weeks to months.[5] Symptoms include vertigo, dizziness, hearing loss, tinnitus, or facial weakness or numbness. Occipital headache is the most common symptom with cerebellar tumors.[5] Ataxia, nystagmus, or hearing loss can also be seen on examination. Late in the course of disease, cranial nerve palsies or vomiting when recumbent can develop. With progression, intracranial pressure may increase causing papilledema and changes in mental status. When patients present with vertigo of central origin, or focal neurologic deficits as described above, neuroimaging is indicated.

POOR PERFUSION STATES

Because many patients have difficulty describing exactly what their symptoms of dizziness are, disease processes that might cause lightheadedness or unsteadiness should also be considered. Causes of dizziness in patients with altered mental status include any disease state that leads to decreased cerebral perfusion or any of the various causes of shock. Although most patients with shock demonstrate tachycardia and hypotension along with altered mental status, patients with chronic hypertension may have a 50 mmHg drop in blood pressure with a normal reading and a resulting altered mental status.[23] Additionally, patients on beta-blockers or calcium channel blockers may not manifest tachycardia.

COMMON CAUSES OF SHOCK PRESENTING WITH DIZZINESS AND ALTERED MENTAL STATUS

A 78-year-old woman arrives at your ED with her family who reports that she seems more confused today. The patient complains of generalized weakness, dizziness, and nausea, but denies any pain. Her vital signs from triage show a heart rate of 108, blood pressure of 106/58, and temperature of 36°C.

Septic Shock

Classic septic shock is not difficult to diagnose in patients with fever, infection, and hypotension, but may not be considered as a differential diagnosis in this patient

with altered mental status and dizziness. However, in the early stages of the disease process, sepsis is often difficult to diagnose. Alterations in CNS function occur at higher blood pressures in septic shock compared to other types of shock because of the influence of inflammatory mediators.[23] Familiarity with the components of the systemic inflammatory response syndrome (SIRS), the first stage of the sepsis syndrome, can lead clinicians in the right direction. The diagnosis of SIRS requires two of the following criteria: temperature greater than 38°C or less than 36°C; heart rate greater than 90 beats/min; a respiratory rate greater than 20 breaths/min or a PaCO2 of less than 32 mm Hg; or a white blood cell count greater than 12,000 cells/μL or less than 4000 cells/μL.[24] Sepsis is then defined by the addition of infection, severe sepsis with the addition of end organ dysfunction, and septic shock with the addition of hypotension despite adequate fluid resuscitation.[25] Mortality rates for sepsis range from 25% to 80%,[26] with the incidence of sepsis and the mortality rate increasing with increasing patient age.[27] The presence of encephalopathy, as measured by changes in the Glasgow Coma Score, has also been associated with increased mortality.[28] It is therefore important for this patient complaining of dizziness to be evaluated for the presence of severe sepsis as the cause of her symptoms.

Aside from having a routine complete blood count (CBC), patients with suspected sepsis should have blood drawn for cultures (preferably before antibiotics are started), a lactate level measured, and prompt imaging to confirm clinically suspected sources of infection.[29] A complete metabolic panel and arterial blood gas analysis are useful to determine organ dysfunction and to calculate a severity of illness score. Once severe sepsis or septic shock is identified, rapid resuscitation dramatically decreases mortality.[30–33] Key components of early resuscitation include timely, broad spectrum antibiotics; adequate fluid resuscitation to restore circulating volume; the use of vasopressors when required to maintain a mean arterial pressure of greater than or equal to 65 mm Hg; and the addition of dobutamine when adequate cardiac output is not achieved with fluid resuscitation and vasopressors.[29] If patients are intubated, low tidal volumes and inspiratory plateau pressures reduce the risk for developing acute respiratory distress syndrome.[29] Less strongly recommended are the use of recombinant activated protein C and steroids.[29]

A 54-year-old man presents with lightheadedness and epigastric pain. He tells you he has had epigastric pain for years, but it seems to be getting worse over the last few months. He has felt lightheaded for the last few weeks. When questioned, he does report that his stool is dark, but he attributes the color to his frequent use of bismuth subsalicylate (Pepto-Bismol). He seems to have trouble concentrating and answering complex questions.

Hemorrhagic Shock

Although hemorrhagic states are generally not a diagnostic dilemma, patients with occult gastrointestinal (GI) bleeds may complain only of dizziness or near syncope. As the disease progresses undiagnosed and untreated, patients may develop altered mental status caused by relative hypotension and poor cerebral perfusion. Physical examination should check for pallor of mucus membranes and for the presence of blood or melena in the stool. Laboratory testing should include a CBC, type and screen, a basic metabolic profile, and coagulation studies. An elevated blood urea nitrogen (BUN)/Cr ratio (>30 or 36) may suggest an upper GI source of bleeding,[34,35] although the degree of overlap between upper and lower bleeds is significant.[36] All patients in whom GI bleeding is suspected of being the cause of their dizziness and altered mental status should have two, large bore needles intravenously (IV) placed, IV fluids administered, and cardiac monitoring started. Oxygen should be given to

elderly patients and those with preexisting cardiac disease to supplement the oxygen-carrying capacity of blood.[37] The patient previously described developed hypotension while in the ED, had new anemia and a BUN/Cr ratio of 38, confirming your suspected diagnosis of bleeding peptic ulcer. He, and most other patients with symptomatic GI bleeding, requires admission to an ICU.

