

Clinical Practice Guideline: Benign Paroxysmal Positional Vertigo (Update)

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Abstract

Objective. This update of a 2008 guideline from the American Academy of Otolaryngology—Head and Neck Surgery Foundation provides evidence-based recommendations to benign paroxysmal positional vertigo (BPPV), defined as a disorder of the inner ear characterized by repeated episodes of positional vertigo. Changes from the prior guideline include a consumer advocate added to the update group; new evidence from 2 clinical practice guidelines, 20 systematic reviews, and 27 randomized controlled trials; enhanced emphasis on patient education and shared decision making; a new algorithm to clarify action statement relationships; and new and expanded recommendations for the diagnosis and management of BPPV.

Purpose. The primary purposes of this guideline are to improve the quality of care and outcomes for BPPV by improving the accurate and efficient diagnosis of BPPV, reducing the inappropriate use of vestibular suppressant medications, decreasing the inappropriate use of ancillary testing such as radiographic imaging, and increasing the use of appropriate therapeutic repositioning maneuvers. The guideline is intended for all clinicians who are likely to diagnose and manage patients with BPPV, and it applies to any setting in which BPPV would be identified, monitored, or managed. The target patient for the guideline is aged ≥ 18 years with a suspected or potential diagnosis of BPPV. The primary outcome considered in this guideline is the resolution of the symptoms associated with BPPV. Secondary outcomes considered include an increased rate of accurate diagnoses of BPPV, a more efficient return to regular activities and work, decreased use of inappropriate medications and unnecessary diagnostic tests, reduction in recurrence of BPPV, and reduction in adverse events

associated with undiagnosed or untreated BPPV. Other outcomes considered include minimizing costs in the diagnosis and treatment of BPPV, minimizing potentially unnecessary return physician visits, and maximizing the health-related quality of life of individuals afflicted with BPPV.

Action Statements. The update group made *strong recommendations* that clinicians should (1) diagnose posterior semicircular canal BPPV when vertigo associated with torsional, upbeating nystagmus is provoked by the Dix-Hallpike maneuver, performed by bringing the patient from an upright to supine position with the head turned 45° to one side and neck extended 20° with the affected ear down, and (2) treat, or refer to a clinician who can treat, patients with posterior canal BPPV with a canalith repositioning procedure. The update group made a *strong recommendation against* postprocedural postural restrictions after canalith repositioning procedure for posterior canal BPPV. The update group made *recommendations* that the clinician should (1) perform, or refer to a clinician who can perform, a supine roll test to assess for lateral semicircular canal BPPV if the patient has a history compatible with BPPV and the Dix-Hallpike test exhibits horizontal or no nystagmus; (2) differentiate, or refer to a clinician who can differentiate, BPPV from other causes of imbalance, dizziness, and vertigo; (3) assess patients with BPPV for factors that modify management, including impaired mobility or balance, central nervous system disorders, a lack of home support, and/or increased risk for falling; (4) reassess patients within 1 month after an initial period of observation or treatment to document resolution or persistence of symptoms; (5) evaluate, or refer to a clinician who can evaluate, patients with persistent symptoms for unresolved BPPV and/or underlying peripheral vestibular or central nervous system disorders; and (6) educate patients regarding the impact of BPPV on their safety, the potential for disease recurrence, and the importance of follow-up. The update group made *recommendations against* (1) radiographic imaging for a patient who meets diagnostic criteria for BPPV in

the absence of additional signs and/or symptoms inconsistent with BPPV that warrant imaging, (2) vestibular testing for a patient who meets diagnostic criteria for BPPV in the absence of additional vestibular signs and/or symptoms inconsistent with BPPV that warrant testing, and (3) routinely treating BPPV with vestibular suppressant medications such as antihistamines and/or benzodiazepines. The guideline update group provided the *options* that clinicians may offer (1) observation with follow-up as initial management for patients with BPPV and (2) vestibular rehabilitation, either self-administered or with a clinician, in the treatment of BPPV.

Keywords

benign paroxysmal positional vertigo, BPPV

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Differences from Prior Guideline

This clinical practice guideline is as an update and replacement for an earlier guideline published in 2008 by the American Academy of Otolaryngology—Head and Neck Surgery Foundation (AAO-HNSF).¹ An update was necessitated by new primary studies and systematic reviews that might suggest a need for modifying clinically important recommendations. Changes in content and methodology from the prior guideline include the following:

- Addition of a patient advocate to the guideline development group
- New evidence from 2 clinical practice guidelines, 20 systematic reviews, and 27 randomized controlled trials (RCTs)
- Emphasis on patient education and shared decision making
- Expanded action statement profiles to explicitly state quality improvement opportunities, confidence in the evidence, intentional vagueness, and differences of opinion
- Enhanced external review process to include public comment and journal peer review

- New algorithm to clarify decision making and action statement relationships
- New recommendation regarding canalith repositioning postprocedural restrictions
- Expansion of the recommendations regarding radiographic and vestibular testing
- Removal of the “no recommendation” for audiometric testing
- Addition of a diagnostic and treatment visual algorithm

Introduction

A primary complaint of dizziness accounts for 5.6 million clinic visits in the United States per year, and between 17% and 42% of patients with vertigo ultimately receive a diagnosis of benign paroxysmal positional vertigo (BPPV).²⁻⁴ BPPV is a form of positional vertigo.

- *Vertigo* is defined as an illusory sensation of motion of either the self or the surroundings in the absence of true motion.
- Positional vertigo is defined as a spinning sensation produced by changes in head position relative to gravity.
- BPPV is defined as a disorder of the inner ear characterized by repeated episodes of positional vertigo (**Table 1**).

Traditionally, the terms “benign” and “paroxysmal” have been used to characterize this particular form of positional vertigo. In this context, the descriptor *benign* historically implies that BPPV was a form of positional vertigo not due to any serious central nervous system (CNS) disorder and that there was an overall favorable prognosis for recovery.⁵ This favorable prognosis is based in part on the fact that BPPV can recover spontaneously in approximately 20% of patients by 1 month of follow-up and up to 50% at 3 months.^{6,7} However, the clinical and quality-of-life impacts of undiagnosed and untreated BPPV may be far from “benign,” as patients with BPPV are at increased risk for falls and impairment in the performance of daily activities.⁸

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Table 1. Definitions of Common Terms.

Term	Definition
Vertigo	An illusory sensation of motion of either the self or the surroundings in the absence of true motion.
Nystagmus	A rapid, involuntary oscillatory movement of the eyeball.
Vestibular system/apparatus	The sensory system within the inner ear that, with the vestibular nerve and its connections in the brain, provides the fundamental input to the brain regarding balance and spatial orientation.
Positional vertigo	Vertigo produced by changes in the head position relative to gravity.
Benign paroxysmal positional vertigo (BPPV)	A disorder of the inner ear characterized by repeated episodes of positional vertigo.
Posterior canal BPPV	A form of BPPV in which dislodged inner ear particles in the posterior semicircular canal abnormally influence the balance system producing the vertigo, most commonly diagnosed with the Dix-Hallpike test.
Lateral canal BPPV	A form of BPPV in which dislodged inner ear particles in the lateral semicircular canal abnormally influence the balance system producing the vertigo, most commonly diagnosed by the supine roll test.
Canalithiasis	A theory for the pathogenesis of BPPV that proposes that there are free-floating particles (otoconia) that have moved from the utricle and collect near the cupula of the affected canal, causing forces in the canal leading to abnormal stimulation of the vestibular apparatus.
Cupulolithiasis	A theory for the pathogenesis of BPPV that proposes that otoconial debris attached to the cupula of the affected semicircular canal cause abnormal stimulation of the vestibular apparatus.
Canalith repositioning procedures (CRPs)	A group of procedures in which the patient moves through specific body positions designed to relocate dislodged particles within the inner ear for the purpose of relieving symptoms of BPPV. The specific CRP chosen relates to the type of BPPV diagnosed. These have also been termed <i>canalith repositioning maneuvers</i> or <i>canalith repositioning techniques</i> .

Furthermore, patients with BPPV experience effects on individual health-related quality of life, and utility measures demonstrate that treatment of BPPV results in improvement in quality of life.⁹ The term *paroxysmal* in this context describes the rapid and sudden onset of vertigo, initiated at any time by a change of position, thus resulting in BPPV. BPPV has also been termed *benign positional vertigo*, *paroxysmal positional vertigo*, *positional vertigo*, *benign paroxysmal nystagmus*, and *paroxysmal positional nystagmus*. In this guideline, the panel chose to retain the terminology of BPPV, as it is the most common terminology encountered in the literature and in clinical practice.⁸

BPPV is most commonly clinically encountered as 1 of 2 variants: BPPV of the posterior semicircular canal (posterior canal BPPV) or BPPV of the lateral semicircular canal (also known as horizontal canal BPPV).¹⁰⁻¹² Posterior canal BPPV is more common than horizontal canal BPPV, constituting approximately 85% to 95% of BPPV cases.¹² Although debated, posterior canal BPPV is most commonly thought to be due to canalithiasis, wherein fragmented otolith particles (otoconia) entering the posterior canal become displaced, cause inertial changes to the cupula in the posterior canal, and thereby result in abnormal nystagmus and vertigo when the head encounters motion in the plane of the affected semicircular canal.^{12,13} Lateral (horizontal) canal BPPV accounts for 5% to 15% of BPPV cases.^{11,12} The etiology of lateral canal BPPV is also felt to be due to the presence of abnormal debris within the lateral canal, but the pathophysiology is not as well understood as that of posterior canal BPPV. Other rare variations include anterior canal BPPV, multicanal BPPV, and bilateral multicanal BPPV.

Guideline Purpose

The primary purposes of this guideline are to improve quality of care and outcomes for BPPV by improving the accurate and efficient diagnosis of BPPV, reducing the inappropriate use of vestibular suppressant medications, decreasing the inappropriate use of ancillary testing such as radiographic imaging, and increasing the use of appropriate therapeutic repositioning maneuvers. The guideline is intended for all clinicians who are likely to diagnose and manage patients with BPPV, and it applies to any setting in which BPPV would be identified, monitored, or managed. The target patient for the guideline is aged ≥ 18 years with a suspected or potential diagnosis of BPPV. The pediatric population was not included in the target population, in part due to a substantially smaller body of evidence on pediatric BPPV. No specific recommendations are made concerning surgical therapy for BPPV.

The guideline focuses on BPPV, recognizing that BPPV may arise in conjunction with other neurologic or otologic conditions and that the treatment of the symptom components specifically related to BPPV may still be managed according to the guideline. This guideline does not discuss BPPV affecting the anterior semicircular canal, as this diagnosis is quite rare and its pathophysiology is poorly understood.^{14,15} It also does not discuss benign paroxysmal vertigo of childhood, disabling positional vertigo due to vascular loop compression in the brainstem, or vertigo that arises from changes in head position *not* related to gravity (ie, vertigo of cervical origin or vertigo of vascular origin). These conditions are physiologically distinct from BPPV.

Table 2. Interventions Considered in Benign Paroxysmal Positional Vertigo Guideline Development.

Diagnosis	Clinical history Review of the medication list Physical examination Dix-Hallpike (positional) testing Supine roll test and bow and lean test side-lying maneuver Post-head-shaking nystagmus Audiometry Magnetic resonance imaging Computed tomography Blood tests: complete blood count, serum chemistry, etc Frenzel lenses and infrared goggle testing Electronystagmography Videonystagmography Vestibular evoked myogenic potentials Balance and gait testing Vestibular function testing Computerized posturography Orthostatic balance testing Vestibular caloric testing
Treatment	Watchful waiting/observation Education/information/counseling Medical therapy (vestibular suppressant medications, benzodiazepines) Cervical immobilization with cervical collar Prolonged upright position Patient self-treatment with home-based maneuvers or rehabilitation Brandt-Daroff exercises Epley maneuver and modifications of the Epley maneuver Semont maneuver Gufoni maneuver Physical therapy/vestibular physical therapy Spinal manipulative therapy Mastoid vibration Posterior semicircular canal occlusion (excluded from guideline) Singular neurectomy (excluded from guideline) Vestibular neurectomy (excluded from guideline)
Prevention	Head trauma or whiplash injury as potential causative factors Use of helmets to prevent head trauma and/or cervical collars Fall prevention

In 2008, the AAO-HNSF published a multidisciplinary clinical practice guideline on benign positional vertigo.¹ As 8 years have elapsed since the publication of that guideline, a multidisciplinary guideline update group was convened to perform an assessment and planned update of that guideline, utilizing the most current evidence base. Our goal was to revise the prior guideline with an *a priori* determined transparent process, reconsidering a more current evidence base while taking into account advances in knowledge with respect to BPPV.

The primary outcome considered in this guideline is the resolution of symptoms associated with BPPV. Secondary outcomes considered include an increased rate of accurate diagnoses of BPPV, a more efficient return to regular activities and work, decreased use of inappropriate medications and unnecessary diagnostic tests, reduction in recurrence of BPPV, and reduction in adverse events associated with undiagnosed or untreated BPPV. Other outcomes considered include minimizing costs in the diagnosis and treatment of BPPV, minimizing potentially unnecessary return physician visits, and maximizing

the health-related quality of life of individuals afflicted with BPPV. The significant incidence of BPPV, its functional impact, and the wide diversities of diagnostic and therapeutic interventions for BPPV (**Table 2**) make this an important condition for an up-to-date evidence-based practice guideline.

Health Care Burden

Overall, the prevalence of BPPV has been reported to range from 10.7 to 140 per 100,000 population.¹⁶⁻¹⁸ However, studies of select patients have estimated a prevalence of 900 per 10,000.¹⁹⁻²¹ Others have reported a lifetime prevalence of 2.4%, a 1-year prevalence of 1.6%, and a 1-year incidence of 0.6%.²² Women are more frequently affected than men, with a female:male ratio of 2.2 to 1.5:1.²³ BPPV is also the most common vestibular disorder across the life span,^{12,24,25} although the age of onset is most commonly between the fifth and seventh decades of life.⁵ Given the noteworthy prevalence of BPPV, its health care and societal impacts are tremendous.

