

*Current Concepts***ACUTE VESTIBULAR SYNDROME**

JOHN R. HOTSON, M.D., AND ROBERT W. BALOH, M.D.

RAPID, unilateral injury to either peripheral or central vestibular structures produces the acute vestibular syndrome, which consists of severe vertigo, nausea and vomiting, spontaneous nystagmus, and postural instability. When this syndrome evolves over days in a healthy person, it is usually attributed to a viral vestibular neuritis, also called vestibular neuronitis or, when acute hearing loss occurs, neurolabyrinthitis.¹⁻⁶ Infarction and hemorrhage of the inferior cerebellum, however, may simulate vestibular neuritis (Fig. 1).^{9,10} As many as 25 percent of patients with risk factors for stroke who present to an emergency medical setting with isolated, severe vertigo, nystagmus, and postural instability have an infarction of the inferior cerebellum.¹¹

It is important to recognize a stroke involving the inferior cerebellum, because it can produce cerebellar swelling that can lead to brain-stem compression and death unless there is neurosurgical intervention (Fig. 1).^{9,12,13} Most patients with an inferior cerebellar infarction, however, do not go on to have brain-stem compression but make an excellent recovery. Their condition may be misdiagnosed as a peripheral vestibular lesion. Infarction of the inferior cerebellum may be caused by occlusion of the posterior or anterior inferior cerebellar artery due to embolism or atherosclerotic stenosis.^{9,11,14,15} Therefore, it is also important to recognize an ischemic acute vestibular syndrome and identify its cause in order to decrease the probability of recurrent stroke. The clinical differentiation of a peripheral vestibular lesion from an inferior cerebellar stroke is a main focus of this article.

From the Department of Neurology and Neurologic Sciences, Stanford University School of Medicine, Stanford, Calif., and the Santa Clara Valley Medical Center, San Jose, Calif. (J.R.H.); and the Department of Neurology and the Division of Head and Neck Surgery (Otolaryngology), University of California at Los Angeles School of Medicine, Los Angeles (R.W.B.). Address reprint requests to Dr. Hotson at the Santa Clara Valley Medical Center, 751 South Bascom Ave., San Jose, CA 95128.

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SIMPLIFIED FUNCTIONAL ANATOMY AND VASCULAR SUPPLY

The peripheral vestibular system includes the vestibular labyrinth and the vestibular nerve. The vestibular labyrinth contains the sensory receptors for balance, which lie in the three semicircular canals and in the otolithic organs (the saccule and utricle). The auditory labyrinth, the cochlea, holds the hearing sensory receptors (Fig. 2).¹⁶ The cristae in the semicircular canals sense angular acceleration, such as that due to brief rotation of the head.¹⁸ The maculae in the otolithic organs sense linear acceleration, such as that produced by translational head movement or tilting of the head from the upright position.¹⁹ The vestibular sensory organs produce nerve action potentials at a high tonic rate of firing that propagate to the brain stem.²⁰ A unilateral disorder that rapidly disrupts this tonic firing rate causes an imbalance between the right and left vestibular afferents that leads to severe vertigo. Bilateral vestibular disorders, such as ototoxicity, usually do not produce vertigo, because there is a minimal imbalance of tonic activity. A slowly evolving unilateral process, such as an acoustic neuroma, generally does not produce vertigo, because the imbalance in vestibular afferents occurs gradually and is compensated for by the central nervous system.^{5,6,21,22}

Peripheral vestibular afferents innervate the brain-stem vestibular nuclei. The vestibular nuclei project to oculomotor, spinal, cerebellar, and cerebral areas.^{16,20} The vestibulo-ocular connections cause smooth movements of the eyes in the opposite direction to that of rotational and translational movements of the head, allowing stable visual fixation during movements of the head.²³ The peripheral and central vestibular structures are also critical in maintaining vestibulospinal reflexes and postural stability.^{23,24} Unilateral labyrinthectomy and sectioning of the eighth nerve attenuate ipsilateral vestibular afferent signals and produce nystagmus, with the fast phase in the direction away from the side with the lesion. There is also a tendency to fall or tilt toward the side with the lesion.²³⁻²⁵ These conditions rapidly improve as a result of adaptation by central pathways.²²

