Dizziness and Vertigo: Emergencies and Management

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KEYWORDS

• Vertigo • Dizziness • Vestibular • Imbalance
• Emergency room • Nystagmus

A 49-year-old woman is brought to the emergency room (ER). For the past two days, she has had a sense of rotation and imbalance. The goal of the ER evaluation is to

- Quickly identify if the problem is peripheral (inner ear or vestibular nerve) or central
- Determine if the patient needs to be admitted to the hospital or referred to a specialty clinic
- Offer initial management of the problem.

There are three key features in the history and five key elements of clinical examination that can be used to make these decisions.

THREE KEY ITEMS IN THE HISTORY

The history is by far the most important part of the evaluation. Unfortunately, taking a good history can be extremely tedious because complaints are often vague and frequently filled with anxiety-provoked symptoms. The three key items to obtain from the history are tempo, symptoms, and circumstances (**Table 1**). Determine if the patient has an acute attack of dizziness (3 days or fewer), chronic dizziness (more than 3 days), or spells of dizziness. If the patient suffers from spells, try to determine the average duration of the spells in seconds, minutes, or hours. Defining and expanding on the patient's vague report of dizziness is critical when describing the patient's symptoms. Dizziness is an imprecise term used to describe a variety of symptoms, each of which has a different pathophysiologic mechanism. A careful history can differentiate disequilibrium, primarily a balance problem, from the

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Table 1
Key items in the history of the dizzy patient

Disorder	Tempo	Symptoms	Circumstances
Acute Vestibular Neuritis	Acute (<3 d)	Vertigo, disequilibrium, N/V, oscillopsia	Spontaneous, exacerbated by head movements
Wallenberg Infarct	Acute	Vertigo, disequilibrium, N/V, tilt, lateropulsion, ataxia, crossed sensory loss, oscillopsia	Spontaneous, exacerbated by head movements
Anxiety or Depression	Chronic	Lightheaded, floating, or rocking	Induced by eye movements with head still
Benign Paroxysmal Positional Vertigo	Spells: s	Vertigo, lightheaded, nausea	Positional: lying down, sitting up or turning over in bed, bending forward
Orthostatic Hypotension	Spells: s	Lightheaded	Positional: standing up
Transient Ischemic Attack	Spells: min	Vertigo, lightheaded, disequilibrium	Spontaneous
Migraine	Spells: min	Vertigo, dizziness, motion sickness	Usually movement-induced

Abbreviation: n/v, nausea and vomiting.

subjective sensation of dizziness in the head. If dizziness is primarily in the head, the physician must determine if the sensation is lightheadedness (presyncope, rocking, or swaying) or vertigo (the abnormal sensation of rotation, linear movement, or tilt). Finally, associated symptoms of nausea and vomiting, vertical diplopia, tinnitus, hearing loss, or oscillopsia (the illusion of visual motion) must be elicited in the history. Knowledge of the circumstances triggering or exacerbating symptoms often helps to elucidate the mechanism behind dizziness. Is symptoms onset spontaneous or are symptoms triggered by movements, an event, or clustered during a specific time of day? If it is reported that movements exacerbate symptoms, it is important to differentiate whether the movements are primarily head movements, body movements or postural changes, or eye movements. Using the history of the 49-year-old woman given above, acute vestibular loss on one side (vestibular neuritis) is the most likely diagnosis (see **Table 1**). The clinical examination will help confirm this diagnosis.

FIVE KEY ELEMENTS OF THE CLINICAL EXAMINATION

The five key elements in the clinical examination of a dizzy patient are assessment of the vestibule-ocular reflex (VOR), spontaneous nystagmus, positional nystagmus, the Romberg test, and gait. **Table 2** lists the most common findings in normal individuals, individuals with acute unilateral peripheral vestibular loss (the case example), benign paroxysmal positional vertigo (BPPV), and central vestibular problems. Techniques to assess each of these examination elements in a normal individual are described below.