A 45-year-old man complains of dizziness and groin pain. He was seen in your ED 3 days ago with chest pain, had a coronary CT with a 70% stenosis, and was then admitted for a coronary angiogram. He had no lesion requiring intervention and was discharged later that day on aspirin, clopidogrel, metoprolol, and lovastatin. Shortly after returning home, he started to have increasing pain in his groin, noted swelling at the catheterization site, and now feels dizzy. He appears restless and slightly agitated, unusual for him according to his wife.

Patients with recent invasive procedures involving access to femoral vessels or retroperitoneal surgery are at risk for developing retroperitoneal hemorrhage. Patients with bleeding disorders are at risk for spontaneous bleeding into the retroperitoneal space, most commonly as a result of anticoagulant use and less commonly as a result of antiplatelet medications or hemophilia. As the number of patients on anticoagulants increases, the number of patients at risk for bleeding complications also grows. Somewhere between one half and two thirds of patients with retroperitoneal hematomas are not over-anticoagulated, and mortality rates for all patients with retroperitoneal hematomas are as high as 20%.[38]

A classic presentation of retroperitoneal hematoma begins, like the case previously mentioned, with patients reporting pain in the groin, lower back, or abdomen. As the bleeding persists, paresthesias, paresis, and a palpable mass may develop. Large hematomas may lead to abdominal compartment syndrome. Patients may have dizziness and altered mental status from hypotension, and may describe unsteadiness or difficulty walking because of leg weakness stemming from nerve compression caused by the hematoma. Diagnosis is commonly made by CT, but ultrasound may also be helpful. However, ultrasound provides limited information about the extent of the hematoma or the presence of ongoing bleeding. Treatment includes standard resuscitative measures, such as IV fluids, blood transfusion as needed, and reversal of anticoagulation. More invasive therapies are of unproven benefit. The published literature investigating the treatment of retroperitoneal hematomas consists mostly of case reports or limited case series. Embolization by interventional radiology seems to be effective when a bleeding source can be identified.[39] Percutaneous drainage may provide only temporary benefits, but may also prevent permanent nerve damage by relieving compression from the hematoma.[40] Early surgery is advocated by some to ensure that permanent nerve compression does not occur,[41] whereas other authors suggest that surgery should only be used in patients who cannot be treated with less invasive means or in patients who remain unstable or who develop abdominal compartment syndrome. Conservative therapy is usually sufficient in stable patients who do not demonstrate mass effect from a large hematoma, such as paresthesias or paresis, abdominal compartment syndrome, or other signs of end organ damage.[39] In the previous case, the patient should be admitted for close monitoring, transfusion of red blood cells as needed, and stopping further doses of aspirin and clopidogrel. If the hematoma increases in size, more invasive measures would be appropriate.

A 78-year-old woman is brought to the ED by ambulance. She called for an ambulance because she felt too dizzy to get out of bed, but hasn't been able to provide much other history. She arouses to loud questions, but is unable to respond to anything but brief, simple questions. Her vital signs show a heart rate of 116, respiratory rate of 24, and blood pressure of 98/52. She denies chest pain, but does admit

that she is short of breath and nauseated. Her electrocardiogram shows ST elevation in inferior leads.

ACUTE CORONARY SYNDROMES

Decreased perfusion from a primary cardiac cause is a grave complication of acute coronary syndromes (ACS), affecting roughly 2% of unstable angina or non-ST elevation myocardial infarctions and 7% of ST-elevation myocardial infarctions.[42] Patients, such as in the previous case, present with signs of decreased perfusion despite having adequate circulating volume because of decreased cardiac output. In addition to the typical signs and symptoms of ACS, most patients will be hypotensive and have cool skin, dyspnea, and rales. Dizziness and altered mental status result from decreased cerebral perfusion. In the elderly, the classic signs of chest pain and ECG changes may be absent and confusion, altered mental status, or dizziness may be the chief complaint, as seen in the previous case.[43,44]

Left heart failure from a large left ventricular infarction is the most common cause of cardiogenic shock.[42] Right heart failure, likely the cause of the symptoms in the case described here, is less common and will show some improvement with volume resuscitation. Mechanical causes of cardiogenic shock complicating infarction, such as ventricular septal defect, acute valvular regurgitation, myocardial wall rupture, and tamponade, are uncommon, occurring in 2.8% to 10.7% of patients in the Global Utilization of Streptokinase and Tissue Plasminogen Activator to Treat Occluded Arteries (GUSTO) trial.[45] Failure to consider a mechanical cause, however, could delay lifesaving surgical repair.

