

CLINICAL PRACTICE

Vestibular Neuritis

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This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.

A 53-year-old man awoke in the morning with acute dizziness. He staggered to the bathroom, where he vomited repeatedly. When he was seen at a local emergency room 12 hours later, he had left-beating nystagmus in all positions of gaze but otherwise no focal neurologic findings. How should he be evaluated and treated?

THE CLINICAL PROBLEM

Acute spontaneous vertigo results from an imbalance in tonic vestibular activity. The patient has an intense sensation of rotation that is aggravated by head motion and change of position. It is difficult to stand and to walk, and there is a tendency to veer toward the affected side. Autonomic symptoms including malaise, pallor, sweating, nausea, and vomiting are nearly always present.

The first task of the examining physician is to determine whether the vertigo is of central or peripheral origin, since some central causes of acute vertigo, such as cerebellar hemorrhage or infarction, can be life-threatening and may require immediate intervention.¹ This differentiation can usually be made at the bedside on the basis of the type of the spontaneous nystagmus, the results of the head-thrust test (described below), the severity of the imbalance, and the presence or absence of associated neurologic signs. Spontaneous nystagmus of peripheral origin is typically horizontal with a torsional (rotational) component; it does not change direction with a change in gaze. By contrast, spontaneous nystagmus of central origin is often purely horizontal, vertical, or torsional and usually changes direction with changes in the position of the gaze.

The head-thrust test is a simple bedside test of the horizontal vestibulo-ocular reflex.² It is performed by grasping the patient's head and applying a brief, small-amplitude, high-acceleration head turn, first to one side and then to the other. To start, the eyes should be about 10 degrees away from the primary position in the orbit so that after a 10-degree head turn, the eyes will be near the primary position. The patient fixates on the examiner's nose and the examiner watches for corrective rapid eye movements (saccades), which are a sign of decreased vestibular response (i.e., the eyes move with the head rather than staying fixed on the nose). If "catch-up" saccades occur after head thrusts in one direction but not after those in the other direction, this indicates the presence of a peripheral vestibular lesion on that side (in the labyrinth or the 8th nerve including the root's entry zone in the brain stem).

Patients with an acute peripheral vestibular lesion typically can stand, although they will veer toward the side of the lesion. By contrast, patients with vertigo of central origin are often unable to stand without support. Associated neurologic signs such as dysarthria, incoordination, numbness, or weakness suggest a central origin.

The syndrome of acute, prolonged vertigo of peripheral origin is commonly called vestibular neuritis, although other terms such as "vestibular neuronitis," "labyrinthitis," "neurolabyrinthitis," and "unilateral vestibulopathy of unknown cause" have also been

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used.³ The vertigo typically develops over a period of hours, is severe for a few days, and then subsides over the course of a few weeks. Some patients can have residual nonspecific dizziness and imbalance that lasts for months. The condition is thought to result from a selective inflammation of the vestibular nerve, presumably of viral origin. The facts that the disorder often has a viral prodrome, that it occurs in epidemics, that it may affect several members of the same family, and that it occurs more commonly in spring and early summer all support a viral cause.³ Postmortem studies have found atrophy of the vestibular nerve and the vestibular sensory epithelium that is similar to the pathological findings with known viral disorders of the inner ear, such as measles and mumps.^{4,5} Several viruses selectively infect the labyrinth, the 8th nerve, or both in animal models.^{6,7}

A common feature of vestibular neuritis is selective damage to the superior part of the vestibular

labyrinth (horizontal and anterior semicircular canals and utricle) supplied by the superior division of the vestibular nerve, with sparing of the inferior part (posterior semicircular canal and saccule) supplied by the inferior division.⁸ Benign paroxysmal positional vertigo (originating from the posterior semicircular canal) often develops as a sequela even if the patient has no remaining function in the horizontal or anterior semicircular canal.^{3,9} Selective inflammation of the superior division of the vestibular nerve¹⁰ or anatomical differences in the bony canals of the two divisions¹¹ might explain this relative vulnerability.