Vestibular-Ocular Reflex

The intact VOR compensates for head movements by moving the eyes in the opposite direction to head movements, thus fixing the eyes with respect to the world (**Fig. 1**). Examine the VOR using the head thrust. Have the patient fixate a target and observe the eyes after passive head thrusts horizontally and vertically. After the head thrust, the eyes of the patient should remain stable and focused on the target. This indicates

Table 2
Key items in the clinical examination of the dizzy patient

Examination	Normal	Acute Vestibular Neuritis	BPPV	Central
VOR Assessed by Head Thrust	Normal (no corrective saccade)	Corrective saccade needed after head thrust done in direction of defective inner ear	Normal	Normal
Spontaneous Nystagmus Assessed by Ophthalmoscope	None	Horizontal and mild torsional enhanced with fixation blocked	None	May or may not be present; often vertical or gaze evoked
Positional Nystagmus or Vertigo Assessed with Dix-Hallpike or Supine	None	Does not enhance vertigo, may enhance nystagmus	Transient upbeat and torsional nystagmus during Dix-Hallpike	None, or may have sustained or transient nystagmus with or without vertigo
Romberg	Negative	Negative	Negative	Often positive
Gait	Normal	Slow, cautious, wide-based, robotic	Normal	Often impaired

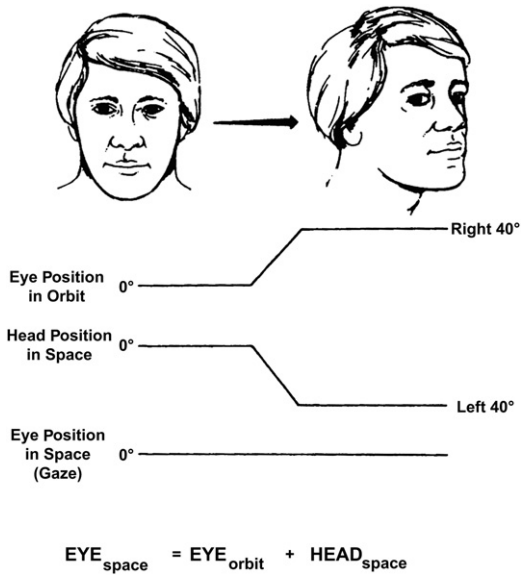

Fig. 1. The VOR in a normal individual. Top row shows the head moving quickly from center (0°) to left 40°, and it shows what happens to the eyes. The next three rows in the figure graphically show eye position in the orbit, head position in space, and eye position in space during this head movement. (*Adapted from* Leigh RJ, Zee DS. The neurology of eye movements. New York: Oxford Univ Press; 1999; with permission.)

a normal VOR. If the VOR is impaired, the eyes of the patient will not remain stable and a corrective, refixation saccade back to the target is observed.[1,2]

Presence of Spontaneous Nystagmus in Light With and Without Fixation Blocked

Normal individuals have no spontaneous nystagmus in the light (fixation present) or in the dark (fixation blocked) (see **Table 2**). The presence of spontaneous nystagmus should be assessed with and without fixation because peripheral causes of nystagmus usually can be suppressed with fixation, whereas central causes cannot be suppressed. An easy way to test this is during the ophthalmoscopic examination with the other eye fixating a target.[3] During this procedure, the optic nerve is visualized and assessed for spontaneous nystagmus with the unobscured eye fixating on a distant target. The examiner then covers the fixating eye with the hand and assesses the optic nerve for an unchanged nystagmus, suggesting a central cause, or an increase in nystagmus, suggesting a peripheral cause. During this examination, the optic nerve head should not show any oscillation. If it does, determine the direction of the quick phases. Remember, the direction of movement of the disk will be opposite to what is occurring.

The Presence of Positional Nystagmus

Positional nystagmus and vertigo is best assessed using the Dix-Hallpike test (**Fig. 2**). Normally, there is no nystagmus or vertigo with this maneuver. A positive Dix-Hallpike will result in transient symptoms of vertigo and torsional nystagmus, generally after a 30-second delay, with the head turned toward the affected ear. When normal individuals sit up, they may complain of lightheadedness due to a transient blood pressure drop.