The cerebellum receives projections from the vestibular labyrinth, the vestibular nerve, and the brain-stem vestibular nuclei and projects back to the vestibular nuclei to control oculomotor and postural reflexes.²⁶ The cerebellum is involved in the visual suppression of vestibulo-ocular responses, including the nystagmus caused by acute, unilateral, peripheral vestibular dysfunction.²⁷

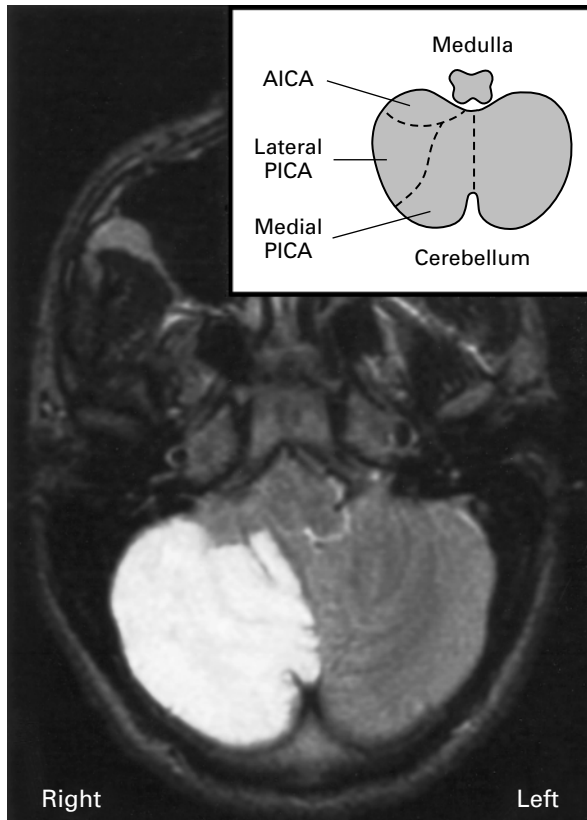


Figure 1. Magnetic Resonance Image of a Right Inferior Cerebellar Infarction in a Man with Acute Vertigo, Vomiting, Nystagmus Elicited by Right, Left, or Upward Gaze, and Severe Gait Instability.

The axial T₂-weighted image shows a high-intensity signal (white) in the right inferior cerebellum, with displacement of the medulla. Neurosurgical intervention was required because of brain-stem compression with noncommunicating hydrocephalus and progressive deterioration in mental status. On the day after surgery, the patient's mental status rapidly improved. Over the next week, his nystagmus resolved and he began walking. The inset illustrates the vascular supply to the inferior cerebellum. The inferior cerebellum is perfused by the medial and lateral branches of the posterior inferior cerebellar artery (PICA) and the anterior inferior cerebellar artery (AICA).^{7,8}

The blood supply to the peripheral vestibular labyrinth and vestibular nerve as well as the brain-stem vestibular nuclei and cerebellum comes from the verteobasilar arterial system. The internal auditory artery, which is usually a branch of the anterior inferior cerebellar artery, supplies the vestibular and auditory labyrinth (Fig. 2).^{5,17} Branches from the vertebral and basilar arteries supply the brain-stem vestibular nuclei. The posterior and anterior cerebellar arteries provide the blood supply to the inferior cerebellum and the flocculonodular lobe, the parts of the cerebellum most closely related to the vestibular system (Fig. 1). Although the posterior inferior cerebellar artery usually provides most of the blood,

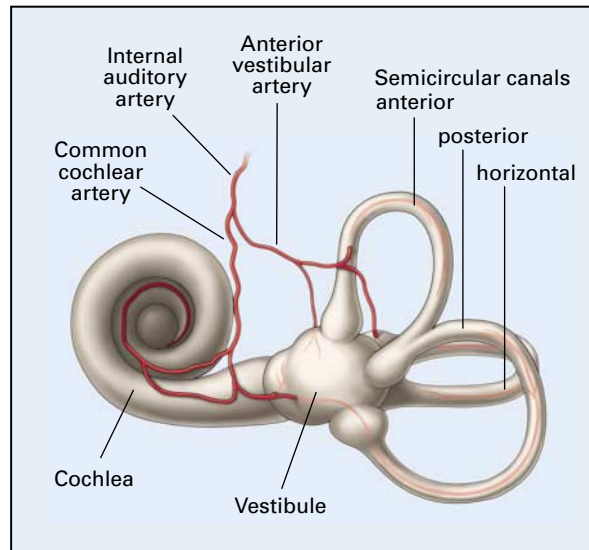


Figure 2. Schematic Drawing of the Bony Labyrinth Containing the Vestibular and Auditory Sensory Organs.

