REVIEW ARTICLE

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Episodic Positional Dizziness

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ABSTRACT

PURPOSE OF REVIEW: This article provides a summary of the evaluation and treatment of patients presenting with episodic positional dizziness.

RECENT FINDINGS: Positional components are nearly ubiquitous among diagnoses of dizziness, so it can be challenging to classify patients with episodic positional dizziness simply based on the history of present illness. Overreliance on the presence of a report of positional components has likely resulted in misapplication or misinterpretation of positional testing and negative experiences with maneuvers to treat positional dizziness. The prototypical episodic positional dizziness disorder is benign paroxysmal positional vertigo (BPPV). BPPV is caused by free-floating particles in a semicircular canal that move in response to gravity. The diagnosis is made by identifying the characteristic patterns of nystagmus on the Dix-Hallpike test. Particle repositioning for BPPV is supported by randomized controlled trials, meta-analyses, and practice guidelines. Other disorders that can present with episodic positional dizziness are migraine dizziness, central lesions, and light cupula syndrome.

SUMMARY: Episodic positional dizziness is a common presentation of dizziness. Neurologists should prioritize identifying and treating BPPV; doing so provides an important opportunity to deliver effective and efficient care. Providers should also recognize that positional components are common in most causes of dizziness and, therefore, should not over-rely on this part of the history of presentation when considering the diagnosis and management plan.

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INTRODUCTION

Episodic positional dizziness is a label used to describe a category of dizziness presentations characterized by recurrent events and prominent positional components or triggers. The dizziness can be a variety of types, including spinning, tilting, and other sensations of movement, lightheadedness, or imbalance. The positional components are also broad, including standing up, turning the head, tilting the head back, or rolling over in bed. Other categories of dizziness presentations include the acute vestibular syndrome, episodic spontaneous dizziness, and chronic constant dizziness. Episodic positional dizziness has a variety of causes. A priority in these presentations is identifying people who may have benign paroxysmal positional vertigo (BPPV) and, therefore, benefit from a canalith repositioning maneuver,

which is evidence-based and guideline supported.[1-4] Despite this, providers continue to have misconceptions about BPPV and underuse or misuse the canalith repositioning maneuver.[5-7] This article provides a summary of the evaluation and management of episodic positional dizziness.

EPIDEMIOLOGY

Approximately 15% of adults in the United States report problems with dizziness in the past 12 months inclusive of symptoms such as imbalance/unsteadiness, lightheadedness, feeling as if passing out/fainting, spinning/vertigo, floating/tilting sensation, and blurring of vision when moving the head.[8] The lifetime prevalence of BPPV has been estimated to be 10% by using data from a random–digit dial national health survey in Germany.[9] Self-reported positional components are extremely common among all patients with dizziness in general, which can make it challenging to classify a case as episodic positional dizziness as opposed to episodic spontaneous dizziness or even the acute vestibular syndrome. More than 90% of individuals who report problems with dizziness in the past 12 months describe one or more of the following triggers: turning the head side to side, looking up or down, rolling over in bed, getting up after sitting or lying down, or standing or being on their feet for a long time (data from the 2008 National Health Interview Survey).[10] Approximately 33% of all patients with problems with dizziness in the past 12 months report typical BPPV triggers of turning the head side to side, looking up or down, and rolling over in bed. The very high frequency of positional components makes it a challenge to rely on the patient's report of positional components to make a diagnosis or even classify the category of dizziness presentation.

TAKING THE HISTORY

The history of present illness in patients with episodic positional dizziness is important. However, the history of the dizziness should not be overemphasized when making management decisions. It has been demonstrated that frontline doctors generally overemphasize the history of present illness in patients with dizziness, and this may be a key factor in negative experiences with treating patients with dizziness. Frontline providers specifically report negative experiences in using the Dix-Hallpike test and canalith repositioning maneuvers.[6] Providers should not use types of dizziness as rigid branch points in diagnostic considerations because verbatim descriptions of dizziness are generally vague, most patients select more than one type of dizziness, the selection of a primary type of dizziness is typically not reliable, and types of dizziness are actually poor discriminators among causes of dizziness.[8,11] Patients are generally more reliable in their report of timing and triggers of symptoms.[11] However, positional triggers are nearly ubiquitous among all causes of dizziness. Experience in clinical practice has found that the history of dizziness symptoms can be nearly identical in patients who present acutely with either BPPV or an acute unilateral vestibulopathy (eg, vestibular neuritis). Patients with either diagnosis often report the onset of symptoms after getting out of bed, severe symptoms with subsequent movements, and improvement when still, with milder constant dizziness.

The evaluation of patients with dizziness generally starts by asking them to use their own words to describe the symptom; though, as previously noted, the neurologist should be careful not to overemphasize the description. Next, it is

KEY POINT

- **Patients with any cause of dizziness typically report positional components. As a result, providers should be cautious in relying on the self-report of positional components to make a diagnosis of benign paroxysmal positional vertigo (BPPV).**

important to obtain details about the timing and triggers of symptoms. Defining how long ago the dizziness started helps in considering whether this is an acute, subacute, or chronic disorder. The frequency and duration of the episodes should be determined. In considering the self-report of timing and triggers, providers also need to be careful to not overemphasize these components. For example, BPPV is considered a brief dizziness, typically lasting less than 1 minute. However, many patients with BPPV report more prolonged symptoms that can even be constant,[4] although usually the duration of the most intense symptoms is still brief. Therefore, even patients who report prolonged symptoms should be assessed for BPPV unless another clear cause is identified before positional testing.

Lastly, providers should also gather information about the course of the symptoms over time in terms of whether they have improved, worsened, or stabilized. It can also be helpful to ask about any patterns with the symptoms, such as whether they tend to occur at certain times of the day or in association with certain foods, stress, or sleep.

EXAMINATION

For patient evaluations in typical office or emergency department settings, the most important diagnostic information is usually obtained on the physical examination. Emphasizing the examination is a strategy that can prevent focusing on the often unreliable or overlapping components of the history of present illness.

The examination should start with a focused general medical and general neurologic assessment. The neuro-otologic components can be incorporated into the general neurologic examination or grouped at the end. The neuro-otologic examination consists of the ocular motor assessment, coordination tasks, balance assessment, and positional testing. Providers may find it useful to incorporate these elements into routine examinations so they can establish internal thresholds of normal versus abnormal findings.

Ocular Motor Examination

For some patients with episodic positional dizziness, the key examination findings are obtained on the bedside ocular motor assessment. For example, some patients who report episodic positional dizziness can have mild cerebellar disorders that are rapidly identified on the ocular motor assessment. These findings include spontaneous downbeat nystagmus, bidirectional gaze-evoked nystagmus, and pathologically impaired smooth pursuit. The ocular motor examination starts with observing the eyes in primary gaze for 5 to 10 seconds and specifically looking for any spontaneous movements such as nystagmus or saccadic intrusions. Nystagmus has alternating slow and fast components that give the appearance that the eyes are beating in the direction of the fast phase. Saccadic intrusions are involuntary saccades that intrude upon gaze. Square-wave jerks are the most common type of saccadic intrusion. Square-wave jerks consist of an involuntary saccade to one side, a brief intersaccade delay, and then a saccade back, giving the appearance of shifting, as opposed to the rhythmic beating of nystagmus. Square-wave jerks are nonspecific and generally not pathologic when an isolated finding; they are particularly common when people are anxious. Other types of saccadic intrusions are ocular flutter and opsoclonus, which are pathologic and commonly occur in people with autoimmune cerebellitis and Friedreich ataxia.

