CME CREDIT

Benign paroxysmal positional vertigo: How to diagnose and quickly treat it

JUDITH WHITE, MD, PhD
Program of Vestibular and Balance Disorders, Department of Otolaryngology and Communicative Disorders, The Cleveland Clinic Foundation

ABSTRACT

Benign paroxysmal positional vertigo, the most common cause of vertigo, can be diagnosed and treated with a simple maneuver that can quickly be performed in the primary care physician's office. How to diagnose and manage other causes of dizziness, including Meniere disease, acute vestibular syndrome, migraine-associated vertigo, and motion sickness, is also covered in this article.

Patients with BPPV feel that the room is spinning

NOT LONG AGO, we advised patients with benign paroxysmal positional vertigo, the most common cause of vertigo, that it would resolve over time. Unfortunately, that did not always happen quickly. Now, however, we know that a maneuver performed in the office that takes just a few minutes can instantly cure the problem in the majority of patients.

This article discusses how to evaluate patients who complain of dizziness and how to perform the maneuver for patients with benign paroxysmal positional vertigo (BPPV). We also discuss other common causes of dizziness, how to diagnose and manage them, and when to refer patients to a specialist or a physical therapist.

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This paper discusses therapies that are experimental or are not approved by the US Food and Drug Administration for the use under discussion.

PINPOINTING DIZZINESS

Dizziness is the most common reason that adults over age 75 see their doctor, and 16% of people in the United States report that they have a problem with chronic dizziness.

"Dizziness," however, is a nonspecific symptom. Clinicians should ask patients the following questions to narrow down the differential diagnoses that go with each complaint:

- During a dizzy spell, does an object on the wall appear to be moving?
- Are you light-headed? Do you feel faint, or feel that you are going to faint?
- When you walk, do you stumble about or feel you can't control your movements?

VERTIGO: AN ILLUSION OF MOVEMENT

A sense that the room is spinning is the hallmark of vertigo. Visual stability is controlled by:

- What our eyes see
- Proprioception—the perception of our head's angle relative to gravity
- The vestibular system (the inner ear with its three semicircular canals), which detects head motion and signals the eyes to move to maintain stability.

Vertigo is typically a problem with the inner ear, either with the peripheral inner ear or less commonly from a central mismatch of our visual, proprioceptive, and vestibular systems. People who complain of dizziness are less likely to have problems with proprioception or vision: proprioception does not usually fail acutely, and if vision fails, the visual loss is the primary complaint.

Why benign paroxysmal positional vertigo occurs

The three semicircular canals in the ear are filled with fluid. As the fluid moves, small nerves bend, signaling movement to the brain. Also inside the inner ear are small stones, called otoconia, statoliths, or canaliths, which sense gravity. These particles are normally contained in the saccule and utricle—special dilations of the inner ear. However, after a head injury, inner ear infection, or with age, the stones can break loose and fall into a semicircular canal, most often the posterior one. When a patient lies back or has a sudden head movement, the stones move and disturb the nerves, giving inaccurate information that the head is spinning. The eyes rapidly move to catch up, causing a rotary nystagmus.

BENIGN PAROXYSMAL POSITIONAL VERTIGO IS COMMON

Oghalai et al[1] did a cross-sectional study of an inner-city geriatric population and found that 61% complained of dizziness and 9% of them had unrecognized BPPV, as diagnosed by a positive Dix-Hallpike test. The US Centers for Disease Control and Prevention are currently conducting a national survey on dizziness and balance because of their contribution to the risk of falling in the elderly.

Kroenke et al[2] studied 100 outpatients who complained of at least 2 weeks of dizziness. Fifty-four had vertigo, and most of these had BPPV. Other causes of vertigo were Meniere disease, peripheral vestibulopathy, and, very rarely, central vertigo. Causes other than vertigo included effects of medications, anxiety, or multiple problems. No brain tumors or cardiac arrhythmias were found.