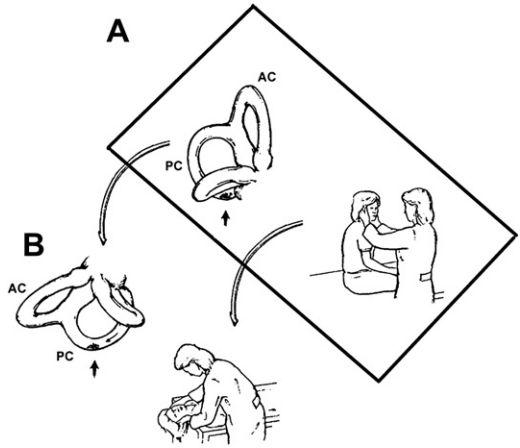

Fig. 2. Dix-Hallpike test for BPPV. (*A*) The patient sits on the examination table and the head is turned 45° horizontally. (*B*) The head and trunk are quickly brought straight back "en bloc" so that the head is hanging over the edge of the examination table by 20°. Nystagmus is looked for and the patient is asked if they have vertigo. Although not shown in the figure, the patient is then brought up slowly to a sitting position with the head still turned 45° and nystagmus is looked for again. This test is repeated with the head turned 45° in the other direction. This figure also shows movement of free-floating otoconia in the right posterior SCC (*large black arrows*) during the Dix-Hallpike test. In this example, the patient would have nystagmus and vertigo when the test is done on the right side, but not when the test is done on the left side. AC, anterior semicircular canal; PC, posterior semicircular canal. (*Data from* Herdman SJ, Tusa RJ, Zee DS, et al. Single treatment approaches to benign paroxysmal positional vertigo. Arch Otolaryngol Head Neck Surg 1993;119:450–4.)

Romberg Test

In the Romberg test, the patient is asked to stand with feet slightly apart, first with eyes open, and then closed. The patient is asked to fold her or his arms across the chest for 30 seconds eyes open, and then 30 seconds eyes closed. A positive Romberg is one in which the patient is stable with eyes open, but loses balance with eyes closed. A positive Romberg may be found in patients with proprioceptive defects from a peripheral neuropathy, dorsal root ganglia, or dorsal column disease. It is rarely found in individuals with acute vestibular loss.

Gait

The patient should be asked to walk at a comfortable pace for about 20 ft in one direction, and then to turn and come back. Features to look for are gait speed, the base width, step length, arm swing, minimal gyration of trunk and deviation of path, and movement of the head especially during turns (the head usually turns before the body).

IMAGING AND LABORATORY TESTS

Vertigo and dizziness can be effectively evaluated in the acute setting primarily based on clinical presentation and examination. However, additional testing may be required to confirm the diagnosis. From a diagnostic standpoint, differentiating peripheral and central causes for these symptoms is paramount to manage the condition appropriately and avoid overlooking a central condition. **Table 3** summarizes some the hallmark examination features differentiating peripheral and central vertigo. The

Table 3
Features that distinguish peripheral from central causes of vertigo

Findings	Peripheral Cause	Central Cause
Direction of Nystagmus	Usually mixed plane (horizontal and torsional)	Usually single plane (horizontal, torsional, or vertical)
Effect of Gaze on Nystagmus	Nystagmus increases with gaze toward direction of quick phase	Nystagmus does not change or reverses direction
Effect of Fixation on Nystagmus	Nystagmus decreases	Nystagmus does not change or increases
Ice-water Caloric Test	Spontaneous nystagmus does not change when affected ear is irrigated; nystagmus decreases or reverses direction when nonaffected side is irrigated	Spontaneous nystagmus increases when affected ear is irrigated; nystagmus reverses direction when nonaffected side is irrigated
Balance	If patient is younger than age 50 years, balance is normal except no sharpened Romberg test; if older than age 50 years, may have positive Romberg test	May have severe defect regardless of age (positive Romberg, patient veers when walking with eyes open)