After observing for a spontaneous movement, the clinician should then have the patient follow the examiner's finger to lateral gaze to each side and then up and down. In each position, the eyes are held at approximately 30 degrees off center and observed for nystagmus for about 5 to 10 seconds. The clinician should take advantage of the transitions of gaze testing to also assess smooth pursuit. Smooth pursuit is normal when the patient can track the finger back and forth very smoothly. Impaired smooth pursuit is considered a nonlocalizing finding. However, when impaired smooth pursuit is identified in patients who are cognitively intact and without other large motor deficits (eg, hemiplegia), it is a strong indicator of cerebellar dysfunction. When smooth pursuit is impaired, the individual must use the saccade system to move the eyes to the side. Therefore, impaired smooth pursuit is typically saccadic pursuit. The saccade system can be assessed by having a patient look back and forth from one finger to the next and observing for the speed of the movement and the accuracy. Hypometric saccades are generally not a pathologic finding if they are isolated. Consistent hypermetric saccades are suggestive of cerebellar dysfunction. This ocular motor assessment generally takes less than 1 minute to perform and is important to perform before proceeding to positional testing. Patients with vestibular neuritis may have a history suggestive of episodic positional dizziness but, on close inspection, will have unidirectional (ie, does not change direction) horizontal nystagmus in primary gaze and lateral gaze. Many providers do not appreciate the physiology of BPPV is such that nystagmus is present only when the particles are in motion, and the particles are typically at rest when the head is still. When shown videos of a typical acute unilateral vestibulopathy pattern of nystagmus in a patient with vestibular neuritis, one study found that primary providers nearly always erroneously selected BPPV as the cause.[6] In the same study, these providers also typically reported negative experiences using the Dix-Hallpike test and canalith repositioning maneuvers. It is likely that their negative experiences in using the Dix-Hallpike test and Epley maneuver are based on an incorrect selection of patients and interpretation of the results.

Head Impulse Test

The head impulse test is another component of the ocular motor assessment. It is a bedside measure of the vestibular ocular reflex (described further in the section Evaluation and Management of the Patient With Episodic Positional Dizziness) and an important part of the evaluation of patients with the acute vestibular syndrome. This is generally less applicable in patients who have episodic positional dizziness such as BPPV.

Coordination and Balance Assessment

General gait and coordination assessments are used to identify cerebellar dysfunction, which can be a key indicator of a central disorder.

Positional Testing

Positional testing includes the Dix-Hallpike test and supine positional testing (FIGURE 3-1[12] and FIGURE 3-2). Different strategies can be used for the approach to positional testing. A common strategy is to start with the Dix-Hallpike test to one side and then, if negative, performing the test on the other side. The reason for the testing positions of the Dix-Hallpike test is that particles can be evaluated one side at a time based on the plane of the canal. The Dix-Hallpike test primarily

KEY POINTS

- Patients with dizziness are often not reliable in their self-report of the type of dizziness. As a result, BPPV should be considered a diagnostic possibility even in patients who do not report vertigo.
- The ocular motor examination starts with observing the eyes in primary gaze for 5 to 10 seconds and specifically looking for any spontaneous movements such as nystagmus or saccadic intrusions.
- When impaired smooth pursuit is identified in patients who are cognitively intact and without other large motor deficits (eg, hemiplegia), it is a strong indicator of cerebellar dysfunction.
- Eye movement testing, including pursuit tracking, gaze testing, and saccadic eye movement observation, takes very little time but can be the key factor in identifying a cerebellar or vestibular disorder.
- The Dix-Hallpike test is designed to identify posterior canal BPPV but can also identify the horizontal and anterior canal variants and central causes of dizziness.

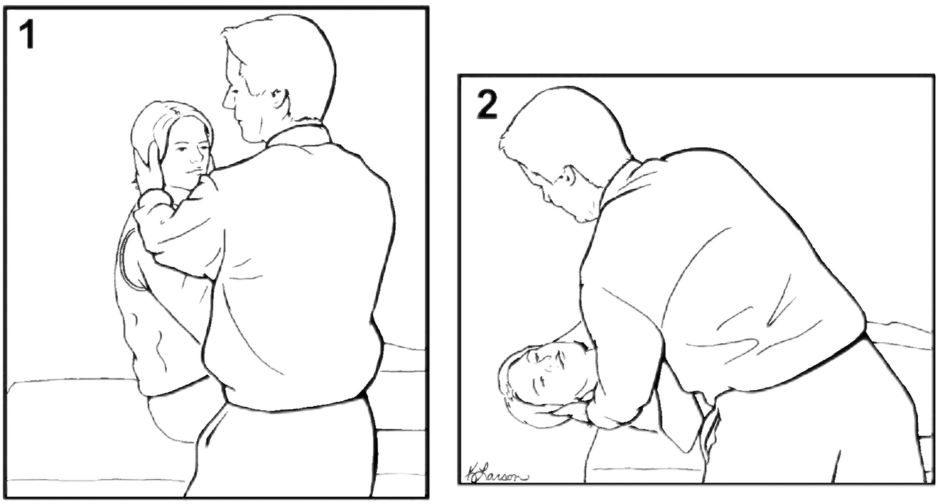

FIGURE 3-1
Dix-Hallpike test for the diagnosis of right posterior canal benign paroxysmal positional vertigo (BPPV). The patient's head is turned 45 degrees to the side to be tested (step 1) and then laid back quickly (step 2). If BPPV is present, nystagmus ensues usually within seconds.
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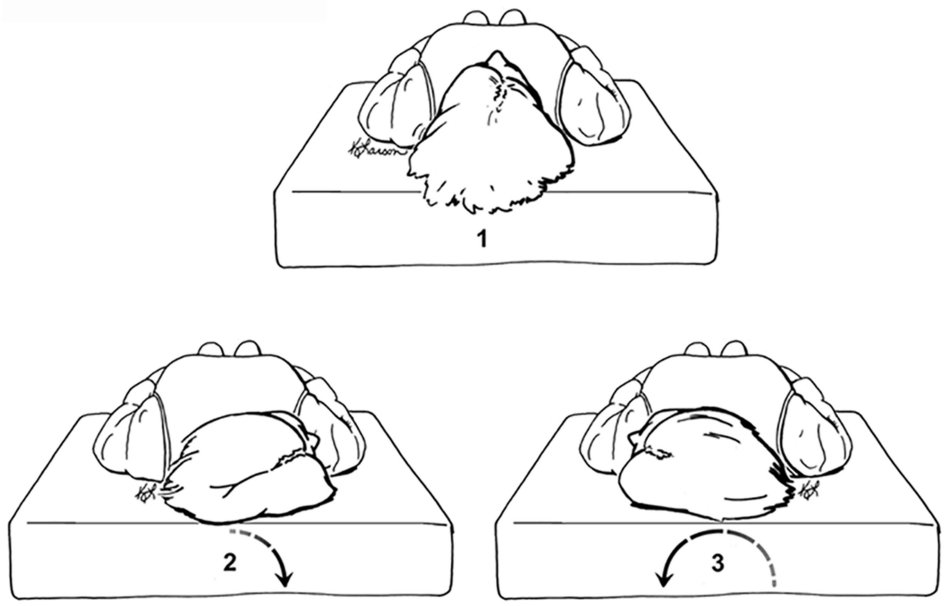