TOXICOLOGIC CAUSES OF DIZZINESS AND ALTERED MENTAL STATUS

There are several toxicologic causes of altered mental status that also produce ataxia and nystagmus, which are often perceived by patients as dizziness. Many also have dizziness as a presenting symptom. Although ethanol is the most common intoxicant that produces these symptoms, it is not discussed here, but is covered in another section of this issue. Cases are described in the following discussion to illustrate some typical presentations.

A 14-year-old boy was found by his brother to have passed out in the garage. By the time you examine him, he complains of dizziness, being tired, and nausea. You note that he has an erythematous rash around his mouth, his speech is slurred, and he has nystagmus. As you lean closer to examine his heart, you note a chemical aroma.

INHALANTS

Inhalants used as drugs of abuse are a diverse group of compounds that are all volatile, and provide a quick-onset high followed by a rapid dissolution of symptoms. Most abused inhalants are available as legal substances, such as paints and solvents, fuels, aerosolized room deodorizers, and nitrous oxide in spray whipping cream, and are abused by adolescents.[46] Inhalants are absorbed through the lungs, cause intoxication in seconds to minutes, and then are excreted through the lungs or rapidly metabolized in the liver, or both.[47] The intoxication produced generally resembles that produced by alcohol, with euphoria, ataxia, slurred speech, and diplopia. Higher levels of intoxication may produce hallucinations, seizures, or coma.

The diagnosis of inhalant abuse is clinical because there are no specific diagnostic tests. Staining of the skin or clothing from pigments and characteristic odors may suggest the diagnosis. Wheezing from pulmonary irritation and a perioral rash may

also be seen. Treatment is generally supportive, with attention to the usual ABCs of resuscitation (ie, airway, breathing, circulation) and no treatment other than substance abuse counseling would be required for the case described previously. In the more serious overdose, cardiac effects are the most critical complication. The myocardium of patients who have abused inhalants becomes more sensitive to circulating catecholamines, provoking arrhythmias that are often not successfully resuscitated.[48] Standard treatment with advanced cardiac life support should be instituted, with the exception that further sympathomimetic medications are avoided and beta-blockers are added to blunt the effects of catecholamine sensitization.[49]

A 42-year-old woman presents to your ED with dizziness, headache, and decreased responsiveness. Her aunt reports that she went to her house today and found her to be confused and complaining of the previously mentioned symptoms. The patient is HIV positive with a recent CD4 count in her medical records of 198. Her aunt reports that she recently had some new medications added to her regimen to protect her from developing infections, but she is not sure what they are. You note that the patient is tachycardic, hypoxic, cyanotic, and confused.

METHEMOGLOBINEMIA

Methemoglobinemia is yet another potential cause of altered mental status and dizziness. Patients present with cyanosis, nausea, tachycardia, and headache at lower methemoglobin levels. Dizziness and altered mental status may occur as methemoglobin levels reach 30% to 50% of normal hemoglobin.[50] Above 50% to 60%, patients become lethargic and then comatose, and are prone to arrhythmias and seizures. Death usually occurs at levels above 70%.[50]

Methemoglobinemia is most commonly drug induced, with dietary and genetic conditions being the next most common causes.[50] Methemoglobinemia can also complicate the sequelae of nitrite abuse. The drugs that most commonly cause methemoglobinemia are local anesthetics, antimalarial drugs, and dapsone. In the case described previously, dapsone had been added to the patient's regimen for pneumocystis carinii pneumonia prophylaxis.[51] Nitroglycerin, phenazopyridine, and sulfonamides are other medications commonly used in the ED that may trigger methemoglobinemia.

A diagnosis of methemoglobinemia should be suspected in patients who are cyanotic with normal pulse oximetry whose cyanosis does not respond to oxygen therapy. Blood drawn from patients will classically appear chocolate brown. An arterial blood gas with co-oximetry should be drawn if the diagnosis is suspected. The partial pressure of O2 and CO2 will be normal, and metabolic acidosis may be present. Because the oxygen saturation of hemoglobin reported on the blood gas is calculated rather than measured, it will be normal. Co-oximetry directly measures the amount of methemoglobin and will confirm the diagnosis.[52] If co-oximetry is unavailable or will be delayed, a drop of the patients' blood can be placed on a piece of filter paper next to a drop of normal blood. The normal blood will appear dark red, but brighten with exposure to oxygen. The methemoglobinemic blood will stay chocolate brown.[50]