STRATEGIES AND EVIDENCE

DIAGNOSIS

As indicated above, the key differentiation is between peripheral and central causes of acute, prolonged vertigo (Table 1). On the basis of the appear-

Table 1. Differential Diagnosis of Common Causes of Acute, Prolonged Vertigo.*

| Cause | History | Physical Examination | Laboratory Testing† |
|--------------------------------------|--|--|---|
| Vestibular neuritis | Develops over a period of hours; resolves over a period of days; may follow an influenza-like illness | Spontaneous “peripheral” nystagmus; positive head-thrust test; imbalance | Electronystagmography: unilateral caloric hypoexcitability Audiography: normal Brain imaging: normal |
| Labyrinthitis | Develops over a period of minutes to hours; may be associated with systemic, ear, or meningial infection | Same as for vestibular neuritis, but also unilateral hearing loss | Electronystagmography: unilateral caloric hypoexcitability Audiography: moderate-to-severe ipsilateral sensorineural hearing loss Brain imaging: normal |
| Labyrinthine infarction | Abrupt onset; previous vascular disease; may be associated with neurologic symptoms | Same as for vestibular neuritis, but also unilateral hearing loss; may be associated with neurologic signs | Electronystagmography: absence of a unilateral caloric response Audiography: severe ipsilateral sensorineural hearing loss Brain imaging: MRI may show silent brain infarcts |
| Perilymph fistula | Abrupt onset associated with head trauma, barotrauma, or sudden strain during heavy lifting, coughing, or sneezing; may be associated with chronic otitis with cholesteatoma | Same as for vestibular neuritis, but usually associated with unilateral hearing loss; possible perforation of the tympanic membrane; positive fistula test (vertigo and nystagmus induced by pressure in external ear canal) | Electronystagmography: unilateral caloric hypoexcitability Audiography: mild-to-moderate ipsilateral sensorineural hearing loss Brain imaging: CT of temporal bone may show erosion from cholesteatoma |
| Brain-stem and cerebellar infarction | Abrupt onset, history of transient ischemic attacks and vascular disease; usually associated with neurologic symptoms | Spontaneous “central” nystagmus; positive head-thrust test if entry zone of root of 8th nerve is involved; usually focal neurologic signs | Electronystagmography: unilateral caloric hypoexcitability if entry zone of root is involved Audiography: ipsilateral hearing loss if anterior inferior cerebellar artery is involved Brain imaging: MRI shows infarction in medulla, pons, or cerebellum |

* Prolonged vertigo is defined as vertigo lasting more than a few hours.

† Expected results of these tests are given. However, it should be noted that these tests are not routinely recommended, except for magnetic resonance imaging (MRI) of the brain, if a central cause is suspected. CT denotes computed tomography.

ance of the nystagmus, a positive head-thrust test, and a negative neurologic examination, one can usually be confident in the diagnosis of a unilateral peripheral vestibulopathy. Electronystagmography, if available, can document the unilateral vestibular loss (i.e., unilateral caloric hypoexcitability) but is rarely necessary. Currently, viral studies (serologic analysis or cultures) are not recommended, since they cannot prove a causal relation between a viral infection and the vestibular syndrome.

When there is associated unilateral hearing loss, inner-ear disorders such as labyrinthitis, labyrinthine infarction, and perilymph fistula should be considered. Meniere's syndrome can initially present with vertigo alone, but attacks rarely last longer than four to five hours. The diagnosis requires recurrent attacks with associated hearing loss. A positive head-thrust test can occur with brain-stem infarction involving the entry zone of the root of the 8th nerve, but invariably, there will be other associated signs of the lateral brain stem (e.g., Horner's syndrome, facial numbness and weakness, hemiataxia, and dysarthria). Magnetic resonance imaging of the brain is indicated if there are associated neurologic symptoms and signs, if the onset is sudden in a patient with risk factors for stroke, or if there is a new, severe headache accompanying the vertigo.¹

THErapy

Of course, the best approach would be to treat the underlying disease, but because the pathophysiology is uncertain and there is no established treatment, symptomatic therapy is typically used. The main classes of drugs used for symptoms of acute vertigo include antihistamines, anticholinergic agents, antidopaminergic agents, and γ -aminobutyric acid-enhancing (GABAergic) agents (Table 2).³ These drugs do not eliminate but rather reduce the severity of vertiginous symptoms. They are effective in most patients with vestibular neuritis, but there have been few controlled studies comparing them in terms of efficacy.¹² Two recent randomized, clinical trials, one comparing intravenous dimenhydrinate (50 mg) with lorazepam (2 mg)¹³ and the other comparing intramuscular dimenhydrinate (50 mg) with droperidol (2.5 mg)¹⁴ for the treatment of acute peripheral vertigo in patients in the emergency department, found that dimenhydrinate was more effective than lorazepam and that dimenhydrinate and droperidol were equally effective. During the acute phase, because of severe nausea and decreased gastric motility, the intramuscular

or intravenous route is usually preferable. The response is clearly dose-dependent, so if the initial dose (Table 2) is not effective, higher doses should be tried.