FIGURE 3-2
The supine roll test to detect horizontal canal benign paroxysmal positional vertigo (BPPV). The patient may be moved from sitting to a straight supine position (step 1). The head is turned to the right side (step 2) with observation of nystagmus and then turned back to face up (step 1). Then the head is turned to the left side (step 3). The side with the most prominent nystagmus is understood to be the affected horizontal semicircular canal. The direction of nystagmus in each position determines whether the horizontal canal BPPV is of the geotropic or apogeotropic type.
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assesses for particles in the posterior canal, although it can also identify the horizontal and anterior canal variants. The outline of the patient's ear on one side can be used as a model of the general anatomy of the posterior canal to teach patients and trainees about BPPV. When the outline of the ear is used as a model, the particles can be described as accumulating in the lobule area of the ear. It can then be explained that the goal in particle repositioning is to move the particles up and around the helix to the superior portion of the ear and then down and out (FIGURE 3-3). To perform the Dix-Hallpike test on the left side, the patient's head is turned approximately 45 degrees to the left side. This puts the plane of the posterior canal in the direction in which the patient is going to move. The right posterior canal is out of the plane such that particles in the right posterior canal will not generally move during the left-side Dix-Hallpike test. The patient is brought back down to a head-hanging position either over the edge of the bed or over some pillows placed behind the upper back. The head should be tilted back approximately 20 degrees so that the particles move enough in the posterior canal to trigger the symptoms and nystagmus. In addition, at least 20 degrees of extension is needed for the particles to move far enough to the superior portion of the posterior canal for the eventual treatment to be successful. Once the patient is in the head-hanging position, the eyes are observed for approximately 10 to 15 seconds. Because many patients want to close their eyes, the examiner should be prepared to open them with the explanation that any eye movements need to be observed by the neurologist. If the test to the left is negative, the patient should be asked to sit back up and then turn their head to the right side approximately 45 degrees; the patient should then lie back down with the head tilted back approximately 20 degrees. The test is considered positive when nystagmus is triggered by the test and the nystagmus is transient in duration and accompanied by symptoms. Some patients report symptoms, but no nystagmus is observed in which case BPPV can still be suspected but not confirmed. The nystagmus pattern of posterior canal BPPV is upbeat torsional.

Supine positional testing is a test more designed to specifically move particles in the horizontal canal. However, the positional nystagmus of horizontal canal BPPV will generally be elicited even on the Dix-Hallpike test. If a horizontal nystagmus is triggered by the Dix-Hallpike test, then posterior canal BPPV is excluded. To perform supine positional testing, the patient starts in the supine position (FIGURE 3-2). Next, the head is turned to one side and held there for 10 to 15 seconds. The head is then brought back to midline and turned to the opposite side and held for approximately 10 to 15 seconds.

COMMON CAUSES OF EPISODIC POSITIONAL DIZZINESS

The most common vestibular causes of episodic positional dizziness are BPPV, migraine dizziness, and structural disorders that affect central vestibular pathways.

Benign Paroxysmal Positional Vertigo

BPPV is the most common peripheral vestibular disorder and the prototypical episodic positional dizziness disorder. *Benign* is not synonymous with *trivial* because 85% of patients with BPPV report missing work, stop driving, or present for a medical evaluation.[9] Patients with BPPV generally report a spinning type of dizziness, but this is not required. The symptoms are generally brief and triggered by lying down or rolling over in bed or looking up to reach for something.

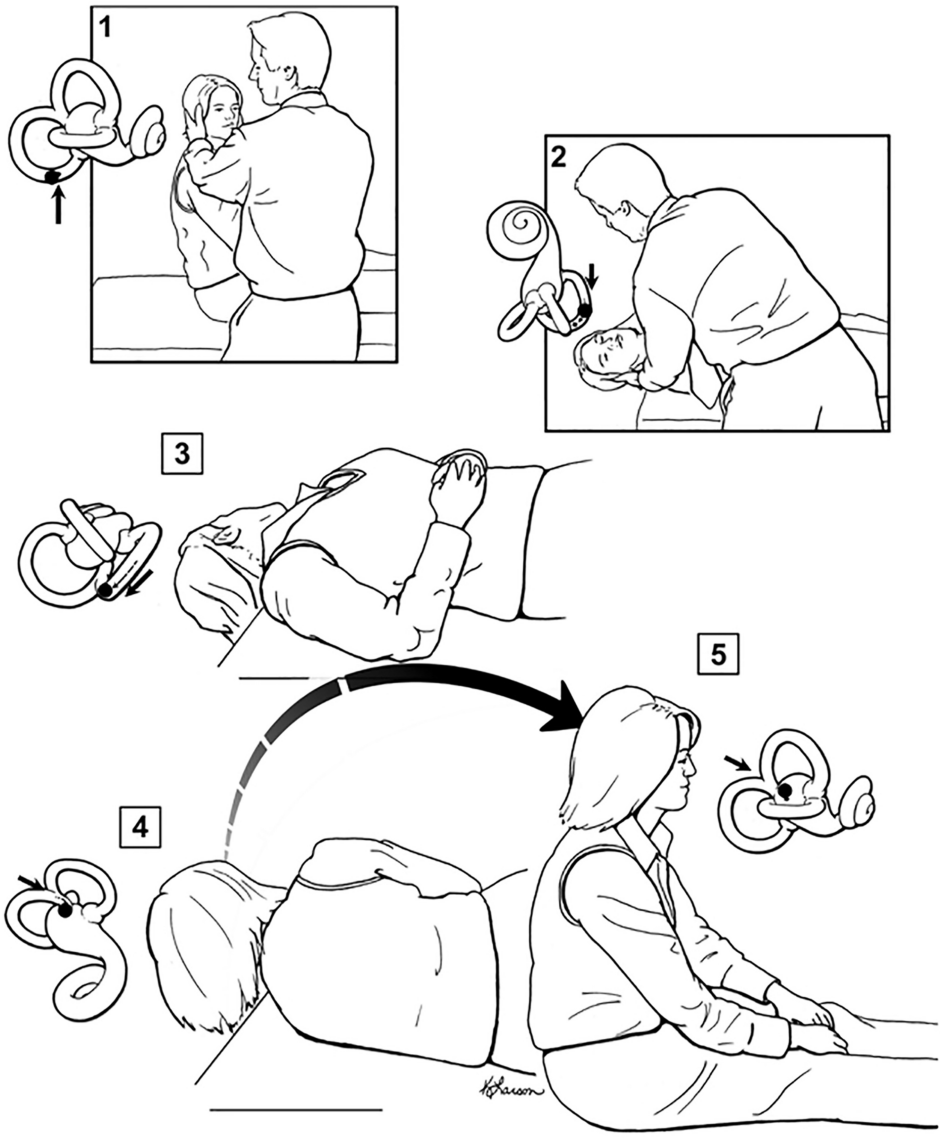

FIGURE 3-3

Canalith repositioning maneuver (ie, the Epley maneuver) for right-sided benign paroxysmal positional vertigo. Steps 1 and 2 are identical to the Dix-Hallpike test. The patient is held in the right head-hanging position (step 2) for 20 to 30 seconds and then in step 3 the head is turned 90 degrees toward the unaffected side. Step 3 is held for 20 to 30 seconds before turning the head another 90 degrees (step 4) so the head is nearly in the face-down position. Step 4 is held for 20 to 30 seconds, and then the patient is brought to the sitting up position (step 5). The movement of the otolith material within the labyrinth is depicted with each step, showing how otoliths are moved from the semicircular canal to the vestibule. Although it is advisable for the examiner to guide the patient through these steps, it is the patient's head position that is the key to a successful treatment.