HISTORY IS IMPORTANT

Before patients come in to our clinic, we mail them a request for old records, evaluations, and imaging studies. We also send a questionnaire that asks about the onset, character, frequency, and duration of the dizziness; what provokes it; and whether hearing loss, roaring, or tinnitus is associated with it.

Patients with BPPV frequently experience dizziness when they lie down and get out of bed, look up quickly, or bend over. They often complain that it occurs during haircuts and dental appointments. Spells last for seconds to a few minutes only. Afterwards, they may feel lightheaded, nauseous, or fatigued, but the vertigo has stopped. In other vestibular disorders, vertigo may persist for hours or days.

Meniere disease or associated retrocochlear problems such as acoustic neuroma should be considered if vertigo occurs with hearing loss, roaring, or tinnitus.

PHYSICAL EXAMINATION

Blood pressure. Vestibular disorders can affect the cardiovascular system. Disturbed proprioception causes problems with blood pressure regulation, frequently resulting in hypotensive symptoms and postural tachycardia.

Cranial nerves should be examined to detect any focal, motor, or sensory weakness.

Hearing should be tested and ears examined for active infection or a cholesteatoma—a cyst that looks like cottage cheese and can erode bone even into the inner ear.

Eye movements. In our specialty clinic, we observe eye movements by having patients wear infrared video goggles, which put patients in the dark and make them unable to fixate and control nystagmus. However, with BPPV, the nystagmus is so obvious that no special equipment is needed.

BPPV episodes are short; other vestibular disorders may last for hours or days

The Dix-Hallpike test, also called the Nylen-Barany test, is a maneuver to diagnose BPPV by eliciting nystagmus (FIGURE 1).[3] Results are obvious, and the patient complains of feeling dizzy.

This phenomenon was formerly tested with electrodes attached above each eye, but this system does not detect the rotary component of eye movements well. Most modern diagnostic laboratories use computer-based videonystagmography recording systems, but such sophisticated equipment is not necessary for most cases.

TREATING BPPV

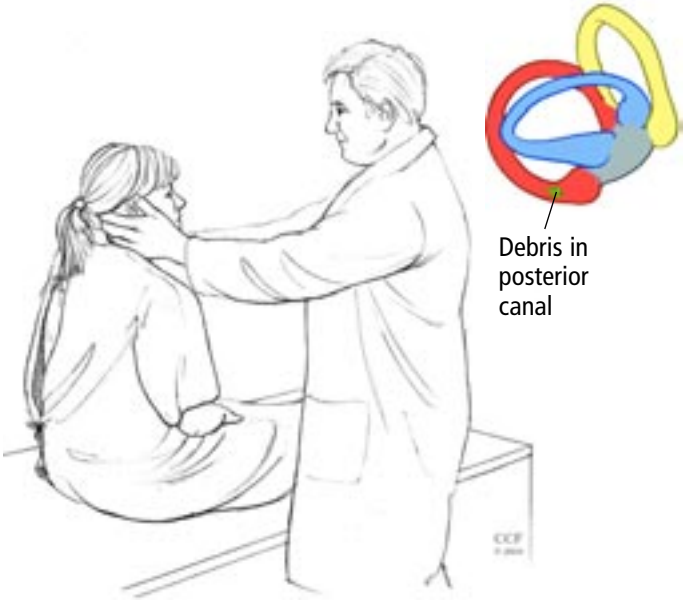
BPPV can be treated in the office in about 10 minutes with canalith repositioning (FIGURE 1).

Before this maneuver became standard practice, physicians told patients that the

A test and a treatment for benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo is thought to be due to debris in the posterior semicircular canal of the inner ear in over 90% of cases. Rarely, other canals can be affected.

The Dix-Hallpike test should elicit nystagmus if the problem is present. Start with the patient seated.



1 To check for a problem in the right ear, rotate the patient's head to the right about 45° to align the right posterior semicircular canal with the sagittal plane of the body, and extend the neck slightly with the chin pointed slightly upward. Tell the patient to keep his or her eyes open.