differential diagnosis of dizziness and vertigo always includes infarcts or hemorrhage in the cerebellum, thalamus, or brainstem. The presentation of dizziness is associated with stroke in an estimated 3% to 4% of cases presenting to ERs.[4] Concern for stroke often leads to imaging studies in the acute setting; however, recent work has demonstrated superior sensitivity with oculomotor examination compared with early MRI.[4,5] When a central cause for dizziness and vertigo is suspected based on the clinical examination, imaging is certainly warranted. Based on the location of lesions likely to result in dizziness and vertigo stroke syndromes and with the acute onset of such symptoms, diffusion-weighted MRI is preferred over CT scan. Unnecessary tests are frequently performed when benign, peripheral causes for vertigo, such as BPPV, are demonstrated by clinical examination.[6] When a peripheral cause for dizziness and vertigo is suggested by clinical examination, additional testing is warranted. Viral cultures are not necessary because they do not alter treatment. Blood work should include fluorescent treponemal antibody absorption, rheumatoid factor, antinuclear antibody, and erythrocyte sedimentation rate to screen for otic syphilis and vasculitis. An audiogram should be obtained if the patient complains of hearing loss. A bedside caloric can be done immediately to help distinguish a peripheral from a central defect. A quantified caloric study, or electronystagmography (ENG), should be obtained several days after onset to document the extent of vestibular defect. Only admit the patient to the hospital if extreme dehydration is present from vomiting (rarely necessary) or if a central disorder is suspected. The patient should return in a few days to make certain the symptoms are resolving. It is then important to refer them promptly for vestibular rehabilitation.

CLINICAL CASE EXAMPLES

Acute Unilateral Peripheral Vestibular Loss

Acute vestibular neuritis: history and clinical exam

This disorder presents with intense vertigo, nausea, oscillopsia (illusion of movement of the visual world due to spontaneous movement of the eyes), and disequilibrium that

persists for days. The vertigo is quite severe and patients prefer to lie quietly. Thus, the vertigo, nausea, and oscillopsia are occurring spontaneously, but they are exacerbated by head movements. Within a few days these symptoms begin to resolve and the patient is left with a dynamic deficit (vertigo and disequilibrium induced by rapid head movements), which can last for weeks to months until central compensation occurs. Thus, the tempo is acute; the symptoms include vertigo, imbalance, nausea, and oscillopsia; and the circumstances are that the vertigo is continuous and exacerbated with head movements. Based on these key items in the history, acute vestibular neuritis is likely (see **Table 1**).

The clinical examination for acute vestibular neuritis is detailed in **Table 2**. Examine the VOR using head thrust. Have the patient fixate a target and observe the eyes after thrusting the head horizontally at an angle of approximately 20°. After the head thrust, a saccadic eye movement will occur about 250 milliseconds after the head thrust. This saccade allows the patient to refixate on the target. It occurs because the VOR is impaired in the direction that the head was thrust.[1] Next, look for the presence of spontaneous nystagmus, with and without fixation, using an ophthalmoscope. It is important to do this with fixation blocked because peripheral vestibular loss causes nystagmus that can be suppressed with fixation. Acute, unilateral, vestibular loss causes a horizontal jerk nystagmus in which the quick phases are directed toward the normal ear. In addition, there is usually a torsional nystagmus in which the quick phases move the superior pole of each eye toward the normal ear. Thus, if the individual has a right vestibular loss, a left beat nystagmus and left torsional nystagmus will occur (the direction of the nystagmus is labeled based on the direction of the quick phase). Next, look for the presence of positional nystagmus. In vestibular neuritis, the spontaneous nystagmus may be enhanced during positional testing but there is usually no increase in vertigo. Next, examine Romberg and gait. The Romberg is negative; that is, there is no falling, sidestepping, or eye opening for 30 seconds. The gait will be slow, cautious, wide-based, and robotic with no movement of the head on body, especially during turns.