The costs to the health care system and the indirect costs of BPPV are also significant. It is estimated that it costs approximately \$2000 to arrive at the diagnosis of BPPV and that >65% of patients with this condition will undergo potentially unnecessary diagnostic testing or therapeutic interventions.²⁶ Therefore, health care costs associated with the diagnosis of BPPV alone approach \$2 billion per year. Furthermore, despite the fact that the natural history of BPPV includes a spontaneous resolution rate ranging from 27% to 50%, this often takes a significant amount of time, and almost 86% of patients with BPPV will suffer some interrupted daily activities and lost days at work due to BPPV.^{22,27} In addition, 68% of patients with BPPV will reduce their workload, while 4% will change their job and 6% will quit their job as a result of the condition.²⁸ Furthermore, BPPV is more common in older individuals, with a correspondingly more pronounced health and quality-of-life impact. It has been estimated that 9% of elderly patients undergoing comprehensive geriatric assessment for nonbalance-related complaints have unrecognized BPPV.¹⁹ More recent studies of symptomatic individuals have found BPPV to be present in 40% of geriatric patients seen for dizziness, with an overall general prevalence of 3.4% in individuals aged >60.^{22,29}

Older patients with BPPV experience a greater incidence of falls, depression, and impairments of their daily activities.¹⁹ Persistent untreated or undiagnosed vertigo in the elderly leads to increased caregiver burden with resultant societal costs including decreased family productivity and increased risk of nursing home placement. Among an estimated 7.0 million elderly individuals reporting dizziness in the prior 12 months, 2.0 million (30.1%) reported vertigo, and there were 230,000 office visits among the elderly with a diagnosis of BPPV.^{30,31} With the increasing age of the US population, the incidence and prevalence of BPPV may correspondingly increase over the next 20 years.

BPPV may be diagnosed and treated by multiple clinical disciplines. Despite its significant prevalence and quality-of-life and economic impacts, considerable practice variations exist in the management of BPPV across disciplines.³² These variations relate to diagnostic strategies for BPPV, timeliness of referral and rates of utilization of various treatment options available for BPPV within and across the various medical specialties and disciplines involved in its management. For example, the utilization of medications for the treatment of BPPV vary substantially among primary care providers and across specialties.³³ Delays in the diagnosis and treatment of BPPV have cost and quality-of-life implications for patients and their caregivers.

Fife and FitzGerald found that patients with BPPV suffer from delays in diagnosis and treatment on the order of months.³³ Other authors have found that only 10% to 20% of patients with BPPV seen by a physician will receive appropriate repositioning maneuvers.^{22,34} Furthermore, a large number of patients with BPPV will undergo unnecessary diagnostic testing and treatments prior to referral to a specialist. A recent study reported that 70% of patients with BPPV will undergo magnetic resonance imaging scanning, 45% will have a computed tomography scan, and 41% will have

an electrocardiogram, while 53% will be treated with medications.³⁵ Therefore, significant improvements in the diagnosis and treatment of patients with BPPV may lead to significant health care quality improvements as well as medical and societal cost savings. Such improvements may be achievable with the composition and implementation of a well-constructed clinical practice guideline for BPPV.

Methods

General Methods and Literature Search

In developing this update of the evidence-based clinical practice guideline, the methods outlined in the third edition of the AAO-HNSF's guideline development manual were followed explicitly.³⁶

An executive summary of the original BPPV guideline¹ was sent to a panel of expert reviewers from the fields of general otolaryngology, otology, neurotology, neurology, family practice, nursing, physical therapy, emergency medicine, radiology, audiology, and complementary medicine who assessed the key action statements to decide if they should be kept in their current form, revised, or removed and to identify new research that might affect the guideline recommendations. The reviewers concluded that the original guideline action statements remained valid but should be updated with minor modifications. Suggestions were also made for new key action statements.

An information specialist conducted 2 systematic literature searches using a validated filter strategy to identify clinical practice guidelines, systematic reviews, and RCTs published since the prior guideline (2008). Search terms used were "Benign Paroxysmal Positional Vertigo"[Mesh] OR "Benign Paroxysmal Positional Vertigo"[tab] OR "Benign Positional Vertigo"[tiab] OR BPPV[tiab] OR (BPV[tiab] AND vertigo). In certain instances, targeted searches for lower-level evidence were performed to address gaps from the systematic searches identified in writing the guideline. The original search was updated from January 2008 to September 2015 to include MEDLINE, National Guidelines Clearinghouse, Canadian Medical Association Database, NHS Evidence ENT and Audiology, National Institutes for Health and Care Excellence UK, Australian National Health and Medical Research Council, Guideline Internal Network, Cochrane Database of Systematic Reviews, EMBASE, Cumulative Index to Nursing and Allied Health, Web of Science, and the Allied and Complementary Medicine Database.

1. The initial search for clinical practice guidelines identified 2 guidelines. Quality criteria for including guidelines were (a) an explicit scope and purpose, (b) multidisciplinary stakeholder involvement, (c) systematic literature review, (d) explicit system for ranking evidence, and (e) explicit system for linking evidence to recommendations. The final data set retained 2 guidelines that met inclusion criteria.
2. The initial search for systematic reviews identified 44 systematic reviews or meta-analyses that were

distributed to the panel members. Quality criteria for including reviews were (a) relevance to the guideline topic, (b) clear objective and methodology, (c) explicit search strategy, and (d) valid data extraction methods. The final data set retained was 20 systematic reviews or meta-analyses that met inclusion criteria.

3. The initial search for RCTs identified 38 RCTs that were distributed to panel members for review. Quality criteria for including RCTs were (a) relevance to the guideline topic, (b) publication in a peer-reviewed journal, and (c) clear methodology with randomized allocation to treatment groups. The total final data set retained 27 RCTs that met inclusion criteria.

The AAO-HNSF assembled a guideline update group representing the disciplines of otolaryngology–head and neck surgery, otology, neurotology, family medicine, audiology, emergency medicine, neurology, physical therapy, advanced practice nursing, and consumer advocacy. The guideline update group had several conference calls and 1 in-person meeting, during which it defined the scope and objectives of updating the guideline, reviewed comments from the expert panel review for each key action statement, identified other quality improvement opportunities, and reviewed the literature search results.

The evidence profile for each statement in the earlier guideline was then converted into an expanded action statement profile for consistency with our current development standards.³⁶ Information was added to the action statement profiles regarding the quality improvement opportunity to which the action statement pertained, the guideline panel's level of confidence in the published evidence, differences of opinion among panel members, intentional vagueness, and any exclusion to which the action statement does not apply. New key action statements were developed with an explicit and transparent a priori protocol for creating actionable statements based on supporting evidence and the associated balance of benefit and harm. Electronic decision support software (BRIDGE-Wiz; Yale Center for Medical Informatics, New Haven, Connecticut) was used to facilitate creating actionable recommendations and evidence profiles.³⁷

The updated guideline then underwent GuideLine Implementability Appraisal to appraise adherence to methodologic standards, to improve clarity of recommendations, and to predict potential obstacles to implementation.³⁸ The guideline update group received summary appraisals and modified an advanced draft of the guideline based on the appraisal. The final draft of the updated clinical practice guideline was revised according to comments received during multidisciplinary peer review, open public comment, and journal editorial peer review. A scheduled review process will occur at 5 years from publication or sooner if new compelling evidence warrants earlier consideration.

Classification of Evidence-Based Statements. Guidelines are intended to reduce inappropriate variations in clinical care, to produce optimal health outcomes for patients, and to minimize harm. The evidence-based approach to guideline

development requires that the evidence supporting a policy be identified, appraised, and summarized and that an explicit link between evidence and statements be defined. Evidence-based statements reflect both the *quality of evidence* and the *balance of benefit and harm* that is anticipated when the statement is followed. The definitions for evidence-based statements are listed in **Tables 3** and **4**.

Guidelines are never intended to supersede professional judgment; rather, they may be viewed as a relative constraint on individual clinician discretion in a particular clinical circumstance. Less frequent variation in practice is expected for a strong recommendation than what might be expected with a recommendation. Options offer the most opportunity for practice variability.³⁹ Clinicians should always act and decide in a way that they believe will best serve their individual patients' interests and needs, regardless of guideline recommendations. Guidelines represent the best judgment of a team of experienced clinicians and methodologists addressing the scientific evidence for a particular topic.⁴⁰

Making recommendations about health practices involves value judgments on the desirability of various outcomes associated with management options. Values applied by the guideline update group sought to minimize harm and diminish unnecessary and inappropriate therapy. A major goal of the panel was to be transparent and explicit about how values were applied and to document the process.

Financial Disclosure and Conflicts of Interest. The cost of developing this guideline, including travel expenses of all panel members, was covered in full by the AAO-HNSF. Potential conflicts of interest for all panel members in the past 5 years were compiled and distributed before the first conference call and were updated at each subsequent call and in-person meeting.⁴¹ After review and discussion of these disclosures, the panel concluded that individuals with potential conflicts could remain on the panel if they (1) reminded the panel of potential conflicts before any related discussion, (2) recused themselves from a related discussion if asked by the panel, and (3) agreed not to discuss any aspect of the guideline with industry before publication.⁴¹ Last, panelists were reminded that conflicts of interest extend beyond financial relationships and may include personal experiences, how a participant earns a living, and the participant's previously established "stake" in an issue.⁴²

Guideline Key Action Statements

Each evidence-based statement is organized in a similar fashion: a key action statement is in **bold**, followed by the strength of the recommendation in *italics*. Each key action statement is followed by an "action statement profile" that explicitly states the quality improvement opportunity, aggregate evidence quality, level of confidence in evidence (high, medium, low), benefit, harms, risks, costs, and a benefits-harm assessment. Additionally, there are statements of any value judgments, the role of patient preferences, clarification of any intentional vagueness by the panel, exceptions to the statement, any

Table 3. Strength of Action Terms in Guideline Statements and Implied Levels of Obligation.

Strength	Definition	Implied Obligation
Strong recommendation	A strong recommendation means that the benefits of the recommended approach clearly exceed the harms (or, in the case of a strong negative recommendation, that the harms clearly exceed the benefits) and that the quality of the supporting evidence is high (grade A or B). ^a In some clearly identified circumstances, strong recommendations may be made according to lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits strongly outweigh the harms.	Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
Recommendation	A recommendation means that the benefits exceed the harms (or, in the case of a negative recommendation, that the harms exceed the benefits), but the quality of evidence is not as high (grade B or C). ^a In some clearly identified circumstances, recommendations may be made according to lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits outweigh the harms.	Clinicians should also generally follow a recommendation, but should remain alert to new information and sensitive to patient preferences.
Option	An option means either that the quality of evidence is suspect (grade D) ^a or that well-done studies (grade A, B, or C) ^a show little clear advantage to one approach versus another.	Clinicians should be flexible in their decision making regarding appropriate practice, although they may set bounds on alternatives; patient preference should have a substantial influencing role.

^aSee **Table 4** for definitions of evidence grades.

Table 4. Aggregate Grades of Evidence by Question Type.^a

Grade	CEBM Level	Treatment	Harm	Diagnosis	Prognosis
A	I	Systematic review ^b of randomized trials	Systematic review ^b of randomized trials, nested case-control studies, or observational studies with dramatic effect	Systematic review ^b of cross-sectional studies with consistently applied reference standard and blinding	Systematic review ^b of inception cohort studies ^c
B	2	Randomized trials or observational studies with dramatic effects or highly consistent evidence	Randomized trials or observational studies with dramatic effects or highly consistent evidence	Cross-sectional studies with consistently applied reference standard and blinding	Inception cohort studies ^c
C	3-4	Nonrandomized or historically controlled studies, including case-control and observational studies	Nonrandomized controlled cohort or follow-up study (postmarketing surveillance) with sufficient numbers to rule out a common harm; case-series, case-control, or historically controlled studies	Nonconsecutive studies; case-control studies; or studies with poor, nonindependent, or inconsistently applied reference standards	Cohort study, control arm of a randomized trial, case series, or case-control studies; poor-quality prognostic cohort study
D	5	Case reports, mechanism-based reasoning, or reasoning from first principles			
X	n/a	Exceptional situations where validating studies cannot be performed and there is a clear preponderance of benefit over harm			

Abbreviation: CEBM, Oxford Centre for Evidence-Based Medicine.

^aAdapted from Howick and coworkers.²⁸⁹

^bA systematic review may be downgraded to level B because of study limitations, heterogeneity, or imprecision.

^cA group of individuals identified for subsequent study at an early uniform point in the course of the specified health condition or before the condition develops.

Table 5. Summary of Guideline Key Action Statements.

Statement	Action	Strength
1a. Diagnosis of posterior semicircular canal BPPV	Clinicians should diagnose posterior semicircular canal BPPV when vertigo associated with torsional, upbeat nystagmus is provoked by the Dix-Hallpike maneuver, performed by bringing the patient from an upright to supine position with the head turned 45° to one side and neck extended 20° with the affected ear down. The maneuver should be repeated with the opposite ear down if the initial maneuver is negative.	Strong recommendation
1b. Diagnosis of lateral (horizontal) semicircular canal BPPV	If the patient has a history compatible with BPPV and the Dix-Hallpike test exhibits horizontal or no nystagmus, the clinician should perform, or refer to a clinician who can perform, a supine roll test to assess for lateral semicircular canal BPPV.	Recommendation
2a. Differential diagnosis	Clinicians should differentiate, or refer to a clinician who can differentiate, BPPV from other causes of imbalance, dizziness, and vertigo.	Recommendation
2b. Modifying factors	Clinicians should assess patients with BPPV for factors that modify management, including impaired mobility or balance, central nervous system disorders, a lack of home support, and/or increased risk for falling.	Recommendation
3a. Radiographic testing	Clinicians should <i>not</i> obtain radiographic imaging in a patient who meets diagnostic criteria for BPPV in the absence of additional signs and/or symptoms inconsistent with BPPV that warrant imaging.	Recommendation (against)
3b. Vestibular testing	Clinicians should <i>not</i> order vestibular testing in a patient who meets diagnostic criteria for BPPV in the absence of additional vestibular signs and/or symptoms inconsistent with BPPV that warrant testing.	Recommendation (against)
4a. Repositioning procedures as initial therapy	Clinicians should treat, or refer to a clinician who can treat, patients with posterior canal BPPV with a canalith repositioning procedure.	Strong recommendation
4b. Postprocedural restrictions	Clinicians should <i>not</i> recommend postprocedural postural restrictions after canalith repositioning procedure for posterior canal BPPV.	Strong recommendation (against)
4c. Observation as initial therapy	Clinicians may offer observation with follow up as initial management for patients with BPPV.	Option
5. Vestibular rehabilitation	The clinician may offer vestibular rehabilitation, either self-administered or with a clinician, in the treatment of BPPV.	Option
6. Medical therapy	Clinicians should <i>not</i> routinely treat BPPV with vestibular suppressant medications such as antihistamines and/or benzodiazepines.	Recommendation (against)
7a. Outcome assessment	Clinicians should reassess patients within 1 month after an initial period of observation or treatment to document resolution or persistence of symptoms.	Recommendation
7b. Evaluation of treatment failure	Clinicians should evaluate, or refer to a clinician who can evaluate, patients with persistent symptoms for unresolved BPPV and/or underlying peripheral vestibular or central nervous system disorders.	Recommendation
8. Education	Clinicians should educate patients regarding the impact of BPPV on their safety, the potential for disease recurrence, and the importance of follow-up.	Recommendation

Abbreviation: BPPV, benign paroxysmal positional vertigo.

differences of opinion, and a repeat statement of the strength of the recommendation. Several paragraphs subsequently discuss the evidence base supporting the statement. An overview of each evidence-based statement in this guideline can be found in **Table 5**.