2 Then, keeping the patient's head turned to the side, lay the patient down so that the head is over the end of the table, with the right ear down and the neck slightly extended. This maneuver should allow any debris to move within the posterior canal. Observe for the typical nystagmus, which is delayed by 5 to 20 seconds and beats on a torsional direction towards the undermost ear, lasting less than 30 seconds and reversing in direction when the patient is brought back up to the sitting position. Repeat the maneuver on the left side.

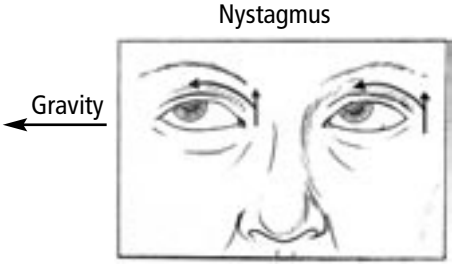


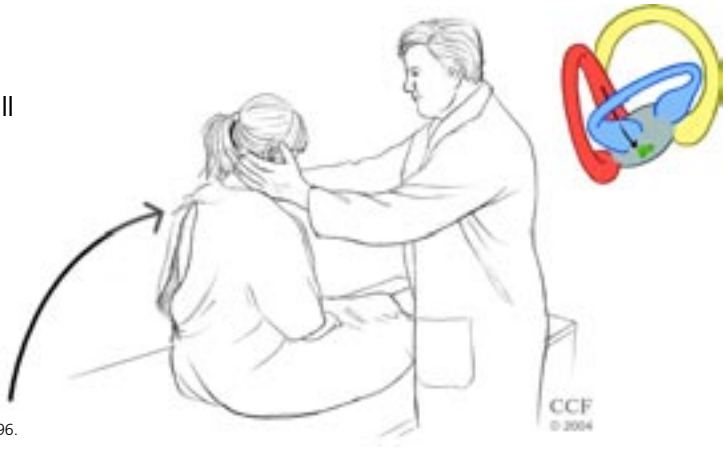
FIGURE 1

3 ***To clear the debris*** from the semicircular canal, start with the Dix-Hallpike maneuver.

4 Wait until the vertigo and nystagmus provoked by this maneuver stop, then rotate the patient's head to the opposite side.

5 Continue by rolling the patient on to his or her side with the face down.

6 Wait about 10 to 15 seconds until nystagmus stops, then sit the patient up with the head still turned to the opposite side.



ADAPTED WITH PERMISSION FROM FURMAN JM, CASS SP. BENIGN PAROXYSMAL POSITIONAL VERTIGO. N ENGL J MED 1999; 341:1590–1596.

problem would resolve in time. However, only 28% of patients have spontaneous resolution of symptoms after 1 month, and 30% still have symptoms after 1 year. In contrast, a single maneuver is effective more than 75% of the time, and two maneuvers are effective up to 97% of the time.

The maneuver starts with the Dix-Hallpike maneuver. Then, the patient's head is rolled away from the affected ear, and the patient continues to roll in the same direction onto the shoulder. After about 10 seconds, the patient is helped to sit up.

The risks are small. There is a 15% chance that the particles will drop into another canal, which can also be treated by further maneuvers, such as rolling the patient 360° towards the healthy ear. Another risk is that the procedure could result in neck strain if the clinician is overly vigorous. This maneuver, however, does not require any rapid or violent movements.

Postprocedure recommendations remain controversial. Patients have traditionally been told to keep their head elevated for 24 to 48 hours and to avoid laying on the affected ear. However, there is little evidence that these recommendations are necessary. We hope to soon undertake a randomized controlled trial using different post-treatment regimens.

Vestibular suppressants should not be used long-term

Vestibular suppressant medications are not recommended for BPPV. They provide little symptomatic relief, and most are sedating and have anticholinergic side effects.

If the patient is not cured with the maneuver, I recommend referral to a vestibular disorders center. There, specialized video equipment can better determine the diagnosis and the efficacy of treatment. Sometimes particles become stuck inside the canals, and the head needs to be tapped or vibrated during the maneuver.