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BPPV is caused by otoconia, calcium carbonate particles that have detached from the utricular macula and enter a semicircular canal on one side (or occasionally both). The reason the particles detach is not clear, but an animal study found that as rats age the otoconia in both maculae become pitted, fissured, and fragmented with weakened or broken linking filaments.[13] It is likely that this degeneration makes the otoconia susceptible to detaching or at least eroding. It is also believed that the otoconia can detach after head trauma or an acute unilateral vestibulopathy from either neuritis or ischemia. The particles are more dense than the endolymph and therefore will move in the canal in response to gravity. When enough particles in the canal move at a sufficient speed, fluid drag is created, resulting in aberrant endolymph flow that deflects the cupula and then modulates the activity of the vestibular afferents of the affected canal. This is referred to as *canalithiasis*. It is also possible for the particles to attach to the cupula of a semicircular canal and render it sensitive to gravity, which is referred to as *cupulolithiasis*. The modulation of the vestibular afferents results in a brief burst of nystagmus and the associated symptoms. The positional nystagmus can "fatigue" on repeated testing likely because the particles disperse in the canal. **CASE 3-1** describes a case of BPPV of the posterior canal, and **CASE 3-2** describes a case of acute unilateral vestibulopathy.

Some patients with BPPV report a constant milder dizziness before and even for a time after treatment for unclear reasons.[4] However, the severe recurrent positional attacks of dizziness occur only when the particles are in the semicircular canal. Particles can enter any of the three canals: the anterior, the horizontal, and the posterior (**TABLE 3-1**). BPPV of the posterior canal is by far the most common type reported in large case series.[2] However, these case series are derived from outpatient specialty clinics comprising patients who have had symptoms for days to weeks or even longer. No particular reason exists for the particles to be more likely to enter the posterior canal than other canals; however, the reasons are clear why particles are more likely to be stuck in the posterior canal rather than the anterior or horizontal canal. If the particles enter the anterior or horizontal canal, natural head positions such as sitting up, lying down, or rolling over are generally adequate for the particles to fall out of the canal. However, natural head positions are much less likely to guide particles out of the posterior canal, which is why the Dix-Hallpike test uses deep extension to guide the particles to the superior aspect of the posterior canal and then around and out.

The gold standard test for benign positional vertigo is the Dix-Hallpike test.[2,4] A positive finding is a triggered and transient nystagmus and associated symptoms. The key feature is that the nystagmus is not present when the patient is sitting still but is then triggered and transient after the patient is placed in the head-hanging position. The pattern of nystagmus in the posterior canal is upbeating torsional in the head-hanging position as the particles move into the superior portion of the posterior canal, creating drag that deflects the cupula (**TABLE 3-1**). If the patient is then brought back up to the sitting position without being treated for the BPPV, generally a burst of downbeat-torsional nystagmus occurs as the particles move in the opposite direction. The nystagmus usually has a latency to onset of 1 or a few seconds with the Dix-Hallpike test, a peak velocity of 30 to 50 degrees per second, and a duration of approximately 10 to 30 seconds.[14]

KEY POINTS

- Some patients with BPPV report a constant milder dizziness before and even for a time after treatment for unclear reasons.
- The gold standard test for BPPV is the Dix-Hallpike test. A positive finding is a triggered and transient nystagmus.

Variants of Benign Paroxysmal Positional Vertigo

The main variants of BPPV are the horizontal canal variant and the anterior canal variant. The pattern of nystagmus in horizontal canal BPPV is direction-changing horizontal positional nystagmus, which can be either geotropic (a label used to describe that the fast phase of nystagmus beats toward the ground in each position: the head positioned to right while supine triggers nystagmus to right, and the head to left while supine triggers nystagmus to left) or apogeotropic (a label used to describe that the fast phase of nystagmus beats away from the ground in each position: the head positioned to the right while supine triggers nystagmus to left, and the head to left while supine triggers nystagmus to right) (**TABLE 3-1** and **FIGURE 3-4**[15]). Note that direction-changing positionally triggered nystagmus is different from direction-changing gaze-evoked nystagmus, which is a central sign.

CASE 3-1

A 65-year-old woman presented for dizziness symptoms that started approximately 3 weeks before. The dizziness was described as an odd sensation, and, on specific questioning, she reported a feeling of spinning and lightheadedness. She also had a mild constant sense of imbalance. The most significant symptoms occurred primarily after rolling over in bed during the night, getting up from bed in the morning, and looking up to reach for something during the day. Her past medical history was unremarkable.

General medical and neurologic examinations were normal. No spontaneous or gaze-evoked nystagmus was present. Smooth pursuit was intact. No coordination abnormalities were noted. She walked with a narrow base with a good heel strike and stride length.

The Dix-Hallpike test performed with her head turned to the right side did not trigger symptoms or nystagmus. The Dix-Hallpike test to the left side, however, triggered a robust burst of upbeat-torsional nystagmus. The patient was substantially symptomatic and attempted to close her eyes; therefore, the examiner needed to open the patient's eyes to be able to see the nystagmus and assess its duration. The nystagmus slowly dissipated and was gone within 15 seconds.

COMMENT

This is a classic presentation of benign paroxysmal positional vertigo (BPPV) of the posterior canal, which is the most common variant. Posterior canal BPPV can last for weeks or months because the particles are trapped in the inferior portion of the canal. The Dix-Hallpike test is negative on the unaffected side because the affected canal is out of the plane of movement such that the particles do not move during that test. However, when that affected canal is lined up with the plane in which the head is moving in the left Dix-Hallpike test, a robust burst of upbeat-torsional nystagmus occurs in a pattern that is consistent with stimulation of the posterior canal vestibular afferents. When performing the Dix-Hallpike test, the examiner should be prepared to open the patient's eyes if necessary so that nystagmus can be identified. The provider can then complete the Epley maneuver to guide the particles around and out of the canal.

CASE 3-2

A 65-year-old woman presented to the emergency department with a new onset of dizziness symptoms described as a spinning and a woozy-type sensation in addition to a feeling of imbalance. She reported feeling fine the night before. While getting out of bed in the morning, she felt a rapid onset of severe spinning that was soon followed by nausea and vomiting. She collapsed back into bed and felt better after approximately 10 minutes. However, with any subsequent attempts to move and get up, the symptoms substantially worsened. She denied any other focal symptoms. Her past medical history was unremarkable.

She was examined in the emergency department. The general medical and neurologic assessments were unremarkable. On the ocular motor assessment, she was noted to have a spontaneous right-beating nystagmus. The velocity of nystagmus increased when she looked to the right and decreased, but did not change direction, when she looked to the left. On the head impulse test, an abnormal corrective saccade was present after quick movements to the left side but not to the right side. No coordination abnormality was noted. The patient had mild unsteadiness when walking with difficulty taking 10 steps in tandem. On positional testing, right-beating nystagmus was present on the right Dix-Hallpike test. On the left Dix-Hallpike test, the right-beating nystagmus was also present. The same nystagmus was noted on supine positional testing to the right side and the left sides.