The role of patient preferences in making decisions deserves further clarification. The guideline update group classified the role of patient preference based on consensus among the group as “none, small, moderate, or large.” For some statements where the evidence base demonstrates clear benefit, although the role of patient preference for a range of treatments may not be relevant (eg, with intraoperative decision making), clinicians should provide patients with clear and comprehensible information on the benefits to facilitate patient understanding and shared decision

making, which in turn lead to better patient adherence and outcomes. In cases where evidence is weak or benefits unclear, the practice of shared decision making—again, where the management decision is made by a collaborative effort between the clinician and an informed patient—is extremely useful. Factors related to patient preference include (but are not limited to) absolute benefits, adverse effects, cost of drugs or procedures, and frequency and duration of treatment, as well as certain less tangible factors such as religious and/or cultural beliefs or personal levels of desire for intervention.

STATEMENT 1a. DIAGNOSIS OF POSTERIOR SEMI-CIRCULAR CANAL BPPV: Clinicians should diagnose

posterior semicircular canal BPPV when vertigo associated with torsional, upbeat nystagmus is provoked by the Dix-Hallpike maneuver, performed by bringing the patient from an upright to supine position with the head turned 45° to 1 side and neck extended 20° with the affected ear down. The maneuver should be repeated with the opposite ear down if the initial maneuver is negative.

Strong recommendation based on diagnostic studies with minor limitations and a preponderance of benefit over harm.

Action Statement Profile for Statement 1a

- Quality improvement opportunity: Promoting accurate and efficient diagnosis of BPPV (National Quality Strategy domains: promoting effective prevention/treatments, affordable quality care)
- Aggregate evidence quality: Grade B based on diagnostic studies with minor limitations
- Level of confidence in evidence: High
- Benefits: Improved diagnostic accuracy and efficiency
- Risks, harms, costs: Risk of provoking temporary symptoms of BPPV
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Conclusion that paroxysmal positional nystagmus induced by the Dix-Hallpike maneuver confirms the diagnosis of BPPV and is the gold standard test for diagnosis. The panel emphasized that a history of positional vertigo alone is not adequate to make the diagnosis of posterior canal BPPV
- Role of patient preferences: Small
- Intentional vagueness: None
- Exceptions: Patients with physical limitations including cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, known cerebrovascular disease, and the morbidly obese
- Policy level: Strong recommendation
- Differences of opinion: None

Supporting Text. The purpose of this statement is to emphasize that clinicians should diagnose posterior semicircular canal BPPV when vertigo associated with torsional, upbeat nystagmus is provoked by the Dix-Hallpike maneuver (**Figure 1**), performed by bringing the patient from an upright to supine position with the head turned 45° to 1 side and neck extended 20° with the affected ear down. If the testing of the first side is negative, the Dix-Hallpike maneuver should be conducted with the other ear down before concluding a negative overall maneuver.

Posterior semicircular canal BPPV is diagnosed when (1) patients report a history of vertigo provoked by changes in

head position relative to gravity and (2) when, on physical examination, characteristic nystagmus is provoked by the Dix-Hallpike maneuver (**Table 6**). Although most cases of BPPV are due to freely mobile calcium carbonate material within the lumen of the affected semicircular canal (so-called canalolithiasis), a form of posterior canal BPPV due to calcium carbonate material actually attached to the cupula (cupulolithiasis) may occur that results in nystagmus that may persist for >1 minute.⁴³

History. Vertigo has been defined as an “illusory sensation of motion of either the self or the surroundings.”⁴⁴ The symptoms of vertigo resulting from posterior canal BPPV are typically described by the patient as a rotational or spinning sensation when she or he changes head position relative to gravity. The episodes are often provoked by everyday activities and commonly occur when rolling over in bed or when the patient is tilting the head to look upward (eg, to place an object on a shelf higher than the head) or bending forward (eg, to tie his or her shoes).^{22,45-47}

Patients with BPPV most commonly report discrete, episodic periods of vertigo lasting ≤1 minute and often report modifications or limitations of their general movements to avoid provoking the vertiginous episodes.⁴⁸ Other investigators report that true “room spinning” vertigo is not always present as a reported symptom in posterior canal BPPV, with patients alternatively complaining of light-headedness, dizziness, nausea, or the feeling of being “off balance.”^{3,22,45,49-54} Approximately 50% of patients also report subjective imbalance between the classic episodes of BPPV.²² In contrast, a history of vertigo *without* associated light-headedness may increase the a priori likelihood of a diagnosis of posterior canal BPPV.¹⁹ In up to one-third of cases with atypical histories of positional vertigo, Dix-Hallpike testing will still reveal positional nystagmus, strongly suggesting the diagnosis of posterior canal BPPV.⁵⁴

Other authors have loosened the historical criteria required for a BPPV diagnosis and have coined the term “subjective BPPV” without a positive Dix-Hallpike test.^{52,55} However, in clinical practice, there is a practical need to balance inclusiveness of diagnosis with accuracy of diagnosis. Given that the majority of treatment trials and systematic reviews of BPPV require both a history of episodic positional vertigo symptoms and a positive Dix-Hallpike test, history alone is insufficient to render an accurate diagnosis of BPPV.

Physical Examination. In addition to the historical criteria for the diagnosis of posterior canal BPPV, clinicians should confirm the diagnosis of posterior canal BPPV by performing the Dix-Hallpike maneuver (**Figure 1**).

The nystagmus produced by the Dix-Hallpike maneuver in posterior canal BPPV typically displays 2 important diagnostic characteristics. First, there is a latency period between the completion of the maneuver and the onset of subjective rotational vertigo and the objective nystagmus. The latency period for the nystagmus onset with this maneuver is largely unspecified in the literature, but the panel felt that a typical latency

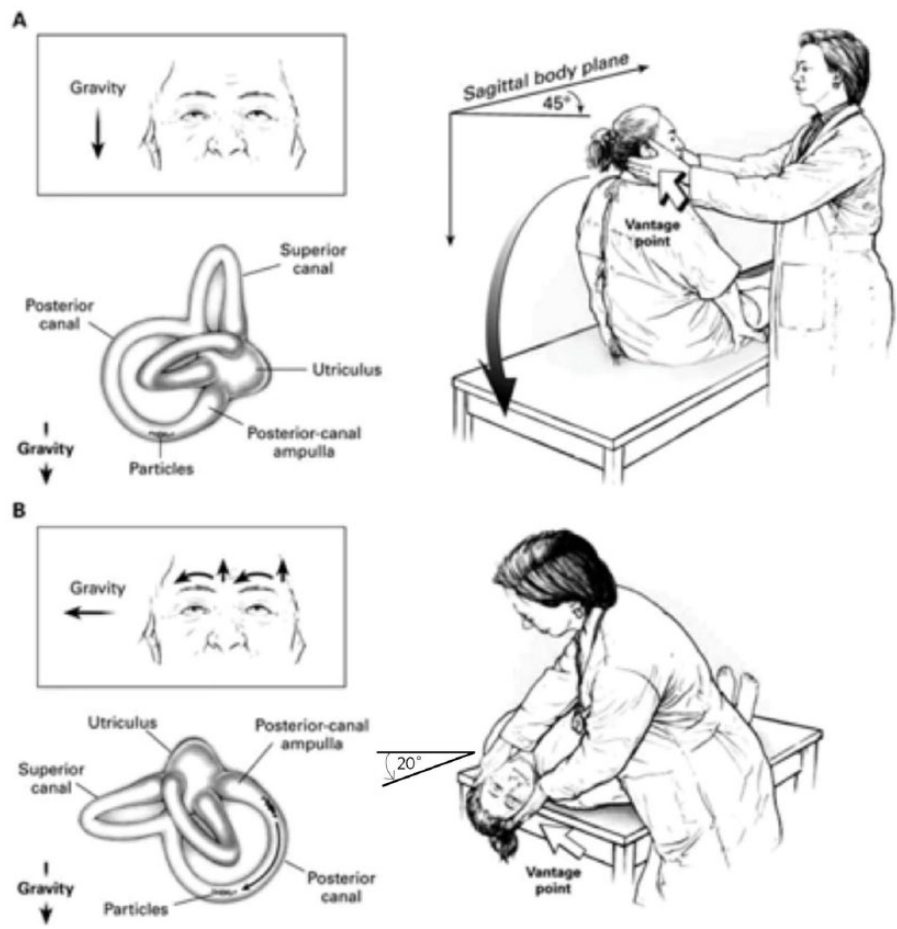


Figure 1. Diagrammatic representation of performance of the Dix-Hallpike maneuver for the diagnosis of posterior canal benign paroxysmal positional vertigo (BPPV). In panel A, the examiner stands at the patient’s right side and rotates the patient’s head 45° to the right to align the right posterior semicircular canal with the sagittal plane of the body. In panel B, the examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient’s neck 20° so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The arrows in the inset depict the direction of nystagmus in patients with typical BPPV. A presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown. From *New England Journal of Medicine*, Furman JM, Cass SP, “Benign Paroxysmal Positional Vertigo,” 341:1590-1596. Copyright © 1999 Massachusetts Medical Society. Adapted and reprinted with permission from Massachusetts Medical Society.

Table 6. Diagnostic Criteria for Posterior Canal Benign Paroxysmal Positional Vertigo.

History	Patient reports repeated episodes of vertigo with changes in head position relative to gravity.
Physical examination	Each of the following criteria is fulfilled: <ul style="list-style-type: none">• Vertigo associated with torsional (rotatory), upbeat (toward the forehead) nystagmus is provoked by the Dix-Hallpike test.• There is a latency period between the completion of the Dix-Hallpike maneuver and the onset of vertigo and nystagmus.• The provoked vertigo and nystagmus increase and then resolve within 60 seconds from the onset of the nystagmus.

period would range from 5 to 20 seconds. In rare cases, the latency period may be as long as 1 minute.⁵ Second, the provoked subjective vertigo and the nystagmus increase and then resolve within 60 seconds from the nystagmus onset.

The fast component of the nystagmus provoked by the Dix-Hallpike maneuver demonstrates a characteristic mixed torsional and vertical movement (often described as

upbeating-torsional) with the upper pole of the eye beating toward the dependent ear and the vertical component beating toward the forehead (when the eyes are positioned looking straightforward in the midorbit when the provoking position is assumed; **Figure 1**).^{45,56} Temporally, the rate of nystagmus typically begins gently, increases in intensity, and then declines in intensity as it resolves. This has been termed

crecendo-decrescendo nystagmus. After the patient returns to the upright head position, the nystagmus is again commonly observed, and the direction of the nystagmus may be reversed.

Another classic feature associated with posterior canal BPPV is that the nystagmus typically fatigues (a reduced nystagmus response) when the maneuver is repeated.^{46,56} However, repeating the Dix-Hallpike maneuver to demonstrate fatigability is not recommended, because it unnecessarily subjects patients to repeated vertigo symptoms, which is discomforting. Furthermore, repeating Dix-Hallpike maneuvers may interfere with the immediate bedside treatment of BPPV.⁴⁵ Therefore, the panel did not include nystagmus fatigability as a diagnostic criterion.

In addition to posterior canal BPPV, patients may rarely have anterior canal BPPV. Even though anterior canal BPPV is uncommon accounting for 1% to 3% of cases,⁵⁷ it is important to recognize the direction of the vertical component of the provoked torsional nystagmus to make the correct diagnosis. A downbeating vertical component in addition to the torsional nystagmus toward the dependent ear could imply anterior canal rather than posterior canal BPPV.⁵⁷⁻⁵⁹ This diagnosis should be considered with caution because downbeating positional nystagmus related to brainstem or cerebellar lesion can produce a similar pattern and should be ruled out.⁶⁰

Performing the Dix-Hallpike Diagnostic Maneuver. The Dix-Hallpike maneuver is performed by the clinician moving the patient through a set of specified head positions to elicit the expected characteristic nystagmus of posterior canal BPPV (**Figure 1**).^{45,46} Before beginning the maneuver, the patient should be counseled regarding the upcoming movements and that he or she may experience a sudden onset of intense subjective vertigo, possibly with nausea, which should subside within 60 seconds. Since the patient is going to be placed in the supine position relatively quickly with the head position slightly below the body, the patient should be oriented so that when placed supine, the head can “hang” with support off the posterior edge of the examination table by about 20°. The examiner should ensure that she or he can support the patient’s head and guide the patient through the maneuver safely and securely, without the examiner losing support or balance.