Recurrences of BPPV are common. Patients can be taught to perform the maneuvers at home whenever needed.

In some cases, we perform surgery to plug the canal, a procedure that does not affect hearing.

If disequilibrium persists

The neck is closely connected with the vestibular system and postural control. A diagnostic test, vestibular evoked myogenic potentials, involves playing a loud noise in the ear to disturb the statoliths, which triggers reflexes that change the tone of the sternocleidomastoid muscle. These reflexes can be maladaptive: patients may respond to chronic balance problems by stiffening the neck muscles so often that they develop pain, spasms, and limited range of motion. Neck muscle tone that is chronically asymmetric can contribute to a patient's sense of disequilibrium.

Even after the inner ear problem has improved, patients may continue to have neck problems. This should be considered in patients who have been treated successfully for BPPV (ie, nystagmus has been eliminated) but who continue to complain of feeling off-balance. In such cases, physical therapy can be extremely helpful.

Elderly patients frequently have persistent problems with postural control after BPPV due to such problems as peripheral neuropathy, spinal degeneration, arthritis, spinal stenosis, muscle weakness, and medication effects. Some researchers recommend that most elderly patients with BPPV should be evaluated by a physical therapist. I feel that the large number of elderly patients with the problem makes this impractical, but I do refer patients to physical therapy if balance problems persist despite successful canalith repositioning.

MENIERE SYNDROME

Meniere syndrome has a prevalence of about 6 per 1,000 people and tends to run in families. Its etiology is uncertain, but it is thought to be due to a fluid imbalance in the inner ear.

It is characterized by:

- Low-frequency hearing loss that may come and go
- Episodes of vertigo, lasting for hours to a day, that arise spontaneously and do not depend on position
- Possibly tinnitus between vertigo episodes.

We treat it with a low-sodium diet, a diuretic (triamterene 37.5 mg plus hydrochlorothiazide 25 mg by mouth daily), and lorazepam 1 mg sublingually during an attack. (Lorazepam and triamterene/hydrochlorothiazide are not approved by the US Food and Drug Administration for this indication.)

Two thirds of patients get better over time. For patients with persisting problems, transtympanic therapies are effective.

ACUTE VESTIBULAR SYNDROME

Acute vestibular syndrome usually results from a rapid, unilateral injury to the peripheral vestibular structures. Both ears have a normal static discharge rate: when one side discharges more, it triggers the brain to move the eyes. If one ear is injured and stops discharging, the brain senses head turning and signals the eyes to move away from the injured ear, resulting in nystagmus.

Less commonly, a problem with central vestibular structures is to blame.

Patients with acute vestibular syndrome often present to the emergency room with the acute onset of:

- Dizziness
- Nausea or vomiting
- Hearing loss (if labyrinthitis is present).

Risk factors for central injury

It is important to determine who is at risk for a central problem and therefore needs to be evaluated with imaging studies. Hotson and Baloh[4] established guidelines for which patients need further workup. Risk factors include advanced age, anticoagulation therapy, valvular heart disease, occlusive vascular disease, heavy smoking, and diabetes with high blood pressure.

Postural control may be the best predictor of a central problem: patients with peripheral disease (such as acute viral vestibular neuritis or labyrinthitis) can walk, albeit perhaps in a "drunken" fashion. Patients with central cerebellar or brainstem acute vestibular syndrome, however, have severe balance problems, and are unable to take more than a few steps without falling or crashing into a wall.

Other high-risk indicators for a central problem include focal neurologic findings that suggest stroke, such as double vision or other vision changes, speech disturbances, trouble swallowing, or sensory or motor deficits. Problems with the anterior-inferior or posterior-inferior cerebellar arteries can present with nystagmus, incoordination, facial numbness, crossed sensory loss to pain and temperature, and Horner syndrome (characterized by a constricted pupil, drooping eyelid, lack of sweating, and flushing). Damage to the anterior-inferior artery also typically involves hearing loss, tinnitus, and facial weakness, while posterior-inferior cerebellar artery involvement causes hoarseness and vocal cord paralysis.