Pathophysiology and cause

Vestibular neuritis is preceded by a common cold 50% of the time. The prevalence of vestibular neuritis peaks at age 40 to 50 years.[7,8] Vestibular neuritis behaves similar to Bell palsy and is thought to frequently represent a reactivated dormant herpes infection in the Scarpa ganglia.[9] Vestibular neuritis primarily affects the superior division of the vestibular nerve, which innervates the anterior and lateral semicircular canals (SCCs).[10]

Management, including laboratory tests

Vestibular neuritis is diagnosed based primarily on the clinical presentation. Viral cultures are not necessary because they do not alter treatment. The differential diagnosis includes infarcts or hemorrhage in the cerebellum, or in the brainstem distribution of the posterior inferior cerebellar artery and anterior inferior cerebellar artery. Additional imaging, specialized tests, such as ENG, or hospital admission may be indicated if a central cause is suspected.

Management of acute vertigo from vestibular neuritis varies depending on how many days have elapsed since the onset (**Table 4**). Only admit the patient to the hospital if extreme dehydration is present from vomiting (rarely necessary) or if a central disorder is suspected. Blood work should include fluorescent treponemal antibody absorption, rheumatoid factor, antinuclear antibody, and erythrocyte sedimentation rate to screen for otic syphilis and vasculits. An audiogram should be

Table 4
Management of acute vertigo from vestibular neuritis

Days One to Three	After Day Three
Administer vestibular suppressants (Phenergan, Antivert, or Valium)	Stop vestibular suppressants
Administer prednisone	Taper over the course of 10 d
Prescribe bed rest (hospitalize if patient dehydrated or central defect suspected)	Prescribe vestibular adaptation exercises
Perform laboratory tests If central defect is suspected, obtain CT scan or MRI of head	Perform laboratory tests Audiogram (obtain immediately if Meniere disease suspected) Electronystagmography (limited) Blood work (rheumatoid factor, sedimentation factor, antinuclear antibody, fluorescent treponemal antibody absorption)

obtained if the patient complains of hearing loss. A minimal ice-water caloric test may be a suitable procedure for bedside assessment of vestibular function.[11] In this test, 0.5 to 1 mL of ice water is inserted into the external auditory canal and allowed to sit for 40 seconds before it is removed. The number of quick phases of nystagmus over the course of 15 seconds are counted, preferably with fixation blocked. Peripheral vestibular loss will results in reduced nystagmus on that side. A quantified caloric study (ENG) should be obtained several days after onset to document the extent of vestibular defect. By this time, the spontaneous nystagmus should be significantly decreased. A variety of vestibular suppressant medications can be employed for symptomatic treatment. These should be used for 1 week or less because the acute phase is a self-limited disorder. The authors use intramuscular Phenergan (25–50 mg) at the onset of severe vertigo, and then send the patient home for 3 days of bed rest with Phenergan suppositories to be taken as needed. This medication is causes sedation and reduces nausea. The patient returns in a few days to make certain the symptoms are resolving. It is then important to stop the medication and refer them promptly for vestibular rehabilitation. Prednisone (1 mg/kg) during the first 10 to 20 days of the attack may shorten the course of the illness.[12–15]

Spells of Dizziness

BPPV; history and clinical examination

There are several causes of spells of dizziness, but the most common cause is BPPV. Patients with BPPV usually complain of vertigo that lasts less than 1 minute. It usually occurs in the morning when they get up or turn over in bed. It may also occur when they lie down in bed or move their head back. After a bad attack, they frequently complain of disequilibrium that lasts for several hours. Therefore, the tempo is spells lasting seconds, the symptoms are vertigo and nausea, and the circumstances are positional when lying down, sitting up, or bending over (see **Table 1**). The diagnosis of BPPV is secured by the clinical examination. All key elements of the clinical examination are usually normal except for the positional test (Dix-Hallpike test; see **Table 2**). This test elicits a torsional-upbeat nystagmus associated with vertigo when the affected ear is inferior. The nystagmus usually has a latency of 3 to 20 seconds, fatigues less than 1 minute, and habituates with repeat maneuvers. If nystagmus and vertigo persist while the patient is in this position, and is not present while sitting,

a central disorder (central positional vertigo) should be considered, although there are exceptions.[16]

Pathophysiology and cause

BPPV is usually idiopathic, but can also occur after head trauma, labyrinthitis, and ischemia in the distribution of the anterior inferior cerebellar artery. The pathophysiological mechanism for BPPV is usually due to canalithiasis; that is, portions of otoconia from the utricle that are misplaced and free-floating in the posterior SCC (see **Fig. 2**). This condition inappropriately causes the afferents from the posterior SCC to discharge when the head stops moving after head rotation.