1. The maneuver begins with the patient in the upright seated position with the examiner standing at the patient’s side.⁴⁵ If present, the patient’s eyeglasses should be removed. We initially describe the maneuver to test the right ear as the source of the posterior canal BPPV.
2. The examiner rotates the patient’s head 45° to the right to align the posterior semicircular canal with the midsagittal plane of the body and, with manual support, maintains the 45° head turn to the right during the next part of the maneuver. The patient is instructed to keep the eyes open. Fairly quickly, the examiner moves the patient from the seated to the supine right-ear-down position and then extends the patient’s neck slightly (approximately 20° below the horizontal plane) so that the chin is pointed slightly

upward with the head hanging off the edge of the table (supported by the examiner). The examiner observes the patient’s eyes for the latency, duration, and direction of the nystagmus.^{10,61} Again, the provoked nystagmus in posterior canal BPPV is classically described as a mixed torsional and vertical movement with the upper pole of the eye beating toward the dependent ear (in this example, the right ear). The patient should also be queried about the presence of subjective vertigo.

3. After the resolution of the subjective vertigo and the nystagmus, if present, the patient may be slowly returned to the upright position. During the return to the upright position, a reversal of the nystagmus may be observed and should be allowed to resolve.
4. If the initial result for the right side is negative, the Dix-Hallpike maneuver (steps 1-4) should then be repeated for the left side, with the left ear arriving at the dependent position.⁵⁵ Again, the examiner should inquire about subjective vertigo and identify objective nystagmus, when present. This completes the Dix-Hallpike test.

The Dix-Hallpike maneuver is considered the gold standard test for the diagnosis of posterior canal BPPV.⁶² It is the most common diagnostic criterion required for entry into clinical trials and for inclusion of such trials in meta-analyses.^{63,64} The lack of an alternative external gold standard to the Dix-Hallpike maneuver limits the availability of rigorous sensitivity and specificity data. Although it is considered the gold standard test for posterior canal BPPV diagnosis, its accuracy may vary between specialty and nonspecialty clinicians. Lopez-Escamez et al reported a sensitivity of 82% and a specificity of 71% for the Dix-Hallpike maneuvers in posterior canal BPPV, primarily among specialty clinicians.⁶⁵ In the primary care setting, Hanley and O’Dowd have reported a positive predictive value for a positive Dix-Hallpike test of 83% and a negative predictive value of 52% for the diagnosis of BPPV.⁶⁶ Therefore, a negative Dix-Hallpike maneuver does not necessarily rule out a diagnosis of posterior canal BPPV. Because of the lower negative predictive values, it has been suggested that the Dix-Hallpike maneuver may need to be repeated at a separate visit to confirm the diagnosis and to avoid a false-negative result.^{55,67,68}

Factors that may affect the diagnostic accuracy of the Dix-Hallpike maneuver include the speed of head movements during the test, the time of day, and the angle of the occipital plane during the maneuver.⁵⁵ The Dix-Hallpike maneuver may in certain circumstances be performed bilaterally to determine which ear is (or ears are) involved, particularly if the diagnosis is not clear with the first performance of the maneuver.⁵⁵ In a small percentage of cases, the Dix-Hallpike maneuver may be bilaterally positive (ie, the correspondingly appropriate nystagmus is elicited for each ear in the dependent position). For example, bilateral posterior canal BPPV is more likely to be encountered after head trauma.³

While the Dix-Hallpike maneuver is the test of choice to confirm the diagnosis of posterior canal BPPV, it should be

avoided in certain circumstances. Although there are no documented reports of vertebrobasilar insufficiency provoked by performing the Dix-Hallpike maneuver, clinicians should be careful to consider the risk of stroke or vascular injury in patients with significant vascular disease.⁶⁹ Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity.^{47,69} Patients who are obese may be difficult for a single examiner to fully support the head through the maneuver, and additional assistance may be required. For patients with the above concerns or other physical limitations, special tilting examination tables may allow the safe performance of the Dix-Hallpike maneuver. Such patients may benefit from referral to more specialized clinicians and/or facilities with additional resources.

STATEMENT 1b. DIAGNOSIS OF LATERAL (HORIZONTAL) SEMICIRCULAR CANAL BPPV: If the patient has a history compatible with BPPV and the Dix-Hallpike test exhibits horizontal or no nystagmus, the clinician should perform, or refer to a clinician who can perform, a supine roll test to assess for lateral semicircular canal BPPV. *Recommendation based on diagnostic studies with limitations and a preponderance of benefit over harm.*

Action Statement Profile for Statement 1b

- Quality improvement opportunity: Improve accurate and efficient diagnosis of lateral canal BPPV (National Quality Strategy domains: promoting effective prevention/treatment, affordable quality care)
- Aggregate evidence quality: Grade B based on several RCTs with supine roll test as the reference entry standard
- Level of confidence in evidence: High
- Benefits: Avoid missed diagnoses of lateral canal BPPV; allows accurate diagnosis of lateral canal BPPV, thereby avoiding unnecessary diagnostic tests and inappropriate treatment; increased awareness of lateral canal BPPV
- Risks, harms, costs: Risk of provoking temporary symptoms of BPPV
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: Small
- Exceptions: Patients with physical limitations including cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and the morbidly obese
- Policy level: Recommendation
- Differences of opinion: None

Supporting Text. The purpose of this statement is to clarify the diagnosis of lateral semicircular canal BPPV, also called *horizontal semicircular canal BPPV*; to determine whether it is geotropic or apogeotropic type; and, when possible, to identify the affected side.

Incidence. Lateral semicircular canal BPPV is the second-most common type of BPPV.⁷⁰⁻⁷² Several studies have cited an incidence of approximately 5% to 22% in populations referred for evaluation and treatment of BPPV.^{10,11,58,73-76} The wide range of incidence of lateral semicircular canal BPPV reported in the literature is probably a function of how soon after the onset of vertigo the patient can be seen at each institution. Lateral semicircular canal BPPV tends to self-resolve more quickly than posterior semicircular canal BPPV,⁷⁰ so clinics seeing patients after more time has elapsed since symptom onset will likely see a lower percentage of the lateral semicircular canal form of BPPV cases and proportionally more posterior semicircular canal.

Lateral semicircular canal BPPV may occur following performance of the canalith repositioning procedure (CRP; eg, Epley maneuver) for an initial diagnosis of posterior semicircular canal BPPV. This transition from posterior semicircular canal BPPV to lateral semicircular canal BPPV is thought to occur as freely mobile calcium carbonate material originating from otoconia of the utricle moves from the posterior semicircular canal to the lateral semicircular canal (so-called canal conversion). Since this type of transition is possible but uncommon, clinicians should be aware of lateral semicircular canal BPPV and its diagnosis.¹⁰

Distinguishing Features. Lateral semicircular canal BPPV differs from the more common posterior semicircular canal BPPV in 2 important ways. First, the nystagmus elicited by the supine roll test in lateral semicircular canal BPPV is predominantly horizontal, whereas the nystagmus from the Dix-Hallpike test in posterior semicircular canal BPPV is upbeating and torsional. Second, the vertigo and nystagmus are evoked by turning the head side to side while supine (supine head roll test; **Figure 2**), whereas vertigo and nystagmus are induced by the Dix-Hallpike maneuver in the cases of posterior semicircular canal BPPV. Patients with a history compatible with BPPV (ie, repeated episodes of vertigo produced by changes in head position relative to gravity) who do not appear to have posterior semicircular canal BPPV by Dix-Hallpike positioning should be tested for lateral semicircular canal BPPV. The patient's presenting symptomatic report of positional dizziness due to lateral semicircular canal BPPV is often indistinguishable from posterior semicircular canal BPPV.^{71,77}

Supine Head Roll Test (Pagnini-Lempert or Pagnini-McClure Roll Test). The supine head roll test is the preferred maneuver to diagnose lateral semicircular canal BPPV.^{11,58,77,78} The supine roll test is performed by initially positioning the patient supine with the head in neutral position, followed by quickly rotating the head 90° to 1 side with the clinician observing the patient's eyes for nystagmus (**Figure 2**). After the nystagmus subsides (or if no nystagmus is elicited), the head is then returned to the straight faceup supine position. After any additional elicited nystagmus has subsided, the head is then

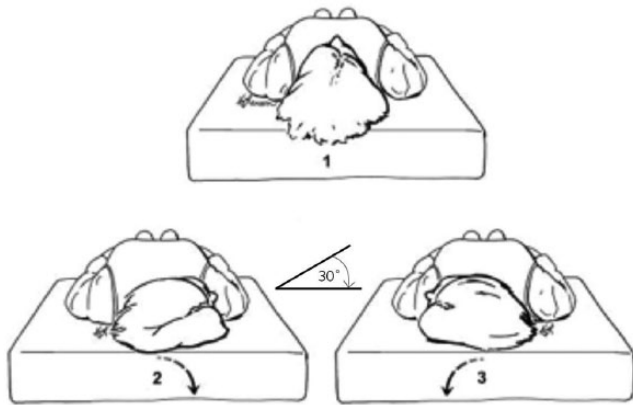


Figure 2. Diagrammatic views of the supine roll test. (1) The patient in the starting neutral position. The patient's head is turned rapidly to the right side (2) to examine for characteristic nystagmus. The head is returned to the faceup position (1), allowing all nystagmus to subside, and then turned rapidly to the left side (3) to examine once again for nystagmus. Adapted and reproduced with permission from Fife et al.⁶² © 2008 Barrow Neurological Institute, Phoenix, Arizona.

quickly turned 90° to the opposite side, and the eyes are once again observed for nystagmus.

Nystagmus Characteristics of Lateral Canal BPPV. Two potential nystagmus findings may occur with this maneuver, reflecting 2 types of lateral semicircular canal BPPV. Both types are so-called direction-changing positional nystagmus. That is, the direction of the positional nystagmus changes with changes in the head position.^{10,77-79}

Geotropic type: In most cases of lateral semicircular canal BPPV, when the patient is rolled to the pathologic (affected) side, there is a very intense horizontal nystagmus beating toward the undermost (affected) ear. The nystagmus beats toward the earth and is therefore geotropic nystagmus. When the patient is rolled to the healthy (nonaffected) side, there is a less intense horizontal nystagmus again beating toward the undermost ear (again geotropic but the direction of the nystagmus has now changed). It seems probable that when lateral canal BPPV exhibits this form of nystagmus, the calcium carbonate debris is located in the long arm of the semicircular canal.

Apogeotropic type: Less commonly, the roll test results in a horizontal nystagmus beating toward the uppermost ear (apogeotropic nystagmus). Upon rolling to the opposite side, the nystagmus will change direction, again beating toward the uppermost ear. It seems likely that when lateral semicircular canal BPPV exhibits the apogeotropic form of nystagmus, the calcium carbonate debris is located adherent to (cupulolithiasis) or close to the ampulla of the semicircular canal.^{58,80}

Identifying the Affected Side. Effective treatments for lateral semicircular canal BPPV are somewhat predicated on

knowing which side is affected, although it is recognized that determining the affected side can be complex and may require specialty referral after the initial diagnosis is made. **Table 7** outlines some of the methods for determining which side is affected in lateral canal BPPV. The supine roll test is the most commonly utilized method for determining the affected ear in therapeutic trials of lateral semicircular canal BPPV.^{71,75,81,82} Among the 2 types of lateral semicircular canal BPPV, the geotropic variant is the most common and the most amenable to treatment.^{58,71,78} Despite use of some of the methods described in **Table 7**, clear lateralization remains unclear in about 20% of cases.^{77,81,83} In such situations, one may simply treat one side and then the other. Alternatively, other testing methods, such as the bow and lean procedure (**Table 7**), may be applied to add to the diagnosis certainty of side of involvement.

Risk and Benefit Analysis. Reports of harm or patient injury from the performance of the supine roll test were not identified in the literature review, although many authors simply stated that patients who could not tolerate positional maneuvers were excluded. Care should also be exercised for patients with the same exclusionary criteria as for the Dix-Hallpike maneuver.^{47,69} The benefit of performing the supine roll test is that it allows clinicians to confirm a diagnosis of lateral semicircular canal BPPV quickly and efficiently.^{10,62} It also allows clinicians to more accurately and comprehensively diagnose positional vertigo that is not due to the posterior canal, whereas without supine roll testing, patients with lateral semicircular canal BPPV might be diagnostically missed if only traditional Dix-Hallpike testing were done. Further benefit may be realized if the supine roll test is done and the diagnosis recognized, obviating unnecessary or unhelpful diagnostic testing.

STATEMENT 2a. DIFFERENTIAL DIAGNOSIS: Clinicians should differentiate, or refer to a clinician who can differentiate, BPPV from other causes of imbalance, dizziness, and vertigo. *Recommendation based on observational studies and a preponderance of benefit over harm.*

Action Statement Profile for Statement 2a

- **Quality improvement opportunity:** Avoid incorrect diagnosis of BPPV (National Quality Strategy domain: promoting effective prevention/treatment)
- **Aggregate evidence quality:** Grade C based on observational studies with limitations
- **Level of confidence in evidence:** Medium
- **Benefits:** Prevent false-positive diagnosis of BPPV when another condition actually exists
- **Risks, harms, costs:** Health care costs of referral to another clinical
- **Benefits-harm assessment:** Preponderance of benefit over harm
- **Value judgments:** None
- **Intentional vagueness:** None
- **Role of patient preferences:** Small

Table 7. Selected Methods to Determine the Affected Ear in Lateral Canal BPPV.

Technique or Circumstance	Conclusion regarding the Affected Ear
Supine roll testing (Figure 2) reveals a direction-changing nystagmus that is either geotropic (beating toward the ground) or apogeotropic (beating away from the ground) and is distinctly stronger on one side than the other. ^{33,58,78,81}	<i>Geotropic form:</i> the side with the strongest nystagmus is the affected ear. <i>Apogeotropic form:</i> the side opposite the strongest nystagmus is the affected ear.
Posterior canal BPPV torsional upbeat nystagmus converts to strongly horizontal nystagmus (lateral canal BPPV) during positioning. ³³	Same ear as was affected by the posterior semicircular canal BPPV.
Patient is moved from sitting to straight supine facing up, which results in transient horizontal nystagmus (lying-down nystagmus). ^{83,194,198}	<i>Geotropic:</i> Nystagmus beats away from the affected ear. <i>Apogeotropic:</i> Nystagmus beats toward the affected ear.
In the straight supine position, the patient sits up, and the head bends down as a “head pitch test” (head-bending nystagmus). ^{83,194,198}	<i>Geotropic:</i> Nystagmus usually beats toward the affected ear. <i>Apogeotropic:</i> Nystagmus beats away from the affected ear. (Opposite of lying-down nystagmus.)
Bow and lean test ³ in which the direction of nystagmus is noted when the patient bends the head forward when facing down (bowing) and when facing upward (leaning). ^{290,291}	Geotropic <i>Bowing position (face down):</i> Nystagmus beats toward the affected ear. <i>Leaning position (face up):</i> Nystagmus beats away from the affected ear. Apogeotropic (reverse of geotropic type) <i>Bowing (face down):</i> Nystagmus beats away from the affected ear <i>Leaning (face up) nystagmus:</i> Nystagmus beats toward the affected ear.