COMMENT

This is a case of an acute unilateral vestibulopathy, which typically arises from vestibular neuritis. The case illustrates just how closely the history of present illness can be in patients with an acute unilateral vestibulopathy and patients with benign paroxysmal positional vertigo (BPPV). On the first day of symptoms, and particularly among patients who present acutely to the emergency department, the history of present illness in these two disorders can substantially overlap. In this case and in CASE 3-1, the patients reported a sudden onset of symptoms after getting out of bed. Patients often report initial symptoms that last longer than would typically be expected for BPPV but also describe a substantial improvement from being still that is more so than expected for acute unilateral vestibulopathy. What discriminates acute unilateral vestibulopathy from BPPV in this circumstance is the pattern of nystagmus. Patients with acute unilateral vestibulopathy have spontaneous unidirectional horizontal nystagmus. However, typically patients with BPPV have only the typical triggered and transient nystagmus observed with positional testing. In this current patient, no indication for attempted particle repositioning was present, and, in fact, this would just likely exacerbate symptoms. The focus is instead on symptomatic management and vestibular rehabilitation for acute unilateral vestibulopathy. A trial of corticosteroids can also be considered, although meta-analyses of clinical trials concluded that currently the evidence is insufficient to support the use of corticosteroids in acute unilateral vestibulopathy.

With the geotropic pattern, the patient has left-beating nystagmus after turning the head to the left and right-beating nystagmus after turning the head to the right. The geotropic pattern occurs when the particles are in the posterior aspect of the horizontal canal; therefore, a head turn to the affected side while supine results in particle movement toward the cupula, and a head turn away from the affected side while supine results in particle movement away from the cupula. The velocity of the nystagmus in the horizontal canal variant can be very high (greater than 100 degrees per second).[14] The duration of the nystagmus is also usually longer than that of the posterior canal, typically approximately from 30 to 60 seconds.[14] In the apogeotropic form, the particles are in the anterior segment of the horizontal canal on or near the cupula. With the apogeotropic variant, the patient has left-beating nystagmus after turning the head to the right while supine and right-beating nystagmus after turning the head to the left while supine. The velocity of the nystagmus in the apogeotropic variant is usually in the moderate range of 10 to 40 degrees per second, whereas the duration may last longer than 1 minute.[14] In the horizontal canal variant, the velocity of the nystagmus is often greater with movement to one side compared with the other and corresponds with the particles moving toward the cupula resulting in an excitation deflection. In the geotropic variant, the affected side is typically the one that generates the higher-velocity nystagmus. However, the affected side in the apogeotropic variant is typically the side that generates the lower-velocity nystagmus (CASE 3-3).

Anterior canal BPPV is the least common variant, likely because the particles should easily fall out of the canal with natural head positions. The nystagmus pattern with anterior canal BPPV is a burst and taper of downbeat-torsional nystagmus. The Dix-Hallpike test also evaluates for the anterior canal variant.

TABLE 3-1 **Patterns of Positional Nystagmus**

Type of episodic positional dizziness	Characteristic nystagmus patterns
Posterior canal benign paroxysmal positional vertigo (BPPV)	Burst and taper of upbeat-torsional nystagmus on the Dix-Hallpike test
Horizontal canal BPPV, geotropic	Positionally triggered direction-changing horizontal nystagmus beating toward the ground triggered by the Dix-Hallpike test or supine positional testing; the nystagmus is right beating on head turns to the right and then left beating on head turns to the left
Horizontal canal BPPV, apogeotropic	Positionally triggered direction-changing horizontal nystagmus beating away from the ground triggered by the Dix-Hallpike test or supine positional testing; the nystagmus is left beating on head turns to the right and then right beating on head turns to the left
Anterior canal BPPV	Burst and taper of downbeat-torsional nystagmus on the Dix-Hallpike test
Central	Persistent positional downbeating nystagmus, apogeotropic unidirectional positional nystagmus, typically associated with other ocular motor abnormalities, speech, or other motor problems
Migraine	A variety of patterns of nystagmus usually less in velocity than BPPV and seen primarily with positional testing in the dark

Central Positional Dizziness

Central nervous system disorders can also cause positional nystagmus.[16] Therefore, clinicians should not think of the Dix-Hallpike test as a test only for peripheral disorders. No studies have assessed the prevalence of central positional nystagmus; however, case series from specialty clinics have reported that approximately 10% of cases with positional nystagmus are central in origin.[16] A 2017 systematic review identified 28 studies that focused on central positional nystagmus.[16] The studies were generally small, ranging anywhere from single cases to a series of 14 patients. This review in total identified only 82 participants. In most cases, the nystagmus was purely vertical (nearly all downbeating), purely horizontal, purely torsional, or a combination involving a downbeating component. The patients who had central horizontal positional nystagmus with supine positional testing differed from the typical pattern of horizontal canal BBPV because the nystagmus in the central cases was usually only present on one side, which was typically apogeotropic. The duration of the positional nystagmus could be brief or persistent in cases with central causes. About half of the patients with central positional nystagmus had at least one other central symptom or abnormality such as weakness, ataxia, or speech change. A large majority (85%) of those with reported ocular motor assessments had gaze-evoked nystagmus, abnormal saccades, or impaired smooth pursuit. In 75% of the central positional nystagmus cases, the lesion was in the cerebellum, 9% had isolated brainstem lesions and 15% had lesions involving the fourth ventricle.

In a 2019 case series from a single center of 27 patients with central positional nystagmus, the most common pattern of positional nystagmus was downbeat nystagmus in 69%, followed by apogeotropic horizontal in 42%, geotropic

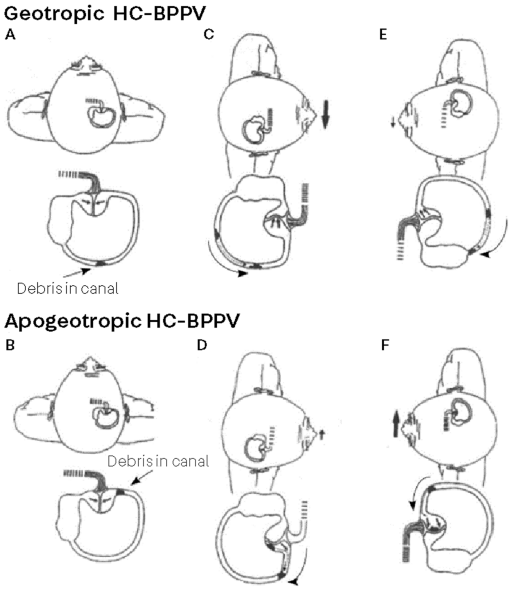

FIGURE 3-4

Head and horizontal canal position in the geotropic and apogeotropic variants of horizontal canal benign paroxysmal positional vertigo (HC-BPPV) affecting the right side. The curved arrows along the canal show the direction of otolithic debris movement after a head turn. *A* and *B* demonstrate debris position within the canal when a patient with right HC-BPPV is lying supine. *C* and *D* demonstrate debris movement, the different effects on the cupula, and the direction of the fast phase of horizontal nystagmus when the patient turns to the right side. The size of the arrow in front of the subject's nose also indicates nystagmus intensity (amplitude). The density of lines in the afferent vestibular nerve indicates the firing rate, which accounts for the stronger nystagmus when debris moves toward the cupula (ampullopetal) as opposed to away from it (ampullofugal). *E* and *F* demonstrate debris movement, the effect on the cupula, and the direction of the fast phase of horizontal nystagmus when the patient turns the head to the left side.