Nystagmus differs after peripheral vs central injury

Qualities of nystagmus also provide clues as to whether the event is caused by a peripheral or a central injury.

Froehling et al[5] reported that the typical rotary nystagmus seen on the Dix-Hallpike test is predictive of BPPV, indicating that no further evaluation is necessary.

Characteristically, nystagmus from a peripheral cause beats in one direction, gets a little worse if the patient looks in the direction of the fast phase, and lessens if the patient looks away from that direction (a phenomenon known as Alexander's law). Nystagmus also decreases when the patient fixates on a bright light.

Nystagmus from a central cause does not obey any of those rules: it changes direction, amplitude, and intensity; it violates Alexander's law; and it does not lessen with fixation. In an emergency setting without special equipment, it is sometimes hard to determine the exact nature of the nystagmus, but nystagmus from a central cause appears more chaotic.

Symptoms of acute vestibular syndrome: dizziness, nausea, vomiting, sometimes hearing loss

MIGRAINE-ASSOCIATED VERTIGO

Migraine-associated vertigo should be suspected in a patient who does not fit any of the other typical patterns of vertigo.

About 30% of the US population is affected by migraine disorder. Attacks are not always associated with headache: patients may have a lot of headaches during adolescence, then have attacks of vertigo in middle adulthood instead. Attacks tend to be brief, repetitive, and not associated with hearing loss, tinnitus, or position.

Retrocochlear workup, magnetic resonance imaging with contrast, and vestibular testing are negative.

Patients tend to respond well to diet, lifestyle modifications, and medications that

are recommended for migraine.

In a young, healthy patient without hearing loss and with a history of migraines, I recommend a trial of prophylactic migraine medication to see if symptoms resolve before recommending additional testing.

ASSOCIATED CARDIOVASCULAR PROBLEMS

Vestibular input helps regulate autonomic and postural blood pressure. Either a unilateral or a bilateral vestibular loss may cause postural orthostatic tachycardia syndrome. Patients with persistent light-headedness and disequilibrium after a vestibular insult should be referred for a cardiology consultation.

VESTIBULAR COMPENSATION

Patients with vestibular loss can learn to compensate. Some people are found during testing to have a unilateral vestibular loss, which may have occurred in childhood, and which has been completely compensated.

I strongly recommend not overmedicating patients after an acute vestibular insult. A vestibular suppressant, such as a benzodiazepine, may be needed for a few days, but prolonged use inhibits compensatory mechanisms from developing. Patients should be encouraged to start walking, holding on to someone if necessary. As they become comfortable, they should challenge themselves as they walk by turning their head and then focusing on their finger as they turn their head. These exercises will help train the good ear to take over complete vestibular function.

Patients with bilateral vestibular dysfunction, such as after aminoglycoside toxicity, may not experience vertigo because there is no mismatch between the two sides of the head. They do, however, feel off-balance, and things appear to bob in front of them as they walk or turn their head. They may also have a high-frequency sensorineural hearing loss.

Such patients should not be given vestibular suppressants but should be referred for vestibular rehabilitation, where their needs can be assessed and treated. Exercise programs can help them to recruit their cervico-ocular reflexes to stabilize their eye movements.

MOTION SICKNESS

The tendency to develop motion sickness seems to be genetically mediated. It is more common in patients with migraine disorder. People who get motion sickness easily are extremely sensitive to the mismatch between what they see and their sensation of movement, which triggers nausea centers.

Before a ride, motion sickness can be avoided with a vestibular suppressant, such as a scopolamine patch. After motion sickness starts, antihistamines can be helpful, as can ginger, either crystallized or in tea or cookies.

Some patients have no balance problems while on a cruise, but upon return, feel "seasick" on land, a phenomenon known as *mal de débarquement*. This curious disorder may persist for months, and additional seagoing may provoke relapse.

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ADDRESS: *Judith White, MD, PhD, Department of Otolaryngology and Communicative Disorders, A71, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH 44195; e-mail whitej3@ccf.org.*