The otolithic organs (utricle and saccule) lie in the vestibule. The internal auditory artery divides into the common cochlear artery and the anterior vestibular artery. The anterior vestibular artery provides the blood supply to the anterior and horizontal semicircular canals but not to the cochlea. Isolated occlusion of the anterior vestibular artery may therefore cause acute vestibular syndrome without hearing loss.^{5,8,16,17}

the vascular supply of the inferior cerebellum is variable.^{7,8,14}

EVALUATION OF PATIENTS WITH ACUTE VESTIBULAR SYNDROME

Vertigo and Its Differential Diagnosis Based on Time Course

Vertigo, an illusion of movement, is the cardinal symptom of vestibular dysfunction. Vertigo is typically rotational, but it can be an illusion of tilting to one side or swaying. Rotational vertigo may indicate a disease of the semicircular canals or their central connections. A feeling of tilting or linear displacement may occur in disorders affecting the otolithic organs or their projections. It is common for acute vertigo to cause a feeling of imbalance during standing or walking. Patients want to lie still and avoid movement. Acute vertigo is accompanied by nausea, vomiting, and autonomic distress of varying degrees of severity. Different causes of acute vertigo can be distinguished by the time course, duration, and recurrence of the illusion of movement.

Vertigo Lasting for a Day or Longer

Vestibular neuritis typically begins over a period of a few hours, peaks in the first day, and then improves within days.^{4,6} Disabling vertigo usually resolves within a week, but it may be followed by a

sensation of unsteadiness or transient episodes of dizziness. Complete recovery from the symptoms usually occurs within weeks to months.⁴ Proof of a viral or postviral inflammation that caused a vestibular neuritis is usually lacking. Acute vertigo is preceded by or associated with a viral illness in less than half of patients.^{1,4} Since vestibular neuritis has a benign outcome, pathological confirmation of a viral process is rare.^{3,5,28,29}

Ischemia in the vertebrobasilar-artery system can produce infarction of the labyrinth, the brain stem, the cerebellum, or some combination of these sites. Infarction causes a vestibular syndrome that typically has an abrupt onset in patients with risk factors for stroke, such as hypertension, diabetes mellitus, smoking, known occlusive vascular disease, or myocardial abnormalities, including atrial fibrillation and valvular heart disease.^{8,11,15,30-32} Disabling vertigo may remain for days; it usually begins to improve within the first week of symptoms and then gradually resolves within weeks to months.

Acute vertigo from a brain-stem stroke is usually accompanied by additional evidence of vertebrobasilar ischemia, such as diplopia, reduced vision, dysarthria, dysphagia, and focal sensory or motor deficits. These additional findings distinguish a brain-stem stroke from vestibular neuritis. Infarction and hemorrhage of the inferior cerebellum, however, can cause vertigo, nystagmus, and postural instability, with few additional symptoms that distinguish this condition from vestibular neuritis.⁹⁻¹¹

Multiple sclerosis may also produce a vestibular syndrome that evolves over a period of hours to days.³³ There is usually a history of multiple neurologic events or evidence of multiple white-matter lesions on brain imaging.