Abbreviation: BPPV, benign paroxysmal positional vertigo.
^aThe supine head roll test will still be needed to determine if there is a pattern of geotropic or apogeotropic direction-changing nystagmus.

Table 8. Basic Differential Diagnosis of Benign Paroxysmal Positional Vertigo.

Otologic Disorders	Neurologic Disorders	Other Entities
Ménière’s disease	Vestibular migraine	Anxiety or panic disorder
Vestibular neuritis	Posterior circulation transient ischemic attack and stroke	Cervicogenic vertigo
Labyrinthitis	Demyelinating diseases	Medication side effects
Superior canal dehiscence syndrome	Central nervous system lesions	Postural hypotension
Posttraumatic vertigo	Vertebrobasilar insufficiency	Various medical conditions (eg, toxic, infectious, and metabolic conditions)
Perilymphatic fistula	Central positional vertigo	
Inner ear lesions		

- Exceptions: None
- Policy level: Recommendation
- Differences of opinion: None

Supporting Text. The purpose of this statement is to improve the diagnostic accuracy of BPPV by reducing misdiagnosis of other potential causes of dizziness.

Despite being the most common cause of peripheral vertigo,⁸⁴ BPPV is still often underdiagnosed or misdiagnosed.³⁴ Other causes of vertigo that may be confused with BPPV can be divided into otologic, neurologic, and other entities. Among patients presenting with dizziness, the frequency of various causes depends on the setting. In a German telephone survey of >1000 patients with dizziness, BPPV accounted for 8% of cases.²² In an analysis of nearly 10,000 US emergency department visits for dizziness, nearly half of patients had a medical diagnosis (nonvestibular and nonneurologic).⁸⁵ Only a third of patients were given a vestibular-related diagnosis. In an evaluation of patients presenting with vertigo in a British general practice setting, BPPV accounted for 42% of cases, followed by vestibular neuritis (41%), Ménière’s disease (10%), vascular causes (3%), and other causes (3%).⁶⁶ In

subspecialty populations, BPPV accounts for 20% to 53% of patients referred to ear, nose, and throat specialty clinics for dizziness.⁸⁶

The most common diagnoses that require distinction from BPPV are listed in **Table 8**. These conditions require distinction from BPPV as their natural history, treatment, and potential for serious medical sequelae are significantly different from BPPV. Patients with BPPV may not specifically describe true vertigo and may complain of light-headedness or nonspecific dizziness; thus, the clinician may need to initially consider a broader differential diagnosis.³² BPPV has been described as occurring in conjunction with, or as a consequence of, other vestibular disorders as well, such as Ménière’s disease and vestibular neuritis.⁸⁷ Therefore, clinicians must consider the possibility of >1 vestibular disorder being present in any patient who does not clearly have the specific symptoms of a single vestibular entity.

Recent studies emphasize that taking a history that focuses on the timing and triggers of a patient’s dizziness is more important than the specific descriptor that a patient uses.^{85,88,89} Timing (acute vs episodic vs chronic) and triggers (discrete

Table 9. Common Causes of Acute Dizziness: Differential Diagnosis by Timing and Triggers Category.

Acute Vestibular Syndrome ^a	Triggered Episodic Vestibular Syndrome ^b	Spontaneous Episodic Vestibular Syndrome ^c	Chronic Vestibular Syndrome ^d
Vestibular neuritis	Benign paroxysmal positional vertigo	Vestibular migraine	Anxiety or panic disorder
Labyrinthitis		Ménière's disease	Medication side effects
Posterior circulation stroke	Postural hypotension	Posterior circulation transient ischemic attack	Posttraumatic vertigo
Demyelinating diseases	Perilymph fistula	Medication side effects	Posterior fossa mass lesions
Posttraumatic vertigo	Superior canal dehiscence syndrome	Anxiety or panic disorder	Cervicogenic vertigo (variable)
	Vertebrobasilar insufficiency		
	Central paroxysmal positional vertigo		

^aAcute vestibular syndrome: acute persistent continuous dizziness lasting days to weeks and usually associated with nausea, vomiting, and intolerance to head motion.

^bTriggered episodic vestibular syndrome: episodic dizziness triggered by specific and obligate actions, usually a change in head or body position. Episodes generally last <1 minute.

^cSpontaneous episodic vestibular syndrome: episodic dizziness that is not triggered and that can last minutes to hours.

^dChronic vestibular syndrome: dizziness lasting weeks to months or longer.

trigger vs spontaneous) of the dizziness and its evolution over time define 4 distinct vestibular syndromes⁸⁹ (**Table 9**). These include an acute vestibular syndrome, triggered episodic vestibular syndrome, spontaneous episodic vestibular syndrome, and chronic vestibular syndrome. Each of these entities has its own differential diagnosis, with BPPV fitting the triggered episodic vestibular syndrome criteria given its positional trigger and brief episodic occurrences of vertigo.

Otologic Disorders. Whereas BPPV is characterized by acute, discrete episodes of brief positional vertigo without associated hearing loss, other otologic disorders causing vertigo may be differentiated by their clinical characteristics, including temporal pattern and the presence or absence of hearing loss.⁹⁰ Ménière's disease is characterized by discrete episodic attacks, each attack exhibiting a characteristic clinical constellation of sustained vertigo with fluctuating hearing loss, aural fullness, and tinnitus in the affected ear.⁵ As opposed to BPPV, the duration of vertigo in an episode of Ménière's disease typically lasts longer (usually on the order of hours), is typically more disabling due to both severity and duration, and is not triggered by any obligate head position changes. In addition, an associated contemporaneous decline in sensorineural hearing is required for the diagnosis of a Ménière's attack, whereas acute hearing loss should not occur with an episode of BPPV.⁹¹ Protracted nausea and vomiting are also more common during an attack of Ménière's disease. Ménière's disease would be categorized as an spontaneous episodic vestibular syndrome.

Acute peripheral vestibular dysfunction syndromes (characterized as an acute vestibular syndrome above), such as vestibular neuritis or labyrinthitis, present with sudden, unanticipated, severe vertigo with a subjective sensation of rotational (room spinning) motion. If the auditory portion of the inner ear is affected, hearing loss and tinnitus may also occur, and clinically this is consistent with labyrinthitis.⁹² These syndromes are commonly preceded by a viral prodrome. The time course of the

vertigo is often the best differentiator between BPPV and vestibular neuritis or labyrinthitis. In vestibular neuritis or labyrinthitis, the vertigo is of gradual onset, developing over several hours, followed by a sustained level of vertigo lasting days to weeks.^{90,93,94} The vertigo is present at rest (not requiring positional change for its onset), but it may be subjectively exacerbated by positional changes. These acute peripheral vestibular syndromes may also be accompanied by severe levels of nausea, vomiting, sweating, and pallor that are also typically sustained along with the vertigo. Although they are distinct entities, BPPV may be more common after acute vestibular syndrome.

Superior canal dehiscence syndrome (SCD) is clinically characterized by attacks of vertigo and oscillopsia (the sensation that viewed objects are moving or wavering back and forth) often brought on by loud sounds, Valsalva maneuvers, or pressure changes of the external auditory canals.⁹⁵ SCD differs from BPPV in that vertigo is induced by pressure changes and not position changes. SCD syndrome may also present with an associated conductive hearing loss attributable to lower bone-conducted thresholds for sound perception, when compared with air-conducted thresholds, and is diagnosed via computed tomography of the temporal bones or, alternatively, if available, vestibular evoked myogenic potential testing.⁹⁶ Given that SCD would be categorized as a spontaneous episodic vestibular syndrome, similar to BPPV, it should be differentiated from BPPV by its characteristic pressure-related trigger (eg, Valsalva). Similar to SCD, a perilymph fistula can produce episodes of vertigo and nystagmus triggered by pressure, thereby allowing differentiation from BPPV. Perilymph fistula can occur after surgery involving the middle ear or mastoid region or spontaneously and may be accompanied by a fluctuating hearing loss.

Posttraumatic vertigo can present with a variety of clinical manifestations, including vertigo, disequilibrium, tinnitus, and headache.^{97,98} These symptoms can be due to damage of the peripheral or central structures and are often complicated

by overlay of depression or anxiety. Post-head trauma vestibular migraine has also been described.⁷⁷ Although BPPV is most often idiopathic, in specific cases traumatic brain injury is associated with BPPV.⁹⁹

Neurologic Disorders. One of the key issues facing clinicians attempting to diagnose the etiology for vertigo is the differentiation between peripheral causes of vertigo (those causes arising from the ear or vestibular apparatus) and CNS causes of vertigo. Although at times this may be difficult, several clinical features may suggest a central cause of vertigo rather than BPPV.^{100,101} Nystagmus findings that more strongly suggest a neurologic cause for vertigo rather than a peripheral cause such as BPPV include downbeating nystagmus on the Dix-Hallpike maneuver (particularly without the torsional component and particularly if not modified or recovered by a positional maneuver), direction-changing nystagmus occurring without changes in head position (ie, periodic alternating nystagmus), gaze holding, direction-switching nystagmus (eg, beats to the right with right gaze and to the left with left gaze), or baseline nystagmus manifesting without provocative maneuvers (which also could be a manifestation of vestibular neuritis apart from a neurologic cause). Failure to respond to conservative management such as CRP or vestibular rehabilitation (VR) should raise concern that the underlying diagnosis may not be BPPV.¹⁰² Among the central causes of vertigo that should be distinguished from BPPV are vestibular migraine, brainstem and cerebellar stroke or transient ischemic attacks, and intracranial tumors or disorders, such as multiple sclerosis.

Vestibular migraine (or migraine-associated vertigo) is very common, with a lifetime prevalence of 3.2%,¹⁰³ and it may account for as many as 14% of cases of vertigo.⁹⁰ Diagnostic criteria include the following: (1) ≥ 5 episodes of vestibular symptoms lasting 5 minutes to 72 hours, (2) current or history of migraine according to International Headache Society Criteria, (3) ≥ 1 migraine symptoms during at least 50% of the dizzy episodes (migrainous headache, photophobia, phonophobia, visual or other aura), and (4) other causes ruled out by appropriate investigations.¹⁰⁴ It is distinguishable from BPPV by virtue of the diagnostic components enumerated above, which are not associated with classic BPPV. Furthermore, vestibular migraine would be characterized as a spontaneous episodic vestibular syndrome.

Brainstem stroke and cerebellar stroke are dangerous causes of vertigo.¹⁰⁵ In 1 series of 240 cerebellar strokes, 10% presented similar to a peripheral vestibular process.¹⁰⁶ The onset tends to be more sudden than with neuritis. Physical examination will often disclose other neurologic findings relating to the posterior circulation, such as dysarthria, dysmetria, dysphagia, or sensory or motor loss, or findings of a Horner's syndrome.¹⁰⁵

Another important cause of vertigo is posterior circulation transient ischemic attack.¹⁰⁷ A study of strokes (N = 1141 patients)—among which 24% were in posterior circulation—showed that patients with vertebrobasilar strokes had an odds ratio (OR) of 15 in terms of having had a posterior circulation

transient ischemic attack in the 90 days preceding the stroke.¹⁰⁸ Half of these attacks were isolated vertigo, and 8% of the patients with vertebrobasilar stroke had a transient ischemic attack of isolated vertigo. Because transient ischemic attacks generally last <1 hour, most patients are asymptomatic on presentation; however, if they were to have symptoms and signs on presentation, they would be the same as those associated with vertebrobasilar stroke.

Intracranial tumors and other brainstem lesions may rarely present with a history and symptomatology similar to those of BPPV.¹⁰² One uncommon but important example is central paroxysmal positional vertigo, due to structural lesions (tumors, strokes, and multiple sclerosis plaques) generally in the cerebellar vermis or region of the fourth ventricle, which can closely mimic BPPV.^{102,109} Multiple sclerosis patients are more often female and will nearly always have other worrisome findings, such as central nystagmus patterns, internuclear ophthalmoplegia, and other abnormalities that localize to the CNS.¹¹⁰ Importantly, among patients with known multiple sclerosis, BPPV was found to be a more common cause of acute dizziness than a multiple sclerosis flare.¹¹¹

Other Disorders. Several other nonotologic and nonneurologic disorders may present similarly to BPPV. Patients with panic or anxiety disorders may complain of symptoms of light-headedness and dizziness. Although these symptoms are usually attributed to hyperventilation, other studies have shown high prevalence of vestibular dysfunction for these patients.^{112,113} Several medications—such as Mysoline, carbamazepine, phenytoin, sedatives, and antihypertensive and cardiovascular medications—may produce side effects of dizziness and/or vertigo and should be considered in the differential diagnosis.

Cervical vertigo has been described as vertigo arising in conjunction with degenerative cervical spine disease.¹¹⁴ Cervical vertigo may produce similar symptoms to BPPV due to proprioceptive abnormalities arising from cervical spine dysfunction.¹¹⁵ Symptoms of cervical vertigo may be triggered by rotation of the head relative to the body while in an upright posture (as opposed to vertigo triggered by changes in head position relative to gravity). Orthostatic (postural) hypotension also may produce episodic dizziness or vertigo. The symptoms, however, are provoked by moving from the supine or sitting to the upright position in distinction to the provocative positional changes of BPPV.

Although the differential diagnosis of BPPV is vast, most of these other disorders can be further distinguished from BPPV based on the responses to the Dix-Hallpike maneuver and the supine roll test. Clinicians should still remain alert for concurrent diagnoses accompanying BPPV, especially in patients with a mixed clinical presentation.

STATEMENT 2b. MODIFYING FACTORS: Clinicians should assess patients with BPPV for factors that modify management, including impaired mobility or balance, CNS disorders, a lack of home support, and/or increased risk for falling. *Recommendation based on observational and*

cross-sectional studies and a preponderance of benefit over harm.