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KEY POINTS

- ***Direction-changing positional nystagmus*** **refers to nystagmus that changes direction with changes in head position and should not be confused with gaze-evoked nystagmus in which nystagmus changes direction with changes in the direction of gaze.**
- **Clinicians should not think of the Dix-Hallpike test as a test only for peripheral disorders; it is also a test for central positional dizziness.**
- **The most common patterns of central positional nystagmus are downbeat, apogeotropic horizontal, geotropic horizontal, and multiplanar.**

horizontal in 8%, and multiplanar in 23%.[17] In this series, 85% of the patients had additional neurologic symptoms, and 70% had other ocular motor abnormalities. The central lesions were most frequently identified in the cerebellar tonsil, nodulus, uvula, or other midline vermis regions.

Degenerative cerebellar ataxia (eg, spinocerebellar type 6) can also have prominent episodic positional dizziness that requires patients to avoid provocative positions by sleeping propped up and avoiding lying flat.[18] The nystagmus pattern can comprise a persistent positional downbeat. These patients inevitably have other central ocular motor findings and ataxia (CASE 3-4).

CASE 3-3

A 65-year-old man presented to the emergency department with new-onset dizziness. The dizziness was described as a spinning-type sensation in addition to lightheadedness, wooziness, and mild imbalance. The patient stated that, when he woke up in the morning of presentation and went to get out of bed, he experienced a substantial spinning sensation that seemed to last 5 to 10 minutes. He felt much better when being still, but with any subsequent movement the symptoms would recur. His past medical history was unremarkable.

On examination, no general medical or neurologic abnormalities were present. On the ocular motor assessment, no spontaneous or gaze-evoked nystagmus was observed. On the Dix-Hallpike test to the right, a burst of high-velocity right-beating nystagmus and associated symptoms were present. The nystagmus slowly decreased in velocity but persisted for longer than 30 seconds. The patient's head was returned to a flat supine position and then turned to the left side, which triggered a significant but lower-velocity left-beating nystagmus lasting approximately 30 seconds.

COMMENT

This case is consistent with horizontal canal benign paroxysmal positional vertigo (BPPV) of the geotropic type. The affected side is likely the right side because having the patient turn to the right elicited the highest-velocity nystagmus. The nystagmus of horizontal canal BPPV can be triggered by the Dix-Hallpike test, although supine positional testing is more specifically designed to assess for horizontal canal BPPV. In this case, it was clearly seen as a horizontal pattern in the Dix-Hallpike test on the right side. Because only an observed prolonged right-beating nystagmus on the Dix-Hallpike test to the right was present, it would be conceivable that the patient could have a mild or recovering left-side acute unilateral vestibulopathy with the left-beating nystagmus best seen on the positional test. The fact that the nystagmus changed to left beating on supine left positional testing, however, is incompatible with an acute unilateral vestibulopathy. The affected side was likely the right side because the highest-velocity nystagmus of this geotropic pattern was triggered by turning the head to the right side. The Gufoni maneuver was used to treat this patient's right geotropic horizontal canal BPPV and resolved the symptoms and positional nystagmus. No further testing was indicated.

Migraine Dizziness

A presumed migrainous etiology is a common cause of many types of dizziness presentations. Although more commonly characterized by recurrent spontaneous dizziness, migraine dizziness can also present as episodic positional dizziness.[19,20] Vestibular migraine can closely mimic BPPV by presenting with purely positional dizziness.[19-22] When assessed in the acute setting, a study of 26 patients with migraine dizziness found that 19% had spontaneous nystagmus whereas 100% had nystagmus provoked with positional testing with fixation removed.[21] The direction of the positional nystagmus varied among the patients. The most common direction of the nystagmus was horizontal in 69% of the patients. This horizontal positional nystagmus remained constant in direction in 72% of the patients when positional testing was performed on both sides. Vertical nystagmus was seen in 19% of the patients either as upbeat nystagmus or downbeat nystagmus.

In a study of 20 patients with migrainous vertigo, positional nystagmus greater than 3 degrees per second was present in 40%.[23] In five of the patients, the nystagmus was present only on positional testing. The positional nystagmus evolved over minutes in the provocative position and persisted in all the patients as long as that head position was maintained. In three patients, positional nystagmus changed direction with different supine positions. Another study of 13 patients found that 8 had a positional upbeat nystagmus and 5 had positional

CASE 3-4

A 75-year-old man presented with approximately 3 years of mild progressive imbalance symptoms and episodes of dizziness after lying down. The symptoms were gradual in onset, and he had recently required the use of a cane for walking stability. His older brother also developed similar symptoms late in life. He had some possible slurring of speech, but he denied frank coordination problems.

On the general medical and neurologic assessment, mild dysarthria was detected. Mild dysmetria on the finger-nose-finger test was also present. On the ocular motor assessment, square-wave jerks were noted in addition to gaze-evoked nystagmus with right-beating nystagmus on right gaze and left-beating nystagmus on left gaze. He also had pathologically impaired smooth pursuit. On the Dix-Hallpike test, downbeating nystagmus was seen, which was persistent for as long as the position was held, with mild associated dizziness symptoms.

COMMENT

This patient's presentation is consistent with a degenerative neurologic disorder such as spinocerebellar ataxia type 6 or multiple system atrophy cerebellar type. The patient had prominent episodic positional dizziness but also a constant sense of imbalance and other symptoms of cerebellar dysfunction. The ocular motor examination is clearly consistent with cerebellar dysfunction. Given the reports of episodic positional dizziness, positional testing was performed, but the pattern of nystagmus was typical of cerebellar dysfunction as opposed to benign paroxysmal positional vertigo. MRI revealed mild cerebellar atrophy.

downbeat nystagmus.[22] The nystagmus was persistent and nonfatigable, and the mean velocity was 17 degrees per second (standard deviation [SD], 9.5 degrees per second; range, 7–40 degrees per second).

Light Cupula Syndrome

Light cupula syndrome is uncommon but important to be aware of because it can closely mimic horizontal canal BPPV.[24,25] Patients with light cupula syndrome typically have profound positional vertigo and a constant sense of imbalance. The nystagmus is persistent geotropic direction-changing positional nystagmus without latency or fatigability. Typically, a null point to the nystagmus exists while the patient is in a supine position with the head rotated about 20 to 30 degrees toward the affected side. The precise cause of light cupula syndrome is unclear, but the mechanisms appear similar to that of positional alcohol nystagmus. If the cupula becomes light, it becomes responsive to gravity such that the head turned to the side will persistently deflect the cupula. A mild spontaneous nystagmus while sitting can be seen in patients with light cupula syndrome, but the nystagmus stops when the head has been tilted slightly forward, about 30 degrees, placing the lateral canal parallel to the horizontal plane. In some patients, accompanying unilateral hearing loss is present, suggesting that they have a full labyrinthitis disorder. When apogeotropic nystagmus is present instead, it could be caused by heavy endolymph rather than a light cupula. Patients with light cupula syndrome are refractory to attempts at particle repositioning, and the recovery course is slow, occurring over days or weeks, similar to an acute unilateral vestibulopathy.