The patient described previously should be treated with high flow oxygen to increase the oxygen-carrying capacity of the blood. Healthy patients with methemoglobin levels below 30% do not need other treatment.[50] Methylene blue will convert methemoglobin to hemoglobin, and should be given to patients with methemoglobin levels higher than 30% or to more symptomatic patients, including the one described here. The dosage given should be proportionate to the symptoms and methemoglobin levels, with 1 mg/kg used for levels around 30% in patients with milder symptoms and

2 mg/kg used for patients with more severe symptoms or with methemoglobin levels near 50%.[52] Patients who do not respond to methylene blue treatment may require treatment with p450 inhibitors, such as cimetidine or ketoconazole; exchange transfusion; or treatment with n-acteylcysteine.[50,52,53]

A 38-year-old woman is sent from the rehabilitation center for evaluation of confusion. She is in rehabilitation because of a seizure disorder that led to a motor vehicle accident 2 months ago with significant injuries. Her husband reports that she had been complaining of feeling off balance and nauseated the day before. Her medications are phenytoin, fentanyl patch, cimetidine, and ibuprofen as needed.

PHENYTOIN

Antiepileptic medications can cause altered mental status and dizziness at supratherapeutic or toxic levels. Although acute phenytoin toxicity is most commonly seen in overdose, chronic toxicity can result after small dosage changes or after the addition of another medication that either alters its binding to serum proteins or its hepatic metabolism.[54] The patient discussed previously may have increased phenytoin concentrations in her blood as a result of her taking cimetidine, or she may have decreased concentrations of serum proteins as a result of her prolonged debilitated state. Supratherapeutic levels of phenytoin cause nausea, confusion, ataxia, and nystagmus.[55] Higher levels produce lethargy and coma. Toxicity is difficult to predict from the total concentration in the blood, especially in patients who are critically ill or hypoalbuminemic, because free serum phenytoin is the pharmacologically active component, and routine blood tests measure bound and free serum concentrations.[56]

Treatment is generally supportive, with focus on fall prevention for milder toxicity, although intubation for airway protection may be required in more severe toxicity. Because phenytoin is mainly protein bound, extra-corporeal methods of elimination are generally not effective. Multidose activated charcoal (MDAC) has been reported to lower the levels of phenytoin,[57–59] but the American Academy of Clinical Toxicology and European Association of Poisons Centres and Clinical Toxicologists do not recommend its use in phenytoin overdose.[60]

CARBAMAZEPINE

Carbamazepine is another widely used antiepileptic medication that can cause altered mental status and dizziness, but has more severe effects in overdose. Most patients will have a decreased level of consciousness, many develop changes in muscle tone, ataxia, vertigo, and nystagmus, and up to 24% will have seizures.[61] Cardiac conduction effects, such as atrioventricular block, ventricular arrhythmias, and QRS widening, are also commonly seen.[62] Many of the toxic effects result from the sodium channel blocking and anticholinergic effects. The severity of presentation appears to correlate with serum level, with a cutoff of 170 μmol/L or 40 mg/L indicating increased risk.[62]

Seizures should be treated with benzodiazepines, and close attention should be paid to the patients' ability to protect their airway because intubation is usually necessary with serious overdoses.[63] Telemetry monitoring should be continued until the serum carbamazepine levels have returned to the therapeutic range,[64] with the understanding that absorption of the drug is often delayed and erratic. Electrocardiogram, complete metabolic profile, and a CBC should be ordered to evaluate for the hyponatremia and cardiac conduction effects that are seen in acute toxicity, and for the hepatic toxicity and blood dyscrasias that can occur with chronic use of the medication.

Decontamination and elimination of carbamazepine will speed recovery. Activated charcoal should be given immediately to patients who are alert and to those with

secured airways. The American Academy of Clinical Toxicology and European Association of Poisons Centres and Clinical Toxicologists recommend the use of MDAC for life-threatening carbamazepine overdoses.[60] One recent, small study of 12 subjects did show decreased time of mechanical ventilation requirements and decreased time comatose after the use of MDAC.[65] If a patient has ileus, or if MDAC is not felt to be appropriate for other reasons, charcoal hemoperfusion is also effective at reducing serum concentrations of carbamazepine.[66]

SUMMARY

The causes of vertigo and dizziness in patients with altered mental status are varied and broad. A careful history and physical examination will guide the clinician's further evaluation and testing. Patients who have vertigo require imaging, CT and often MRI, and most patients will be admitted for further evaluation. Patients with other forms of dizziness require a more broad differential diagnosis. Careful review of vital signs and components of the history and physical examination may suggest decreased perfusion from early cardiogenic, septic, or hemorrhagic shock. Patients with confusion and nystagmus or unsteadiness require a careful review of the medications they have received, because a toxicological cause may explain their symptoms. Careful and systematic evaluation of patients will alleviate the distress associated with the evaluation of dizzy and confused patients.

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