Management, including laboratory tests

BPPV is a clinical diagnosis based on the history and the Dix-Hallpike test. No special testing is required.[6] BPPV is best treated by a maneuver that moves the debris out of the posterior SCC and back into the utricle. The authors primarily use a single-treatment approach modified from Epley,[17] which is now generally referred to as the canalith-repositioning maneuver (CRM) (**Fig. 3**). The authors found total remission or significant improvement from BPPV in 90% of patients treated using this maneuver.[18] Complications from the CRM are rare.[19] During the CRM, a Dix-Hallpike maneuver is first performed toward the side of the affected ear and the head is kept down for

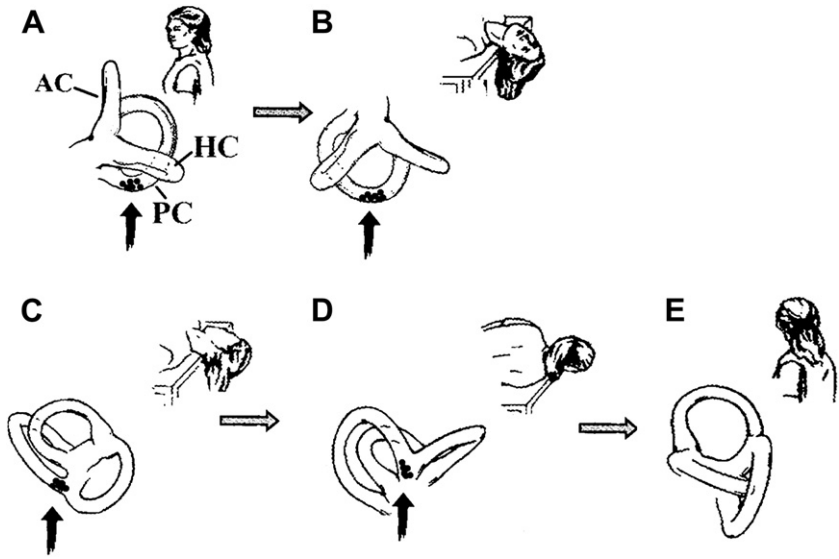

Fig. 3. Canalith repositioning maneuver for treatment of BPPV. Filled arrows indicate location of the posterior SSC. The patient is first moved from sitting (*A*) into the Dix-Hallpike position with the head turned 45° toward the affected side (*B*). After a few minutes, the patient's head is turned so the opposite ear is down (*C*) and then is rolled onto that shoulder with the nose pointed 45° down (*D*). After a few minutes, the patient sits up, keeping the head turned while coming into the sitting position (*E*). Because the debris will move whenever the head is moved during this maneuver, the patient should be advised to expect vertigo to occur several times during the treatment. Some patients only experience vertigo during the initial movement into the Dix-Hallpike position. (*Data from* Herdman SJ, Tusa RJ, Zee DS, et al. Single treatment approaches to benign paroxysmal positional vertigo. Arch Otolaryngol Head Neck Surg 1993;119:450–4.)

2 minutes. Then the head is rotated toward the unaffected side and the patient is rolled over onto this side until the face is pointed down. This position is maintained for 2 minutes. With the head deviated toward the unaffected side, the patient slowly sits up. To make certain the debris does not move back toward the cupula, the patient is asked to sit with the head upright for 20 minutes. A consensus paper that summarizes the best controlled studies on treatment of several types of BPPV was published in 2008.[20] It is unclear what happens to the free-floating otoconia after the treatment, but, presumably, it is reabsorbed into the calcium matrix in the utricle. The patient should have follow-up after the ER with a clinic familiar with BPPV to be certain the treatment worked. Vestibular suppressant drugs do not have a role in the treatment of BPPV unless the patient refuses to do the treatment due to excessive vertigo and nausea.