Action Statement Profile for Statement 2b

- Quality improvement opportunity: Decrease risks for complications from BPPV in at-risk populations (National Quality Strategy domains: safety, coordination of care)
- Aggregate evidence quality: Grade C based on observational and cross-sectional studies
- Level of confidence in evidence: Medium
- Benefits: Allow for management of patients with BPPV with an appropriately structured comprehensive treatment plan; identify patients at risk for falls and prevent fall-related injury
- Risks, harms, costs: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: Factors that modify management are intentionally vague, as all factors cannot be listed and individual clinical judgment is required.
- Role of patient preferences: Small
- Exceptions: None
- Policy level: Recommendation
- Differences of opinion: None

Supporting Text. The purpose of this statement is to consider factors that might modify treatment plans for the management of BPPV.

Although BPPV arises from dysfunction of the vestibular end organ, patients with BPPV often concurrently suffer from comorbidities, limitations, and risks that may affect the diagnosis and treatment. Careful assessment of the patient with BPPV for factors that modify management is essential for improved treatment outcomes and ensuring patient safety. The majority of factors that may modify management of BPPV can be identified if the clinician questions patients for these factors and elicits a detailed history,¹¹⁶ including the potential social and economic impact that this might have for the patient.

Given that BPPV occurs most commonly in the second half of the life span and its prevalence increases with age, patients suffering from BPPV often have medical comorbidities that may alter the management of BPPV.³² In cross-sectional surveys, patients with BPPV demonstrate higher rates of diabetes, anxiety, and history of head trauma.⁵¹ Other case-control studies have found higher relative rates of migraine (34% in BPPV patients vs 10% in nondizziness control group), history of stroke (10% for BPPV patients vs 1% for controls), diabetes (14% vs 5%), and hypertension (52% vs 22%).²² Clinicians should assess patients with BPPV for these comorbidities because their presence may modify management and influence treatment outcomes in BPPV.

One of the major concerns with BPPV and vertiginous conditions in general is the risk for falls and resultant injury.¹¹⁷⁻¹¹⁹

Data from the National Health and Nutrition Examination Survey demonstrated a 12-fold increase in the risk for falls among older individuals who were clinically symptomatic (reporting dizziness).¹¹⁸ Among community-dwelling adults aged >65 years, 1 in 3 falls each year.¹²⁰ This creates a tremendous individual and societal burden related to the health care costs of the associated injuries that occur from falling. It is estimated that the costs from falls in the United States exceed \$20 billion annually.¹²¹ In multiple studies concerning the etiology of falls, dizziness and vertigo were deemed the primary etiology 13% of the time, compared with existing balance and gait problems (17%) and person-environment interactions (31%).¹²² In a study by Oghalai et al, 9% of patients referred to a geriatric clinic for general geriatric evaluation had undiagnosed BPPV, and three-fourths of those with BPPV had fallen within the 3 months prior to referral.¹⁹ Thus, evaluation of patients with a diagnosis of BPPV should include an assessment of risk for falls.³² In particular, elderly patients will be more statistically at risk for falls with BPPV. An initial falls risk screening might start with questions such as those suggested by the Centers for Disease Control and Prevention in 2015: (1) Have you had a fall in the past year? How many times? Were you injured? (2) Do you feel unsteady when standing or walking? (3) Do you worry about falling? A positive response to questions such as these might then prompt the clinician to conduct a more detailed falls risk assessment or refer to a clinician who can use tools such as the Get Up and Go test,¹²³ Tinetti Balance Assessment,¹²⁴ Berg Balance Scale,¹²⁵ or others.

As noted above, comorbid conditions that occur commonly with BPPV, such as a history of stroke or diabetes, should also be identified when evaluating patients with BPPV. Patients with a history of stroke or a history of diabetes, particularly with peripheral neuropathy, may already have a preexisting gait, balance, or proprioceptive deficit.¹²⁶⁻¹²⁸ The additional symptoms of BPPV may increase their risk for fall and injury. Patients with visual disturbances often lack the ability to correct or compensate for a balance deficit with visual cues and may also be at increased risk for falls. Possible associations between osteoporosis (osteopenia) and BPPV have also been reported.¹²⁹ Patients with both conditions may be at greater risk for fractures resulting from falls related to BPPV; therefore, patients with combined osteoporosis and subsequent BPPV should be identified and monitored closely for fall and fracture risk. Examined from a different vantage point, patients with a history of recurrent falls, particularly among the elderly, should be assessed for underlying BPPV as 1 of the potential fall-precipitating diagnoses.¹³⁰

BPPV may occur simultaneously with other CNS disorders. Patients should be questioned about the presence of preexisting CNS disorders that may modify the management of BPPV. BPPV may occur relatively commonly after trauma or traumatic brain injury.^{98,131} Posttraumatic BPPV is most likely to involve the posterior semicircular canal, and studies indicate that posttraumatic BPPV is significantly more likely to require repeated CRP (up to 67% of cases) for resolution, as

compared with nontraumatic forms (14% of cases).^{132,133} Because posttraumatic BPPV may be more refractory and/or bilateral, thus requiring specialized treatment, a history of head trauma preceding a clinical diagnosis of BPPV should be elicited.^{131,134} Although dizziness in the setting of multiple sclerosis may have a variety of etiologies, studies of acute vertigo occurring in multiple sclerosis report that a substantial number of patients may have BPPV with a positive Dix-Hallpike maneuver and successful response to a CRP.^{111,135} These studies support that care should be taken to not miss a diagnosis of BPPV among patients with CNS disorders, as they may be successfully diagnosed and treated with CRP for BPPV.

Finally, in a small percentage of cases, refractory or persisting BPPV may create difficulties from a psychological and/or social-functional perspective for affected individuals.^{136,137} Outcomes studies have shown that patients with BPPV exhibit lower quality-of-life scores as compared with the normative population in multiple subscales of the Short Form–36 quality-of-life outcomes instrument.^{8,137} Patients who have preexisting comorbid conditions may require additional home supervision in the setting of BPPV.⁴⁷ This may include counseling about the risk of falling at home or a home safety assessment.

STATEMENT 3a. RADIOGRAPHIC TESTING: Clinicians should *not* obtain radiographic imaging in a patient who meets diagnostic criteria for BPPV in the absence of additional signs and/or symptoms inconsistent with BPPV that warrant imaging. *Recommendation against radiographic imaging based on diagnostic studies with limitations and a preponderance of benefit over harm.*

Action Statement Profile for Statement 3a

- **Quality improvement opportunity:** Reduce unnecessary testing and costs, reduce unnecessary radiation and radiographic contrast exposure (National Quality Strategy domains: safety, affordable quality care)
- **Aggregate evidence quality:** Grade C based on observational studies for radiographic imaging
- **Level of confidence in evidence:** Medium
- **Benefits:** Facilitate timely treatment by avoiding unnecessary testing associated with low-yield and potential false-positive diagnoses; avoid radiation exposure and adverse reactions to testing
- **Risks, harms, costs:** None
- **Benefits-harm assessment:** Preponderance of benefit over harm
- **Value judgments:** The panel placed heavy value in the accuracy of the BPPV diagnosis at the outset in that a diagnosis made by appropriate history and Dix-Hallpike is adequate to proceed with management without further testing.
- **Intentional vagueness:** None
- **Role of patient preferences:** None

- **Exceptions:** Patients who have separate indications for radiographic or vestibular testing aside from confirming a diagnosis of BPPV
- **Policy level:** Recommendation against
- **Differences of opinion:** None

Supporting Text. The purpose of this statement recommending against radiographic imaging is to optimize patient care, promote effective diagnosis and therapy, and reduce variations in care. The committee chose to focus on radiographic imaging in BPPV (as opposed to other diagnostic measures that can be employed), as the cost of diagnostic imaging can be significant, its use is common, and there is a body of literature available examining its use in BPPV from which to draw conclusions. The diagnosis of BPPV is based on the clinical history and physical examination. Routine radiographic imaging is unnecessary for patients who already meet clinical criteria for the diagnosis of BPPV (**Table 6**). Further radiographic imaging may have a role in diagnosis if the clinical presentation is felt to be atypical, if Dix-Hallpike testing elicits equivocal or unusual nystagmus findings, or if additional symptoms aside from those attributable to BPPV are present, suggesting an accompanying modifying CNS or otologic disorder.

Radiographic imaging, most commonly CNS imaging with magnetic resonance or computed tomographic techniques, is commonly obtained in the evaluation of a primary symptom complaint of vertigo. However, routine imaging is not useful in the diagnosis of BPPV, because there are no radiologic findings characteristic of or diagnostic for BPPV.^{138,139} This is likely due to fact that the pathology presumed to occur in BPPV within the semicircular canals occurs at a microscopic level that is beyond the resolution of current neuroimaging techniques.¹² On a broader scale, previous retrospective reviews of elderly patients with dizziness failed to detect any significant differences in cranial magnetic resonance imaging findings when comparing dizzy versus nondizzy patients.^{140,141} In a retrospective cohort study of 2374 patients, magnetic resonance imaging testing was not contributory to the clinical diagnosis of BPPV, and neuroimaging has been shown to be of little value.³⁵

Radiographic imaging of the CNS should be reserved for patients who present with a clinical history compatible with BPPV but who also demonstrate additional neurologic symptoms atypical for BPPV. Radiographic imaging may also be considered for patients with suspected BPPV but inconclusive positional testing or for patients with other neurologic signs on physical examination that are not typically associated with BPPV. Such symptoms include abnormal cranial nerve findings, visual disturbances, severe headache, among others. It should be noted that intracranial lesions causing vertigo are rare.⁴ Potential lesions causing vertigo identifiable on CNS imaging include cerebrovascular disease, demyelinating disease, or an intracranial mass, and these findings are most often located in the brainstem, cerebellum, thalamus, or cortex.⁴ In small case series, positional vertigo and nystagmus have been

associated with neurovascular compression of the eighth cranial nerve, vestibular schwannoma, Arnold Chiari malformation, and a variety of cerebellar disorders.¹⁴²⁻¹⁴⁴

In contrast to BPPV, such conditions are quite rare and typically present with additional neurologic symptoms in conjunction with the vertigo. Routine neuroimaging has not been recommended to discern these conditions from the more common causes of vertigo.¹⁴⁵ The costs of routine imaging in cases of BPPV are not justified, given that it does not improve diagnostic accuracy in the majority of BPPV cases. Therefore, neuroimaging should not be routinely used in the diagnosis of BPPV.

STATEMENT 3b. VESTIBULAR TESTING: Clinicians should not order vestibular testing in a patient who meets diagnostic criteria for BPPV in the absence of additional vestibular signs and/or symptoms inconsistent with BPPV that warrant testing. *Recommendation against vestibular testing based on diagnostic studies with limitations and a preponderance of benefit over harm.*

Action Statement Profile for Statement 3b

- Quality improvement opportunity: Reduce unnecessary testing and costs (National Quality Strategy domains: safety, affordable quality care)
- Aggregate evidence quality: Grade C based on diagnostic studies with limitations in referred patient populations and observational studies for vestibular testing
- Level of confidence in evidence: Medium
- Benefits: Facilitate timely treatment by avoiding unnecessary testing associated with low-yield and potential false-positive diagnoses; avoid patient discomfort from nausea and vomiting from vestibular testing; reduced costs from unnecessary testing
- Risks, harms, costs: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: None
- Exceptions: Patients who have separate indications for vestibular testing aside from confirming a diagnosis of BPPV
- Policy level: Recommendation against
- Differences of opinion: None

Supporting Text. The purpose of this statement is to emphasize that patients with a history and symptoms consistent with BPPV should not routinely undergo comprehensive vestibular testing unless there are other factors or concerns that would necessitate such testing.

Vestibular function testing involves a battery of specialized tests that primarily record nystagmus in response to labyrinthine stimulation and/or voluntary eye movements. The components of

the vestibular function test battery identify abnormalities in ocular motility, as well as deficits in labyrinthine response to position change, caloric stimulation, rotational movement, and static positions (sitting and supine). Caloric testing is an established, widely accepted technique that is particularly useful in determining unilateral vestibular hypofunction. Rotational chair testing is considered the most sensitive and reliable technique for quantifying the magnitude of bilateral peripheral vestibular hypofunction.¹⁴⁶ There are other tests that may be considered. Postural stability testing allows for assessment of the impact of vestibular dysfunction on balance. Vestibular evoked myogenic potentials (ocular and cervical) provide information about the utricle and saccule, respectively. Video head impulse testing allows for assessment of the function of each semicircular canal. Some or all of these test components may be included in a vestibular test battery. These tests are useful in the evaluation of vestibular disorders that may not be evident from the history and clinical examination, and they may provide information for quantification, prognostication, and treatment planning.¹⁴⁷

The diagnosis of BPPV is based on the clinical history and physical examination with a positive result on the Dix-Hallpike test. Fortunately, this can be accomplished by a trained clinician without specialized testing equipment, and an appropriate CRP can be implemented immediately. In a retrospective chart review of 100 consecutive patients referred for vestibular assessment, Phillips et al estimated that a 9% reduction in referrals for this specialized testing could be realized if the initial provider obtained a thorough case history and completed a Dix-Hallpike test.¹⁴⁸ Comprehensive vestibular testing is unnecessary for patients who already meet clinical criteria for the diagnosis of BPPV (**Table 6**). This does not imply that use of video-oculographic technology with or without recording should not be used when available to help in identification and differentiation of types of BPPV.