Vertigo Lasting for Hours or Minutes

Patients with Meniere's syndrome occasionally present with an isolated episode of severe vertigo that lasts for hours and is followed by a sensation of unsteadiness and dizziness for days. Typically, however, the vertigo is preceded or accompanied by reduced hearing, tinnitus, and a feeling of pressure in the ear. Over time, the attacks of Meniere's syndrome recur, and fluctuations in hearing and episodes of tinnitus may be followed by a residual, low-frequency, sensorineural hearing loss. Fluctuating hearing levels associated with recurrent episodes of vertigo are central to the diagnosis of Meniere's syndrome.^{6,34}

The abrupt onset of isolated vertigo lasting for minutes in a person with risk factors for stroke suggests a transient ischemic attack in the vertebrobasilar system, including transient ischemia of the vestibular labyrinth.³⁰⁻³² Transient ischemic attacks often last for less than 30 minutes. Isolated transient vertigo may precede a stroke in the branches of the vertebrobasilar artery by weeks or months. Such ep-

isodic vertigo may also occur with migraine headaches.^{35,36} In rare cases, patients with partial seizures evolving from areas of cerebral cortex that receive vestibular projections present with isolated vertigo lasting for minutes.³⁷ Episodes of vertigo precipitated by exertional straining or rapid change in air pressure or following otologic surgery may be due to a perilymph fistula that produces an abnormal connection between the inner and middle ears.³⁸

Vertigo Lasting for Seconds

Abrupt onset of vertigo for seconds after a rapid change in head position is characteristic of benign positional vertigo, which usually lasts for less than a minute. Benign positional vertigo, the most common type of vertigo, is caused by a clot of free-floating debris, usually in a posterior semicircular canal. Rapid head movement produces movement of the debris within the semicircular canal and perturbs the vestibular sensory receptors, causing transient vertigo. The diagnosis can be confirmed by the induction of paroxysmal vertigo and combined torsional and vertical nystagmus when the patient is rapidly taken from an erect sitting position to a supine position with the head hanging. A simple positioning maneuver at the bedside can usually cure benign positional vertigo.^{1,39,40}

Distinguishing Vestibular Neuritis from Stroke

The clinical examination of patients with acute vertigo helps differentiate a peripheral vestibular disorder, such as vestibular neuritis, from a brain-stem or cerebellar stroke. The type of nystagmus, the severity of postural instability, and the presence or absence of additional neurologic signs are the main distinguishing factors. A peripheral vestibular lesion may occasionally be accompanied by hearing loss if the cochlear labyrinth or nerve is also involved; the tympanic membrane is usually normal on examination, unless there is an underlying bacterial otomastoid infection.

Acute unilateral disorders of the peripheral vestibular labyrinth or nerve cause spontaneous nystagmus that continues in the same direction when the direction of gaze changes (Fig. 3).^{2,4} The direction is typically horizontal, with a torsional component.⁴¹ As with all types of jerk nystagmus, peripheral vestibular nystagmus increases in intensity when the gaze is in the direction of the fast phase, and decreases in intensity when the gaze is away from the fast phase (Alexander's law). Although the intensity of nystagmus changes with the direction of gaze, its predominant direction remains the same. The intensity of peripheral vestibular nystagmus and the velocity of its slow phase are attenuated by visual fixation and increased by removing fixation (Fig. 3).^{25,27,42-44} A subtle peripheral vestibular nystagmus may be present only during fixation in the direction of its fast phase.