Central Dizziness and Vertigo

Stroke and vertebral basilar ischemia: history and clinical examination

The central causes of dizziness seen in the ER are extensive. A couple of the most common are reviewed here. The most worrisome central cause of dizziness is acute ischemic event in the posterior fossa. The symptoms and signs vary depending on the location of the ischemic event (**Table 5**). Vertigo is among the initial symptoms in 48% of patients with stroke; however, stroke is diagnosed in fewer than 5% of patients presenting with dizziness.[4,21] A small percentage of patients with vertebral basilar ischemia may present with isolated spells of vertigo, presumably due to ischemia in the distribution of the anterior vestibular artery, a branch from the anterior inferior cerebellar artery (AICA) (see **Table 5**). This small artery perfuses the anterior and lateral SCC and the utricular maculae, and spares the cochlea. The clinical examination of central vestibular defects is detailed in **Table 2**. The VOR using the head thrust test is normal for all central defects unless the peripheral vestibular system is involved (infarct in the dorsolateral pons-AICA). Look for spontaneous nystagmus due to central defects with the eyes looking straight ahead, to the left, and to the right. Do this also using the ophthalmoscope to block fixation. Selective lesions in the central vestibular pathways result in spontaneous nystagmus due to unopposed higher spontaneous neural activity in the intact vestibular pathways (**Table 6**). Unlike spontaneous nystagmus from peripheral vestibular lesions, those from central vestibular lesions are not readily suppressed with fixation. Look for central positional vertigo when laying the patient supine or during the Dix-Hallpike test. This is a transient or sustained downbeat nystagmus with severe vertigo and is due to lesions that disrupt the pathways between the midline cerebellar structures and vestibular nucleus (see **Table 6**).[22] A positive Romberg test may be found in patients with proprioceptive defects from dorsal root ganglia or dorsal column disease. Gait is very helpful test to assess patients with central defect because it often is impaired even when few additional findings are noted. The gait may be spastic, ataxic, halting, or freezing. Romberg and gait assessment are also helpful in identifying a functional component.[23]

Pathophysiology and cause

The central vestibular structures are perfused by several vessels within the vertebral-basilar system. **Table 5** describes the structures and resulting signs from ischemic events and strokes.

Dorsolateral pontine infarct The AICA perfuses the lateral cerebellum (cerebellar branch), the dorsolateral pons (pontine branch), and the labyrinth (labyrinth artery). Vertigo can occur from infarcts in either the pontine branch or labyrinth artery. The AICA syndrome may present with just peripheral signs if the labyrinth artery is solely

Table 5
Symptoms and signs from stroke in the posterior fossa

Region	Vessel and Structure	Symptoms and Signs
Dorsolateral Pons	AICA	
	Cerebellar branch	
	Lateral cerebellum	Ipsilateral dysmetria
	Pontine branch	
	CN V and VII	Vertigo, ipsilateral pain, and temp loss (face), peripheral VII loss, dysarthria
	Sympathetic CN III fibers, middle cerebellar, peduncle, paramedian pontine reticular formation	Ipsilateral Horner, dysmetria, saccade palsy
	Spinothalamic tract	—
	Labyrinthine artery branch	
	Cochlea and labyrinth	Contralateral loss pain and temp (body)
	Anterior vestibular artery subbranch	
	Horizontal SCC, anterior SCC, utricle	Vertigo, imbalance, nausea, vomiting, absent ipsilateral VOR
	Posterior vestibular artery subbranch	
	Posterior SCC, saccule, cochlea	Vertigo, ipsilateral sensorineural hearing loss, tinnitus, normal VOR
Dorsolateral Medulla (Wallenberg Syndrome)	Posterior inferior cerebellar artery	
	Cerebellar branch	
	Posterior inferior cerebellum	Imbalance, ipsilateral ataxia
	Medullary branch	
	V nucleus-tract, IX nucleus-tract, X nucleus-tract, sympathetic tract	Ipsilateral pain and temp loss (face), decreased gag, vocal cord paresis, ipsilateral Horner
	Inferior cerebellar peduncle, vestibular otolith	Ipsilateral ataxia, lateropulsion, ocular tilt
	Lateral spinothalamic tract	Contralateral loss pain and temp (body)
	Vestibular VIII nucleus	Vertigo, nausea, vomiting, nystagmus (pure torsional or vestibular with reversal on gaze toward lesion side)
Medial Medulla (Lower Medulla)	Penetrator from anterior spinal artery	
	XII nucleus, pyramidal tract, medial meniscus	Ipsilateral tongue weakness, contralateral weakness (body), decreased vibration and proprioception (body)
	Nucleus intercalatus	Vertigo, nausea, vomiting, upbeat nystagmus, truncal ataxia