Comprehensive vestibular testing may have a role in diagnosis if the clinical presentation is felt to be atypical, if Dix-Hallpike testing elicits equivocal or unusual nystagmus findings, if the diagnosis is unclear, or if additional symptoms aside from those attributable to BPPV are present, suggesting an accompanying modifying CNS or otologic disorder. It may also be beneficial when multiple concurrent peripheral vestibular disorders are suspected.^{5,93,149}

In cases of BPPV where the nystagmus findings are suggestive but not clear, there may be benefit to using video-oculographic recordings of nystagmus associated with posterior canal BPPV, as the eye can be enlarged on a screen for detail and may be replayed for further study or second opinion. In a small percentage of cases, patients with a history of positional vertigo but unclear nystagmus findings may undergo vestibular function testing. Among complex patients referred for subspecialty evaluation of BPPV, such atypical or unclear nystagmus findings may approach 13% among patients with diagnoses suspicious for BPPV.¹⁵⁰

BPPV is relatively frequently associated with additional vestibular pathology. Symptoms associated with an underlying,

previously present chronic vestibular dysfunction may persist following appropriate treatment for BPPV, even if the treatment is effective in resolving the specific complaint of positional vertigo. For example, in highly selected subsets of patients referred for subspecialty evaluation of BPPV, additional otopathology and/or vestibulopathy has been identified in 31% to 53% of BPPV patients.^{5,151,152} Abnormalities of the cervical vestibular evoked myogenic potential have been reported in 25.8% to 34.8% of patients with BPPV.^{153,154} Lee et al found that 50% of patients with recurrent BPPV had abnormalities on either cervical or ocular vestibular evoked myogenic potential, which was significantly more than the 15% of patients with nonrecurrent BPPV.¹⁵⁵ These vestibular evoked myogenic potential abnormalities have been interpreted as being suggestive of more complicated otolith dysfunction in some patients with BPPV, and this negatively affects the quality of life for these patients.¹⁵⁶ These results have typically been measured for patients referred to specialty care centers, such as audiology, neurology, or otolaryngology, and may be higher than expected for patients seen by first-line, nonspecialty clinicians. Vestibular disorders that have been associated with BPPV include Ménière's disease, viral vestibular neuritis, and labyrinthitis.^{87,157} Vestibular function testing may be obtained when these additional diagnoses are suspected on the basis of signs or symptoms in addition to those of BPPV.

In patients with vestibular pathology in addition to BPPV, CRPs appear to be equally effective in resolving the positional nystagmus associated with BPPV, but complete symptom resolution is significantly less likely in this patient population. In 1 study, 86% of patients with BPPV without associated vestibular pathology reported complete resolution of symptoms after CRP versus only 37% reporting complete resolution when additional vestibular pathology was present.¹⁵⁸ Thus, patients with suspected associated vestibular pathology *in addition* to BPPV may be a subset who benefit from the additional information obtained from vestibular function testing. Similarly, 25% to 50% of patients with separate recurrences of BPPV are more likely to have associated vestibular pathology^{155,159}; therefore, patients with recurrent BPPV may be candidates for vestibular function testing, which could lead to additional targeted management.

In summary, patients with a clinical diagnosis of BPPV according to guideline criteria should not routinely undergo vestibular function testing, because the information provided from such testing adds little to the diagnostic accuracy or subsequent management in many cases. The Dix-Hallpike test and CRPs can be completed by most trained clinicians in a variety of health care settings without specialized equipment. This increases access to health care and decreases associated costs. Comprehensive vestibular function testing, or components thereof, is warranted in patients (1) exhibiting atypical nystagmus, (2) suspected of having additional vestibular pathology, (3) with a failed (or repeatedly failed) response to CRP or (4) with frequent recurrences of BPPV.

STATEMENT 4a. REPOSITIONING PROCEDURES AS INITIAL THERAPY: Clinicians should treat, or refer

to a clinician who can treat, patients with posterior canal BPPV with a CRP. *Strong recommendation based on systematic reviews of RCTs and a preponderance of benefit over harm.*

Action Statement Profile for Statement 4a

- **Quality improvement opportunity:** To promote effective treatment of posterior canal BPPV (National Quality Strategy domain: promoting effective prevention/treatments)
- **Aggregate evidence quality:** Grade A based on systematic reviews of RCTs
- **Level of confidence in evidence:** High for otolaryngology or subspecialty settings, lower in primary care settings where evidence is more limited
- **Benefits:** Prompt resolution of symptoms with a relatively low number needed to treat, ranging from 1 to 3 cases
- **Risks, harms, costs:** Transient provocation of symptoms of BPPV by the procedure; risk for falls due to imbalance after the procedure; no serious adverse events reported in RCTs
- **Benefits-harm assessment:** Preponderance of benefit over harm
- **Value judgments:** High value ascribed to prompt resolution of symptoms and the ease with which the CRP may be performed
- **Intentional vagueness:** None
- **Role of patient preferences:** Moderate
- **Exceptions:** Patients with physical limitations including cervical stenosis, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, retinal detachment, carotid stenosis, and spinal cord injuries may not be candidates for this procedure or may need specialized examination tables for performance of the procedure.
- **Policy level:** Strong recommendation
- **Differences of opinion:** None

Supporting Text. The purpose of this statement is to provide evidence for and promote the specific use of CRPs as the initial treatment to resolve symptoms and disability secondary to posterior and lateral canal BPPV. There is high-quality and compelling evidence that patients diagnosed with posterior and lateral semicircular canal BPPV should be offered expeditious treatment with CRP. These are specific and distinct from habituation/movement exercises, such as the Cawthorne-Cooksey exercises or Brandt-Daroff exercises. Treatment of BPPV with CRPs consistently eliminates the disabling vertigo and can also improve quality of life and reduce the risk of falling.

Posterior Canal BPPV Treatments. There are 2 distinct basic types of CRP for posterior canal BPPV: (1) the CRP (commonly referred to as the Epley maneuver) and (2) the liberatory maneuver (LM; commonly referred to as the Semont

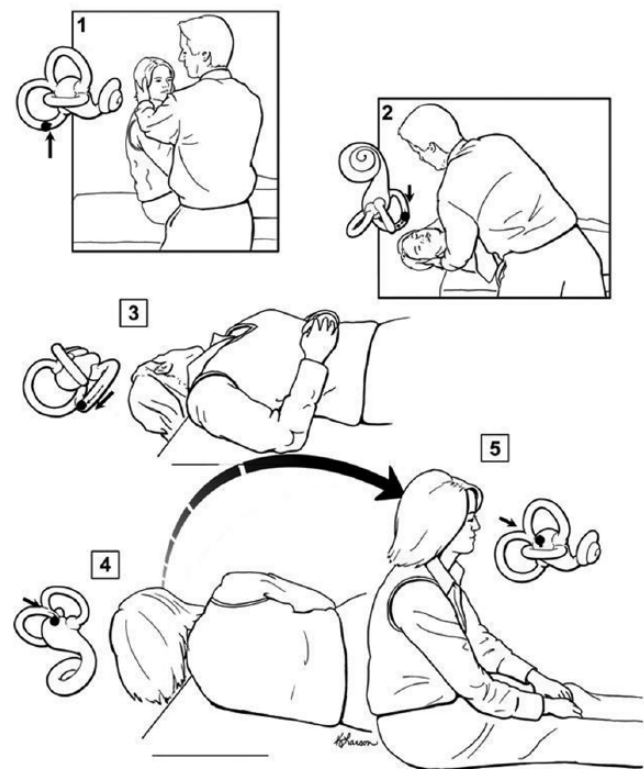


Figure 3. Depiction of the canalith repositioning maneuver (Epley maneuver) for right ear posterior semicircular canal benign paroxysmal positional vertigo. Adapted and reproduced with permission from Fife et al.⁶² © 2008 Barrow Neurological Institute, Phoenix, Arizona. Refer to **Table 10** for description.

maneuver). Where previous therapeutic exercises were based on habituation, these maneuvers work directly on freeing/liberating the adhered otoconia on the cupula (cupulolithiasis) and/or by moving free-floating otoconia (canalithiasis) out of the involved semicircular canal and back into the vestibule. There is significant evidence for the efficacy of both procedures for BPPV in the posterior semicircular canal and steadily advancing evidence for lateral semicircular canal.

Treatment with CRP, or “Epley Maneuver.” CRP was first described by Epley in 1992.¹⁶⁰ Patients are moved sequentially through a series of head position changes, designed to utilize gravity to move free-floating particles through the alignment of the posterior semicircular canal back into the vestibule, thereby relieving the pathologic stimulus that had been producing the vertigo in BPPV. **Figure 3** and **Table 10** illustrate the CRP for posterior semicircular canal BPPV. There are >20 years of evidence to support CRP for this indication, although many studies were nonrandomized case series.^{6,84,161-171} Most studies used symptom resolution as the primary outcome, but more recently, conversion to a negative provocative Dix-Hallpike procedure has been reported. A 2010 meta-analysis of CRP¹⁷² found that patients treated with CRP had a 6.5-times greater chance of improvement in clinical symptoms relative to controls (OR, 6.52; 95% CI,

Table 10. Stepwise Sequence for the Performance of the Canalith Repositioning Maneuver.^a

Step	Action
1	The patient is placed in the upright position with the head turned 45° toward the affected ear (the ear that was positive on the Dix-Hallpike testing).
2	The patient is rapidly laid back to the supine head-hanging 20° position, which is then maintained for 20-30 seconds.
3	Next, the head is turned 90° toward the other (unaffected) side and held for about 20 seconds.
4	Following this, the head is turned a further 90° (usually necessitating the patient’s body to also move from the supine position to the lateral decubitus position) such that the patient’s head is nearly in the facedown position. This is also held for 20-30 seconds.
5	The patient is then brought into the upright sitting position, completing the maneuver.

^aSee Figure 3.

4.17-10.20) and similar likelihood of negative Dix-Hallpike maneuver (OR, 5.19; 95% CI, 2.41-11.17).

The 2014 updated Cochrane review included 11 trials (745 patients) and reported that CRP is more effective when compared with sham maneuvers or controls. Complete resolution of vertigo occurred significantly more often in the CRP treatment group when compared with sham or control (OR, 4.42; 95% CI, 2.62-7.44).¹⁷³ Conversion from a positive to a negative Dix-Hallpike was more likely in the CRP treatment group than the sham or controls (OR, 9.62; 95% CI, 6.0-15.42). Importantly, a single CRP is >10 times more effective than a week of 3-times-daily Brandt-Daroff exercises (OR, 12.38; 95% CI, 4.32-35.47). The randomized prospective clinical trial specifically cited in the Cochrane review showed that by day 7, the Dix-Hallpike was negative in 80.5% of the CRP group versus 25% in the Brandt-Daroff group.¹⁷⁴ Differences between the groups remained statistically significant at 1 month. Bruintjes et al looked at CRP versus sham maneuver over a long term (12 months).¹⁷⁵ They found that both conversion to negative Dix-Hallpike (91% vs 46%; *P* = .001) and perceived disability (*P* = .003) as assessed by the Dizziness Handicap Inventory significantly favored CRP.

The CRP is most commonly performed in the outpatient setting by a clinician after the diagnosis of posterior semicircular canal BPPV has been confirmed.⁶² Patients should be informed that nausea, occasional vomiting, and/or a sense of falling may arise during the CRP.¹⁷⁶ Patients who previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuver may be offered antiemetic prophylaxis 30 to 60 minutes prior to CRP.

Treatment with the LM, or “Semont Maneuver.” The liberatory (Semont) maneuver, developed by Semont et al (illustrated in **Figure 4** and **Table 11**), utilizes both inertial and gravity forces to move patients briskly down into a side-lying position (involved side) and then through a rapid 180° arc to

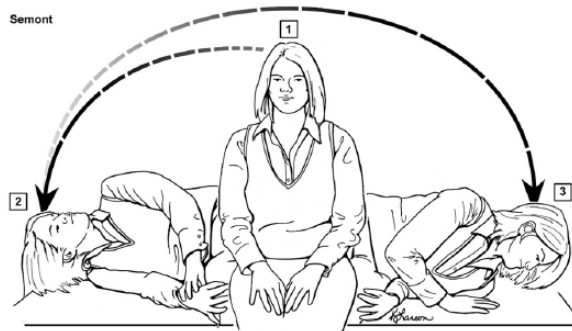


Figure 4. Semont liberatory maneuver for treatment of right posterior semicircular canal benign paroxysmal positional vertigo. Adapted and reproduced with permission from Fife et al.⁶² © 2008 Barrow Neurological Institute, Phoenix, Arizona. Refer to **Table 11** for description.

Table 11. Stepwise Description of the Performance of the Semont Liberatory Maneuver (Right Ear Affected).

Step	Action
1	Start with the patient sitting on a table or flat surface with the head turned away from the affected side.
2	Quickly put the patient into the side-lying position, toward the affected side, with the head turned up. Nystagmus will occur shortly after arriving at the side-lying position. Keep the patient in this position until at least 20 seconds after all nystagmus has ceased (some recommend up to 1-2 minutes).
3	Quickly move the patient back up and through the sitting position so that he or she is in the opposite side-lying position with the head facing down (head did not turn during the position change). Keep the patient in this position for about 30 seconds (some recommend 2-10 minutes).
4	At a normal or slow rate, bring the patient back up to the sitting position.

the uninvolved side.¹⁷⁷ As with all CRPs, the LM was designed to move the debris from the posterior semicircular canal back into the vestibule by principally breaking the canaliths free from adherence to the cupula (cupulolithiasis) and/or reposition free-floating canaliths (canalithiasis). Early studies examining the LM have demonstrated its effectiveness over sham treatments with initial success rates similar to CRP⁶⁴ and better than medication treatment¹⁷⁸ or Brandt-Daroff exercises.¹⁷⁹ A recent Cochrane review showed no difference when comparing the effectiveness of LM with CRP.¹⁷³ Chen et al demonstrated the short-term effectiveness of the LM in a double-blind randomized trial with conversion to a negative Dix-Hallpike on the fourth day in 85% of patients treated LM versus 14% in control group ($P = .001$).¹⁸⁰ Some authors advocate the LM over CRP in cases of resistant BPPV; however, research is lacking to demonstrate a benefit of LM in this subgroup.

Table 12 summarizes recent RCTs evaluating CRP for posterior semicircular canal BPPV. Of note, treatment effects between CRP and control patients tended to diminish over time. The majority of RCTs for CRP continue to take place in specialized or tertiary clinical settings, which may limit the generalizability of these results. For example, investigators were unable to demonstrate a significant benefit for the CRP based on symptomatic outcome in a primary care setting, although the conversion to a negative Dix-Hallpike at 1 week was more likely in the CRP group than among those treated with sham maneuvers.¹⁸¹ Since both the symptomatic response rates and the conversion rates to a negative Dix-Hallpike maneuver are lower than those reported in specialty-setting RCTs, further investigation into the effectiveness of the CRP in the primary care setting is warranted.