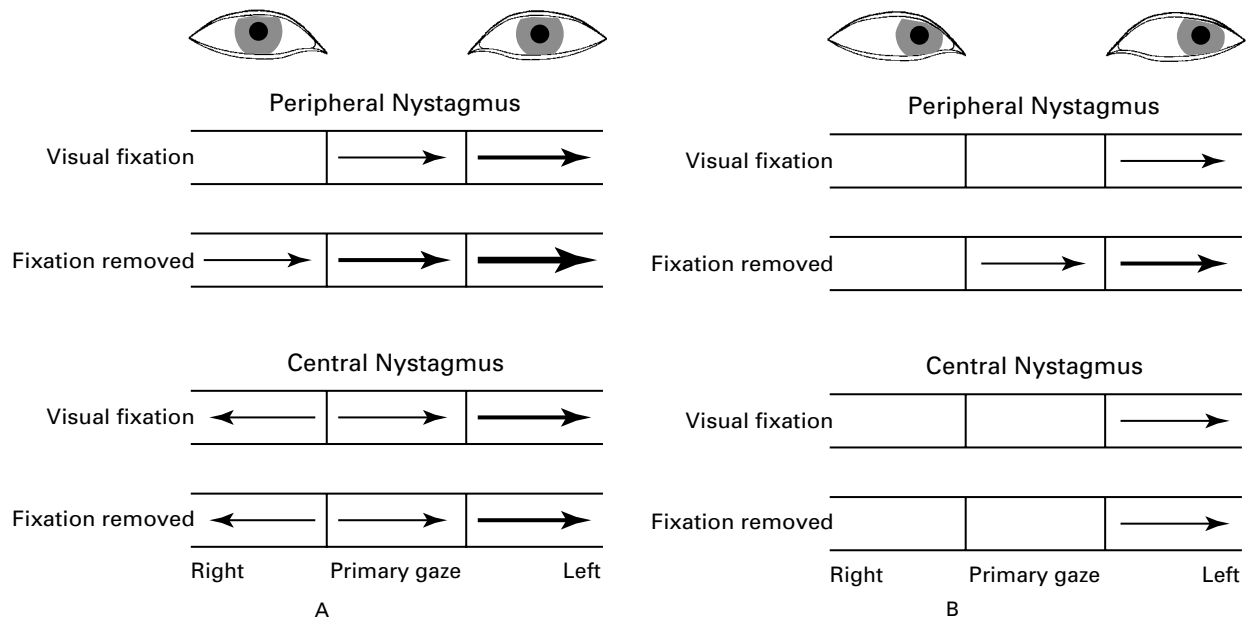


Figure 3. Schematic Drawing of Peripheral and Central Vestibular Nystagmus with and without Visual Fixation.

The direction of the arrows indicates the horizontal direction of the fast phase of the nystagmus (a torsional component is not shown). The thickness of the arrows represents the relative intensity of the nystagmus. Panel A shows findings typical of peripheral nystagmus, which remains in the same direction when the direction of gaze changes, and central nystagmus, which changes direction when the direction of gaze changes. Removal of visual fixation increases the intensity of peripheral nystagmus but not of central nystagmus. Panel B illustrates how removal of fixation helps to differentiate peripheral from central nystagmus when the nystagmus is predominantly in one direction of gaze during fixation. With removal of fixation, peripheral nystagmus may increase in intensity and become apparent in more than one direction of gaze.

If this subtle nystagmus is not observed both with and without visual fixation, the nystagmus may be misinterpreted as representing a central disorder (Fig. 3B).⁴⁵

The effect of fixation on the nystagmus can be determined at the bedside by observing the change in intensity of the nystagmus when the patient is looking through Frenzel glasses. These glasses consist of +30-diopter binocular lenses that prevent visual fixation. The effect of fixation can also be determined by viewing the nystagmus with an ophthalmoscope focused on the optic disk or retinal vessels of one eye while the patient covers and uncovers the fixating eye (the direction of the nystagmus is inverted when it is viewed through an ophthalmoscope).⁴⁶ If the intensity of the nystagmus and the velocity of its slow phase are increased by covering the fixating eye, the nystagmus is consistent with the presence of a peripheral vestibular disorder.^{25,42-44}

Acute central vestibular disorders, such as infarction or hemorrhage of the brain stem or the cerebellum, may cause spontaneous nystagmus that changes its direction with a change in the direction of gaze (gaze-evoked nystagmus). However, in patients with cerebellar stroke, nystagmus may be present only when the patient is gazing in one direction, thereby appearing similar to a peripheral vestibular nystag-

mus. Purely vertical nystagmus and purely torsional nystagmus are almost always due to a central disorder, whereas horizontal and torsional components may occur simultaneously in patients with either peripheral or central disorders. Visual fixation may have little effect on the intensity of central vestibular nystagmus (Fig. 3).^{43,45}

Acute unilateral disorders of the peripheral vestibular labyrinth or nerve produce a tendency to lean or fall in one direction. Most patients are extremely uncomfortable and reluctant to move. When requested, however, they are still able to walk, though with a tendency to veer to one side. During Romberg testing, patients are instructed to allow themselves to lean or fall in the direction in which they feel like leaning or falling. This instruction is important in order to avoid complicating compensatory movements. Falling or tilting to the side opposite to the direction of the fast phase of nystagmus (i.e., toward the side of the lesion) is a characteristic finding in a peripheral vestibular disorder.²³⁻²⁵ In contrast, patients with acute cerebellar stroke are often unable to walk without falling, and with the Romberg test the direction of tilting or falling may be variable.^{7,9,10}