EVALUATION AND MANAGEMENT OF THE PATIENT WITH EPISODIC POSITIONAL DIZZINESS

The most important parts of the assessment in patients presenting with episodic positional dizziness are the details of the history and particularly the examination when in the office setting. Once the history is obtained, the clinician should perform the general neurologic examination including the ocular motor examination. It is probably a mistake to jump straight to positional testing without first inspecting the ocular motor system at rest, because in acute presentations, patients can actually have a fixed lesion (eg, acute unilateral vestibulopathy or stroke) even when the history might suggest episodic positional dizziness.

Positional testing usually begins with the Dix-Hallpike test. Some patients can identify one side that is the most likely affected. A strategy in that circumstance is to start by testing the presumed unaffected side, which serves as the internal control. But ultimately, the order should not matter. If the test starts with the right side, the clinician should turn the patient's head to the right side approximately 45 degrees and then lay the patient back. The key parts for eliciting the nystagmus are to move patients a little faster than they would normally lie down and to make sure that the head is tilted back approximately 20 degrees.

Either moving too slowly or not tilting the head back far enough may not result in sufficient particle movement to initiate the drag force required to deviate the cupula and elicit nystagmus. The patient should stay in the head-hanging position for approximately 10 to 20 seconds. If no nystagmus is seen, then the clinician should have the patient sit back up, turn the patient's head to the other side, and perform the test. If either side is positive for triggered

and transient nystagmus, the examination can immediately proceed with the canalith repositioning maneuver. With the Epley maneuver, the clinician simply turns the patient's head to the opposite side approximately 90 degrees, waits approximately 20 to 30 seconds, and then has the patient roll over until they are looking down to the ground. It is important to make sure the patient does not sit up during this rollover because many patients do try. The patient stays in that position for approximately 20 to 30 seconds and then sits up. After treating patients with the Epley maneuver, the clinician can generally spend some time explaining what was being done and showing the maneuver on a diagram. It is usually helpful to then repeat the Dix-Hallpike test and Epley maneuver because it sometimes takes more than one maneuver to get all the particles out and because doing so can also clearly demonstrate to the patient that the treatment was effective. A Cochrane collaboration meta-analysis of eight randomized controlled trials with a total of 507 participants found that conversion from a positive to a negative Dix-Hallpike test was substantially more common in the group treated with the Epley maneuver compared with the group treated with a sham maneuver (odds ratio, 9.62; 95% confidence interval, 6.0 to 15.42) (TABLE 3-2[23-35]).[1] Each study had a statistically significant benefit of the treatment compared with the control. However, the effect size of the maneuver varied from study to study with the largest effect sizes occurring when the diagnosis and treatment were performed in specialty outpatient clinics, more than one maneuver was allowed at the initial treatment, and the outcome was measured in the first week. In a study that used a 24-hour outcome assessment and up to three maneuvers performed in a specialty clinic, 80% (28 of 35) of patients with BPPV randomly assigned to expert treatment with the canalith repositional maneuver were cured compared with 10% (3 of 31) of the patients randomly assigned to sham treatment (number needed to treat, 1.4; $P < 0.001$).[26] It has also been demonstrated that patients can be taught to self-perform the Epley maneuver or other maneuvers and successfully treat their BPPV.[36-39] The self-treatment studies, however, were limited in their ability to generalize because the patients received in-person instructions and performance feedback from expert providers. The Dix-Hallpike test uses angles of the head specifically oriented to the plane of the posterior semicircular canal, but particles that are instead in the horizontal canal will also typically move enough to trigger horizontal nystagmus. However, if the Dix-Hallpike test is negative, the next step in a patient with a high suspicion of BPPV is to perform supine positional testing, which is more specifically designed to move particles in the horizontal canal. When the horizontal direction-changing positional nystagmus of horizontal canal BPPV is identified, three treatment options are supported by randomized controlled trials: the barbeque roll maneuver (also known as the *Lempert roll maneuver*) (FIGURE 3-5), the Gufoni maneuver, and the head-shaking maneuver.[34,35] The barbecue roll maneuver is similar to the Epley maneuver, but the head is resting on the bed or examination table rather than extended back. It can be used to treat both the geotropic and apogeotropic variants. The maneuver starts with the patient in the supine position, and then head rotations are made in 30- to 60-second intervals toward the healthy side for treating the geotropic variant and toward the affected side for treating the apogeotropic variant.

To use the Gufoni maneuver in the geotropic variant, the patient starts in the sitting position and then is quickly moved to a side-lying position onto the unaffected side, which moves the particles toward the utricle. The patient stays in

KEY POINTS

- Vestibular migraine can closely mimic BPPV by presenting with purely positional dizziness.
- Patients with light cupula syndrome typically have profound positional vertigo and a constant sense of imbalance. The nystagmus is persistent geotropic direction-changing positional nystagmus without latency or fatigability.
- The Dix-Hallpike test should be performed by moving the patient from the sitting to head-hanging position at a pace a bit more quickly than he or she would ordinarily lie down and trying to tilt the head back approximately 20 degrees.
- A Cochrane collaboration meta-analysis of eight randomized controlled trials found that conversion from a positive to a negative Dix-Hallpike test significantly favored the Epley treatment group when compared with a sham maneuver or control.

this position for approximately 2 minutes and then turns their head to a face-down direction for 1 minute before sitting back up. To use the Gufoni maneuver in the apogeotropic variant, the patient starts in the seated position and then is quickly moved to a side-lying position on the affected side, which moves the particles away from the cupula and toward the utricle. The patient stays in this position for approximately 2 minutes and then turns their head to look up and hold the position for approximately 1 minute before sitting up. The maneuver generally either resolves the BPPV or converts it to the geotropic variant, which can then be treated with the Gufoni maneuver for the geotropic variant. Surprisingly, the Gufoni maneuver did not demonstrate evidence of being significantly statistically better than a simple head-shaking maneuver in the apogeotropic variant.[35] The

TABLE 3-2 **Selected Data From Clinical Trials of Canalith Repositioning Maneuver Treatments for Benign Paroxysmal Positional Vertigo**

Study	Treatment, % (n/N)	Control, % (n/N)	Notes
Posterior canal benign paroxysmal positional vertigo (BPPV) clinical trials[a]			
Lynn et al,[27] 1995	89 (16/18)	27 (4/15)	Audiologist vestibular clinic; up to 4 maneuvers; outcome at 1 month.
Froehling et al,[28] 2000	67 (16/24)	38 (10/26)	General medicine clinic; up to 5 maneuvers; outcome at 1–2 weeks.
Yimtae et al,[29] 2003	88 (22/25)	65 (13/20)	Neuro-otology clinic, up to 5 maneuvers, outcome at 1 week
Munoz et al,[30] 2007	34 (13/38)	15 (6/41)	Family practice clinic, single maneuver, outcome within hours
Von Brevern et al,[26] 2006	80 (28/35)	10 (3/31)	Neuro-otology clinic, up to 3 maneuvers, outcome at 24 hours
Bruintjes et al,[31] 2014	91 (20/22)	45 (10/22)	Dizziness outpatient clinic, up to 2 maneuvers, outcome at 1 month
Liang et al,[32] 2010	98 (42/43)	77 (34/44)	1 maneuver, outcome at 4 days
Xie et al,[33] 2012	93 (54/58)	34 (11/45)	1 maneuver, outcome at 4 days
Horizontal canal BPPV clinical trials			
Kim et al,[34] 2012, geotropic	Barbeque roll maneuver: 69 (38/55) Gufoni maneuver: 61 (39/64)	35 (17/48)	10 outpatient dizziness clinics, up to 2 maneuvers, outcome at 2 hours
Kim et al,[35] 2012, apogeotropic	Gufoni maneuver: 73 (38/52) Head-shake maneuver: 62 (33/53)	35 (17/49)	10 outpatient dizziness clinics, up to 2 maneuvers, outcome at 2 hours

n = number of patients with positive outcome; N = total number of patients in trial group.
[a] Selected from the Cochrane collaboration meta-analysis.[1]