Although the exact mechanism of action of these drugs is unclear, they act at the level of the neurotransmitters involved in the propagation of impulses from primary to secondary vestibular neurons and in the maintenance of tone in the vestibular nuclei. They also act on the areas of the nervous system that control vomiting, including central components loosely described as the "emetic center" and peripheral components in the gastrointestinal tract. All the medications can be sedating, so they should not be used when patients are engaged in activities that require a high level of alertness, such as driving, operating machinery, or participating in athletic activities. Less sedating drugs such as oral meclizine and transdermal scopolamine are useful for milder vertigo and prevention of motion sickness. Because of the multiple effects of each of these drugs, possible drug interactions should always be considered before use (Table 2).

Recovery from a peripheral vestibular lesion results from a combination of the restoration of peripheral labyrinthine function (which is usually incomplete in the case of vestibular neuritis)¹⁵ and central vestibular compensation for the imbalance in vestibular tone. In other words, patients will typically get better even if they have a permanent unilateral loss of vestibular function. Recovery from vestibular neuritis typically takes several weeks, although longer periods of recovery are not uncommon. Clinicians have long felt that vestibular compensation occurs more rapidly and is more complete if the patient begins exercising as soon as possible after the occurrence of a vestibular lesion.¹⁶ The goal of vestibular exercises is to accelerate the process of vestibular compensation and improve the final level of recovery. Controlled studies in animals¹⁷ and humans^{18,19} indicate that exercising can accelerate the recovery of balance after a peripheral vestibular lesion, but data are lacking with regard to the final level of recovery. In animals, compensation seems to be accelerated by stimulant drugs (e.g., amphetamine) and slowed by sedating drugs (e.g., diazepam).²⁰ Whether more frequent exercise leads to faster improvement is unknown.

A vestibular exercise program typically includes exercises designed to improve ocular stability and balance.²¹ While nystagmus is present, the patient should try to suppress it with fixation in all posi-

Table 2. Drugs Commonly Used for Symptomatic Treatment of Acute Vertigo.*

| Drug | Usual Starting Dose | Range of Doses and Frequency of Administration† | Antiemetic Action | Common Precautions | Common Side Effects | Common Drug Interactions |
|------------------------------|--|---|-------------------|---|--|---|
| Dimenhydrinate (Dramamine) | 50 mg IM, IV, or orally | 25–100 mg every 4–8 hr | Moderate | Asthma, glaucoma, proptate enlargement | Dryness, drowsiness | Alcohol, hypnotics, antidepressants, sedatives, tranquilizers |
| Promethazine (Phenergan) | 25 mg IM, IV, orally, or by suppository | 12.5–50 mg every 4–8 hr | Moderate | Same as for dimenhydrinate | Same as for dimenhydrinate | Same as for dimenhydrinate |
| Mecizine (Antivert, Bonine) | 25 mg orally | 12.5–50 mg every 4–8 hr | Mild | Same as for dimenhydrinate | Same as for dimenhydrinate | Same as for dimenhydrinate |
| Scopolamine (Transderm-Scop) | 0.2 mg IM or orally Transdermal patch | 0.1–0.4 mg every 4–6 hr 1.5 mg over a 3-day period | Moderate | Same as for dimenhydrinate | Dryness, visual blurring, memory loss, confusion in elderly patients | Alcohol, antidepressants, antihistamines, belladonna alkaloids |
| Droperidol (Inapsine) | 2.5 mg IM or IV | 2.5–10 mg every 3–4 hr | Prominent | Liver or kidney disease | Drowsiness, extrapyramidal reactions | Antidepressants, barbiturates, spinal and peridural anesthetics |
| Prochlorperazine (Compazine) | 10 mg IM, IV, orally, or by suppository | 5–20 mg every 4–12 hr | Prominent | Same as for droperidol | Same as for droperidol | Alcohol, anesthetics, propranolol, phenytoin anticoagulants, levodopa, thiazide diuretics |
| Diazepam (Valium) | 5 mg IM, IV, or orally‡ | 2–20 mg every 4–8 hr | Mild | Glaucoma, additive with other CNS depressants | Drowsiness | Alcohol, phenothiazines, barbiturates, antidepressants, scopolamine |
| Lorazepam (Ativan) | 1 mg IM, IV, or orally‡ | 0.5–2 mg every 4–8 hr | Mild | Same as for diazepam | Same as for diazepam | Same as for diazepam |

* IM denotes intramuscularly, IV intravenously, and CNS central nervous system.