Abbreviations: cn, cranial nerve; temp, temperature.

Table 6
Vestibular nystagmus due central lesions

Nystagmus	Pathology	Possible Mechanism
Torsional Nystagmus	Dorsolateral medulla lesion	Decreased tonic neural activity to the INC from anterior and posterior SCC on one side.
Downbeat Nystagmus	Cerebellar flocculus lesion or floor of fourth ventricle lesion	Decreased tonic neural activity to the INC from posterior SCC on both sides
Central Positional Nystagmus or Vertigo	Midline vestibular cerebellum	Disruption between cerebellar nodulus and vestibular nucleus
Upbeat Nystagmus	Brachium conjunctivum lesion Dorsal upper medulla lesion	Decreased tonic neural activity to INC from central anterior SCC on both sides.
Seesaw Nystagmus	Unilateral lesion of INC	Unilateral inactivation of INC on one side
Periodic Alternating Nystagmus	Cerebellar nodulus lesions	Unstable (high gain) neural activity in the medial vestibular nucleus.

Abbreviation: INC, interstitial nucleus of Cajal.

involved (vertigo, nausea, vomiting, hearing loss, and tinnitus) or may include more central signs if the dorsolateral pons is involved (dysarthria, peripheral facial palsy, trigeminal sensory loss, Horner syndrome, dysmetria, contralateral temperature, and pain sensory loss).[24] The labyrinthine artery originates directly from the basilar artery in approximately 15% of patients.

Dorsolateral medulla infarct The posterior inferior cerebellar artery perfuses the posterior inferior cerebellum (cerebellar branch) and the dorsolateral medulla. Vertigo can occur from infarcts in the lateral medulla (Wallenberg syndrome) due to involvement of the vestibular nucleus. Characteristic signs include crossed sensory signs, ipsilateral lateropulsion, ataxia, and Horner sign. Nystagmus may be pure torsion, or mixed torsion and horizontal. When the nystagmus contains a horizontal component, it reverses direction on gaze toward the lesion side, unlike nystagmus from peripheral vestibular lesions.

Medial medulla infarct The upper medial medulla is usually perfused by a penetrating vessel from the vertebral artery and the lower medial medulla is perfused by a branch from the anterior spinal artery. Infarcts in the latter may result in vertigo from involvement of the intercalatus nucleus.[25] The physiologic role of the intercalatus nucleus is poorly understood, but it is thought to be involved with the vertical neural integrator. Characteristic signs include ipsilateral tongue weakness, contralateral body weakness, sensory loss, and upbeat nystagmus.

Management, including laboratory tests
There are only anecdotal comments about the effectiveness of treatment of vertigo from cerebrovascular disorders. Ondansetron (Zofran) may be appropriate for severe vertigo and nausea from stroke.[26] Coumadin and transluminal angioplasty of vertebral artery stenosis and, occasionally, aspirin or ticlopidine have been found to be effective in stopping spells of central vertigo from vertebrobasilar artery insufficiency.[27–30] Treatment includes reduction of risk factors for cerebrovascular disease and

antiplatelet therapy. These patients usually have known cerebrovascular disease or risk factors for this disease. Magnetic resonance arteriography can be performed to assess posterior circulation vessels and transcranial Doppler may detect decreased flow in the basilar artery.

SUMMARY

The causes of dizziness in patients seen in the ER can usually be determined by a focused evaluation that consists of identifying three key features in the history five key elements of the clinical examination.

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