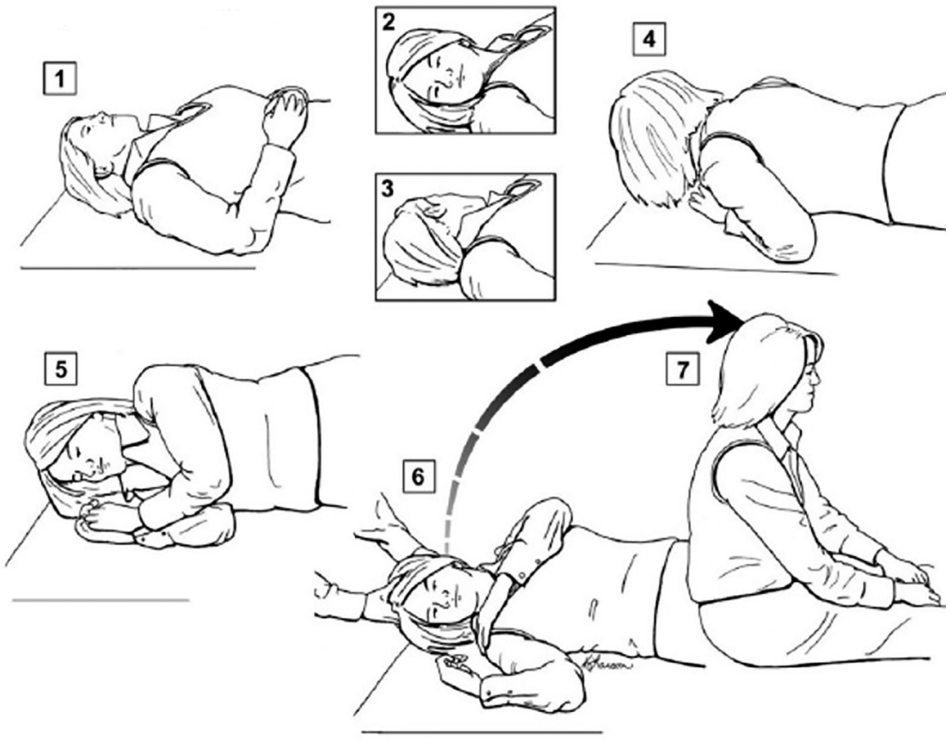

FIGURE 3-5

Barbeque roll maneuver, also referred to as the *Lempert roll maneuver*, for right-sided horizontal canal benign paroxysmal positional vertigo (BPPV). When it is determined to be horizontal canal BPPV affecting the right side, the patient is taken through a series of stepwise 90-degree turns away from the affected side in steps 1 through 5, holding each position for 10 to 30 seconds. From step 5, the patient positions their body to the back (6) in preparation for the rapid and simultaneous movement from the supine face up to the sitting position (7).

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KEY POINT

- **Horizontal canal BPPV can be treated by using the barbeque roll maneuver, the Gufoni maneuver, or the forced prolonged position.**

head-shaking maneuver is performed with the patient in the sitting position. The head is first pitched forward approximately 30 degrees and then moved sinusoidally at a rate of approximately 3 Hz for 15 seconds.

Another simple option to treat the horizontal canal variant is the forced prolonged position, which is performed by having patients lie on their healthy side for the geotropic variant or the affected side for the apogeotropic variant for approximately 12 hours.[40] It is also worth noting that horizontal canal BPPV has a natural history of spontaneous resolution within days, which is different from the typical weeks to months for the posterior canal variant.[41-43] In addition, more than one-third of the patients treated with sham maneuvers in clinical trials of treatments for horizontal canal BPPV had resolution of the BPPV within hours, and more than 80% were cured within 7 days.[34,35]

If a patient is suspected of having BPPV but the positional testing does not trigger nystagmus, several possibilities for this exist. First, a spontaneous resolution may have occurred, and the particles are already out of the canal. Second, it is possible that the positional testing was not adequately performed. This can occur if the patient is moved too slowly into the head-hanging position or if the head was not tilted back far enough. If the particles do not move fast

KEY POINT

- **If a patient is suspected of having BPPV but the positional testing does not trigger nystagmus, it is possible that the patient had spontaneous resolution, the positional testing was not adequately performed, or the patient developed an anxiety response to previous BPPV.**

enough or far enough, then the drag force of endolymph flow in the canal will not be sufficient to deviate the cupula. Lastly, if the patient reports symptoms but no nystagmus is observed, then it is possible that the particles are out of the canal but the patient has developed an anxiety response to previously provocative positions. In all these circumstances, it is still recommended to perform the repositioning maneuver for patient self-treatment education.

If the findings on examination instead suggest a central lesion, then the appropriate evaluation should follow. This typically includes an MRI of the brain to assess for a structural abnormality such as a mass lesion, stroke, demyelinating lesion, or Chiari malformation.

For patients suspected of having migraine-associated episodic positional dizziness, the treatment is usually supportive with symptomatic medications, rest, and hydration. Patients who have many recurrent episodes may benefit from a migraine prophylactic medication. However, randomized controlled trials of migraine preventative medications in patients with migraine-related dizziness have not been conducted.

Patients suspected of having light cupula syndrome require supportive care and symptomatic medications. After the acute phase, they may also benefit from vestibular rehabilitation, although clinical trials are lacking in this uncommon presentation. It is reasonable to attempt a particle repositioning maneuver because horizontal canal BPPV is typically in the differential diagnosis, but this would not be expected to treat light cupula syndrome.

Consensus clinical practice guidelines recommend limiting brain imaging studies to patients with symptoms or signs of concurrent brainstem or cerebellar dysfunction or when positional vertigo and nystagmus have atypical features or fail to resolve with repeated therapeutic positional maneuvers.[2,4]

CONCLUSION

Episodic positional dizziness is a common category of dizziness presentations. Properly placing patients in this category is a challenge because positional components are common in nearly all types of dizziness presentations. For this reason, providers should not overemphasize the presence of positional components in determining the management plan. BPPV is the prototypical episodic positional dizziness disorder and can be readily identified and treated at the bedside. The canalith repositioning maneuvers are supported by numerous randomized controlled trials, meta-analyses, and clinical guideline statements. Therefore, identifying and treating BPPV is a priority in routine practice. Central disorders are much less common causes of episodic positional dizziness but need to be considered when signs of central dysfunction are present, including central patterns of nystagmus. Providers should also be aware of migraine and light cupula syndrome as potential causes of episodic positional dizziness.

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