† It is recommended that the following doses not be exceeded in adults during a 24-hour period: 200 mg of dimenhydrinate, 75 mg of promethazine, 150 mg of meclizine, 1.2 mg of scopolamine (orally), 30 mg of droperidol, 60 mg of prochlorperazine, 60 mg of diazepam, and 6 mg of lorazepam. Smaller doses are used in children and elderly persons.

‡ With intravenous use, equipment to maintain patent airway should be available.

tions of gaze. As the nystagmus diminishes, eye-and-head-coordination exercises can be started (e.g., staring at a visual target while oscillating the head from side to side and up and down). Combined movements of the eyes and head involving jumping quickly back and forth between two widely separated targets are also useful. The patient should try to stand and walk in contact with a wall or with assistance in the early stages. As improvement occurs, head movements should be added while the patient is standing and walking; these head movements should be slow at first and later rapid and in all directions. Balance exercises such as walking with one foot placed directly in front of the other or walking on a narrow beam can then be added.

AREAS OF UNCERTAINTY

The best treatment for a patient who presents with an acute episode of peripheral vestibular loss is controversial, because the pathophysiology is often uncertain. Assuming that vestibular neuritis is the result of a viral or postviral inflammation of the vestibular nerve, treatment aimed at stopping the inflammation has been proposed. The combination of corticosteroids and an antiviral agent such as acyclovir has been reported to be effective in treating Bell's palsy²² and herpes zoster infection involving the 7th and 8th cranial nerves.²³ There are limited data to support such strategies for the treatment of vestibular neuritis. In one small controlled study,²⁴ corticosteroids were more effective than placebo in treating the acute symptoms of vestibular neuritis. In addition, a randomized, double-blind trial²⁵ comparing corticosteroids plus placebo with corticosteroids plus acyclovir for the treatment of idiopathic, sudden sensorineural hearing loss (a disorder thought to represent the auditory-nerve equivalent of vestibular neuritis) revealed no significant difference in hearing in the two groups at one-year follow-up.

A clear problem with studies that have attempted to evaluate treatments for vestibular neuritis is that there are no well-accepted diagnostic criteria for the disorder. By including anyone with isolated attacks of vertigo, these studies may include a range of different conditions. As Furstenberg and colleagues noted in 1934,²⁶ "The tendency to create a disease entity by grouping a number of cases having one symptom in common is one of the temptations of medical practice. Vertigo lends itself admirably to this evil."

GUIDELINES

No major medical organization has proposed specific guidelines for the diagnosis and treatment of vestibular neuritis.

CONCLUSIONS AND RECOMMENDATIONS

The diagnosis of vestibular neuritis rests on the history of spontaneous, prolonged vertigo, findings on physical examination that are consistent with a unilateral peripheral vestibulopathy, and the absence of other neurologic symptoms and signs. The first line of treatment is to suppress vertigo and nausea (Table 2). For the patient in the vignette, I would begin with 25 mg of promethazine intramuscularly, which is usually effective for suppressing vertigo and vomiting. If the vomiting persists, I would add 10 mg of prochlorperazine intramuscularly. Once the vomiting subsides, less sedating oral antivertiginous drugs such as 25 mg of meclizine or 50 mg of dimenhydrinate can be used, but only for a few days, until the acute vertigo subsides. The commonly used oral vestibular suppressants require 20 to 30 minutes for action to be initiated, reach a peak plasma level in 1 to 2 hours, and have a half-life of about 8 hours.

Vestibular exercises should be started when the acute stage of nausea and vomiting has ended.²⁴ Many of the exercises will result in dizziness. This sensation is a necessary stimulus for compensation; antivertiginous medications should be avoided as much as possible in order to maximize the beneficial effect. Exercises should be done for several minutes at least twice daily but may be done as often as the patient can tolerate.

Although corticosteroids and antiviral agents have been suggested for treatment of vestibular neuritis, so far there have been no definitive studies demonstrating the efficacy of these medications. Since the majority of patients will have spontaneous improvement and will be able to return to normal activities, at present, I would only recommend symptomatic treatment of vertigo followed by a program of vestibular exercise while we await the results of more definitive clinical trials.

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