Additional neurologic findings on examination differentiate a peripheral vestibular lesion from a brain-stem disorder. The presence of cranial-nerve signs,

motor weakness, prominent dysmetria, sensory changes, or abnormal reflexes suggests a central process. For example, an occlusion of the anterior inferior cerebellar artery can produce both vertigo and hearing loss that mimics neurolabyrinthitis, except that concomitant brain-stem or cerebellar signs are almost always present.^{8,14} The absence of additional neurologic findings, however, does not exclude the possibility of a stroke limited to the inferior cerebellum. Dysmetria, a major finding of the cerebellar system, may be minimal or absent after an inferior cerebellar stroke.⁹

In summary, the type of nystagmus and the severity of postural instability can help to differentiate a peripheral vestibular disorder from an inferior cerebellar stroke. The former produces nystagmus that remains in the same direction when the direction of gaze changes and is suppressed by visual fixation. The latter causes other forms of nystagmus. A peripheral vestibular lesion produces unidirectional postural instability with preserved walking, whereas an inferior cerebellar stroke often causes severe postural instability and falling when walking is attempted.

EVALUATION AND MANAGEMENT

The first question when a patient presents with the acute vestibular syndrome is whether the history and findings on examination are consistent with a central disorder, including hemorrhage or infarction of the cerebellum. Immediate brain imaging is mandatory when a central process is likely, particularly to rule out an evolving cerebellar hematoma that would require emergency neurosurgical intervention.

Brain imaging is recommended when the examination of a patient with the acute vestibular syndrome does not result in the findings that are typical of a peripheral vestibular disorder. Imaging is also recommended if the onset of symptoms is sudden in a patient with prominent risk factors for stroke or if there is a new, severe headache accompanying the acute vertigo.

The decision whether to perform brain imaging can be deferred for 48 hours if the patient has isolated acute vertigo, has peripheral vestibular nystagmus that is suppressed by visual fixation, and is unstable but can still walk. If there is substantial improvement in 48 hours, the syndrome is consistent with a vestibular neuritis, and brain imaging is not necessary. If there is concurrent acute hearing loss, a peripheral disorder is even more likely, but when hearing loss is present, further evaluation of underlying treatable otologic diseases is indicated.^{5,47-49}

When immediate brain imaging is indicated, magnetic resonance imaging and angiography are preferred. During the first day of vertigo, routine magnetic resonance imaging is a sensitive method to detect an inferior cerebellar infarction, but it may be less sensitive for identifying a hemorrhage. Imaging

sequences that maximize the identification of both infarction and hemorrhage in the posterior fossa should therefore be requested.

If prompt magnetic resonance imaging and angiography are not available, then computed tomography of the brain should be performed, with fine cuts through the cerebellum and clear visualization of the fourth ventricle, to rule out a cerebellar hemorrhage. Computed tomographic scans of the cerebellum are usually normal in the first hours after an infarction in the cerebellum, although asymmetry in the fourth ventricle may be an early sign of swelling. If immediate brain imaging is indicated and a normal computed tomographic scan is obtained on the first day of acute vertigo, then subsequent magnetic resonance imaging and angiography are recommended. The patient's neurologic status should be closely monitored in the interval.

In patients with cerebellar infarctions, cerebellar swelling may develop that compresses the brain stem and produces noncommunicating hydrocephalus (Fig. 1).^{9,12} This complication rapidly evolves in the first few days after a stroke. Therefore, patients with cerebellar infarction should be examined frequently in an intensive care setting and brain imaging should be repeated. If deterioration occurs, then neurosurgical intervention is recommended.^{9,13,50} Once the patient is stable, treatable causes of recurrent stroke can be sought.¹⁵

Healthy young people who present with acute vertigo, peripheral vestibular nystagmus, and postural instability usually have a benign, self-limited process. Persons with risk factors for stroke who do not have the typical findings of a peripheral vestibular disorder should be evaluated promptly for a potentially serious cerebellar stroke.

We are indebted to Dr. Andrew Koo for assistance with Figure 1.

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