Considerable variability exists in terms of the number of times that the CRP is applied for the initial treatment of BPPV, even across RCTs.^{6,84,182} Some investigators perform only 1 CRP cycle at the initial treatment, whereas others repeat a fixed number of cycles or perform the CRP repeatedly until the vertiginous symptoms extinguish or the Dix-Hallpike converts to negative.⁶ Even further variability exists among published case series for CRP.¹⁸³⁻¹⁸⁵ A rapid systematic review in 2014 concluded that multiple studies with high relevance and moderate risk of bias show a benefit of multiple treatments with the CRP in patients with BPPV who are not fully cleared.¹⁸⁶ Specifically, in studies reviewed, 32% to 90% of patients cleared in the first treatment session, 40% to 100% after second treatment session, 67% to 98% after the third treatment session, 87% to 100% after the fourth treatment session, and 100% in studies in which patients received 5 treatment sessions. Based on a review of the literature, it was not possible to determine the optimal number of treatments with the CRP; however, there is a demonstrated beneficial effect of multiple treatment sessions for patients with persistent nystagmus following the initial maneuver.

With respect to complications of treatment, CRP is associated with mild and generally self-limiting adverse effects in about 12% of those treated.⁶² Some patients may experience an immediate falling sensation within 30 minutes after the maneuver and may benefit from counseling prior to the maneuver.¹⁷⁶ Serious complications from the CRP have not been identified in multiple RCTs. The most commonly encountered complications include nausea, vomiting, fainting, and conversion to lateral canal BPPV during the course of treatment (so-called canal switch or conversion). Canal conversion occurs in about 6% to 7% of those treated with CRP, underscoring the importance of recognizing the lateral canal variant of BPPV and the need for more unique and different CRPs.^{182,187} Another potential side effect after the CRP is postural instability that can last 24 hours with a tendency to fall backward or forward. Anecdotally, several investigators have suggested that the CRP should be applied cautiously in patients with cervical spine disease, certain vascular conditions, retinal detachment, and other contraindications to its performance.¹⁸⁸

Lateral (Horizontal) Semicircular Canal BPPV CRP Treatments. Evidence is mounting for the effectiveness of unique repositioning procedures based on semicircular canal involvement.

Table 12. Randomized Controlled Trials Evaluating the Effectiveness of Epley vs Control/Placebo or Epley vs Brandt-Daroff/Semont for Posterior Canal Benign Paroxysmal Positional Vertigo.^a

Reference	Time Point of Assessment	Improvement per Group, n (%)		End Point	P Value	Odds Ratio (95% CI)
		Treatment	Control			
Amor Dorado (2012) ¹⁷⁴	1 wk	33 of 41 (80) Epley	10 of 40 (25) BD	Negative Dix-Hallpike: Epley vs BD exercises	<.001	12.38 (4.34-35.47)
	1 mo	92.00 ^b	42.50 ^b	Negative Dix-Hallpike: Epley vs BD exercises	<.001	
Bruintjes (2014) ¹⁷⁵	12 mo	20 of 22 (91)	10 of 22 (45)	Negative Dix-Hallpike: Epley vs control or placebo	<.001	12.00 (2.24-64.28)
	1 mo	21 of 22 (96)	8 of 22 (36)	Negative Dix-Hallpike: Epley vs control or placebo	<.001	
Froehling (2000) ⁸⁴	1-2 wk	16 of 24 (67)	5 of 26 (19)	Negative Dix-Hallpike: Epley vs control or placebo	.020	3.20 (1.00-10.20)
Liang (2010) ²⁹²	7 d	42 of 43 (98)	34 of 44 (77)	Cured ^c : Epley vs control or placebo	<.05	12.35 (1.51-101.36)
Lynn (1995) ⁶	2 wk	16 of 18 (89)	4 of 15 (27)	Negative Dix-Hallpike: Epley vs control or placebo	<.033	22.00 (3.41-141.73)
Mazoor (2011) ²⁹³	1 wk	22 of 30 (73) Epley	21 of 30 (70) Semont	Negative Dix-Hallpike: Epley vs Semont	.08	1.18 (0.38-3.63)
	4 wk	28 of 30 (93) Epley	25 of 30 (83) Semont	Negative Dix-Hallpike: Epley vs Semont	.30	
Munoz (2007) ^{181,d}	Immediate	13 of 38 (34)	6 of 41 (14)	Negative Dix-Hallpike: Epley vs control or placebo	.04	3.03 (1.01-9.07)
von Brevern (2006) ²⁶⁸	24 h	28 of 35 (80)	3 of 31 (10)	Negative Dix-Hallpike: Epley vs control or placebo	<.001	37.33 (8.75-159.22)
Xie (2012) ^{294,d}	7 d	54 of 58 (93)	11 of 45 (24)	Cured ^c : Epley vs control or placebo	<.05	41.73 (12.29-141.65)
Yimtae (2003) ¹⁸²	1 wk	22 of 25 (88)	13 of 20 (65)	Negative Dix-Hallpike: Epley vs control or placebo	.005	3.95 (0.87-17.99)
	4 wk	16 of 25 (64)	7 of 20 (35)	Negative Dix-Hallpike: Epley vs control or placebo	.336	3.3 (1.0-11.3)

Abbreviations: BD, Brandt-Daroff; OR, odds ratio.

^aAll randomized controlled trials completed in secondary or tertiary care otolaryngology settings except where designated.^bRaw values not given in article.^cCured: outcomes reported as a composite measure of symptom resolution and Hallpike test result.^dPrimary care setting.

Although such evidence exists, the complexities associated with determining the affected side and subtype (geotropic vs apogeotropic) of the lateral canal BPPV may limit the ease of applicability of such procedures, since it is paramount to determine the sidedness prior to CRP treatment in lateral canal BPPV. Nonetheless, the panel felt that information on the use of these procedures would be valuable to include, as it anticipated increased knowledge of this type of BPPV over the next guideline update cycle. Given that any CRP for BPPV is a direct application of anatomy of the semicircular canal with respect to gravity, lateral semicircular canal BPPV is usually unresponsive to CRPs used for posterior semicircular canal BPPV but is being found responsive to other maneuvers intended to move the displaced otoconia in the unique plane of the lateral semicircular canal. Lateral semicircular canal BPPV exists in 2 forms: geotropic or apogeotropic. The best-researched and most clinically responsive form is the

geotropic form. CRP effectiveness^{187,189,190} specific to the lateral semicircular canal were initially described in 1996 with the first maneuver reported as a 270°-360° “barbeque roll” in the plane of the lateral semicircular canal (**Figure 5, Table 13**).^{169,185} A subsequent maneuver, termed the *Gufoni maneuver*, was developed by Gufoni in 1998 (original publication in English by Appiani and colleagues in 2001),¹⁹¹ which involves lying sideways onto the uninvolved side and then turning the head into the terminal nose down position (**Figures 6 and 7, Tables 14 and 15**). As with the CRP for the posterior semicircular canal, either maneuver may be performed in the outpatient setting after a diagnosis of lateral semicircular canal BPPV has been made with the supine roll test (**Figure 2**).

Several cohort studies and case series have reported response rates from 50% to 100% with use of the barbecue roll maneuver to treat lateral semicircular canal BPPV (geotropic

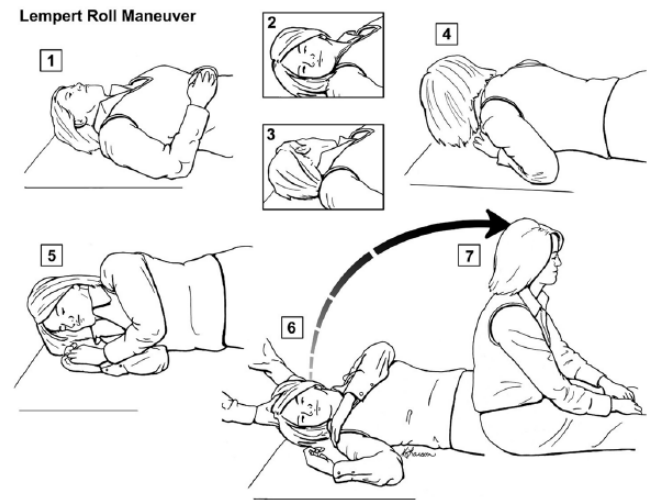


Figure 5. The Lempert 360° roll maneuver (or barbecue roll maneuver) for the treatment of right lateral semicircular canal benign paroxysmal positional vertigo—geotropic type. Adapted and reproduced with permission from Fife et al.⁶² © 2008 Barrow Neurological Institute, Phoenix, Arizona. Refer to **Table 13** for description.

Table 13. Stepwise Description of the Performance of the Lempert 360° Roll Maneuver (Barbecue Roll Maneuver) for the Treatment of Right Lateral Canal Benign Paroxysmal Positional Vertigo—Geotropic Type.

Step	Action ^a
1	Start from the supine position. OR
2	Some recommend rolling to start on the involved side.
3	Roll his or her head (or full body) to the unaffected side.
4	Keep rolling in the same direction until his or her head is completely nose down or prone. Some recommend ending the maneuver here and returning to sit (270° roll) as anatomically the debris is repositioned.
5-7	As originally published, however, complete the final roll (full 360°), and return to sitting.

^aEach position is held for 15-30 seconds or until nystagmus stops.

form).^{*} Lateral semicircular canal BPPV may spontaneously remit more quickly than other forms of BPPV.^{72,184} There have also been several recent randomized controlled studies on both forms of lateral semicircular canal BPPV.^{58,196-198} Casani et al demonstrated the effectiveness of these 2 types of CRPs in treating the geotropic form of lateral semicircular canal BPPV. They compared the results of the barbecue maneuver plus forced prolonged positioning (resting in bed for at least 12 hours with the head turned toward the unaffected ear) versus the Gufoni maneuver in a randomized prospective clinical trial with 81% success versus 93%, respectively, as determined by absence of vertigo and nystagmus on the supine roll test at follow-up examination.⁵⁸ A study by Kim et al in 2012 for geotropic lateral semicircular canal BPPV with 170

^{*}References 78, 79, 169, 185, 189, 190, 192-195.

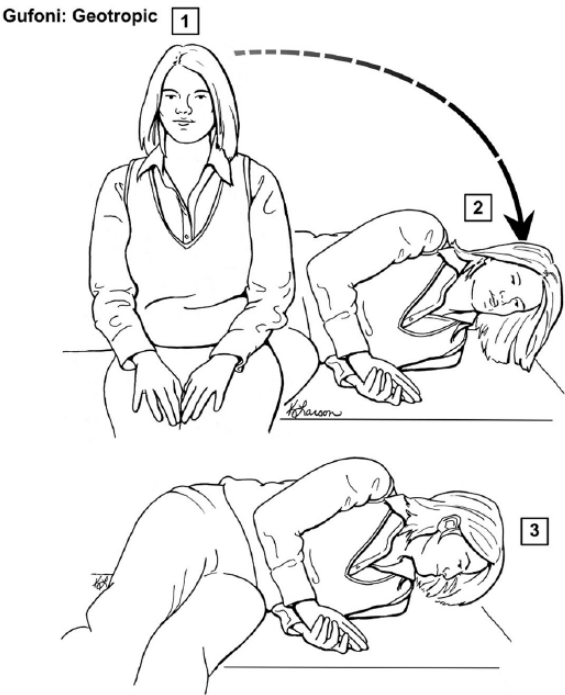


Figure 6. Gufoni maneuver for treatment of right-sided lateral semicircular canal benign paroxysmal positional vertigo—geotropic type. Adapted and reproduced with permission from Fife et al.⁶² © 2008 Barrow Neurological Institute, Phoenix, Arizona. Refer to **Table 14** for description.

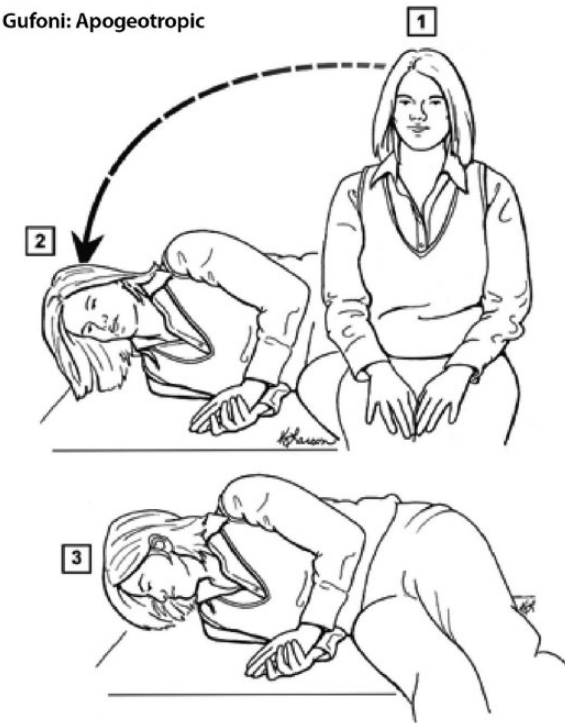


Figure 7. Gufoni maneuver for treatment of right-sided lateral semicircular canal benign paroxysmal positional vertigo—apogeotropic type. Adapted and reproduced with permission from Fife et al.⁶² © 2008 Barrow Neurological Institute, Phoenix, Arizona. Refer to **Table 15** for description.

Table 14. Gufoni Maneuver for Treatment of Right-Sided Lateral Semicircular Canal Benign Paroxysmal Positional Vertigo—Geotropic Type.

Step	Action
1	The patient is taken from the sitting position to the straight side-lying position on the unaffected side for about 30 seconds.
2	Then patient's head is quickly turned toward the ground 45°-60° and held in position for 1-2 minutes.
3	The patient then sits up again with the head held toward the left shoulder until fully upright and then may be straightened.

Table 15. Gufoni Maneuver for Treatment of Right-Sided Lateral Semicircular Canal Benign Paroxysmal Positional Vertigo—Apogeotropic Type.

Step	Action
1	The patient is taken from the sitting position to the straight side-lying position on the affected side (right side in this instance) for about 30 seconds.
2	From this point there are 2 variations of this maneuver that have been utilized, based on the possibility that debris can be on either the utricular OR the canal side of the cupula (or just lodged in the anterior arm of the lateral semicircular canal).
3	(Pictured in Figure 7) The patient's head is then quickly turned toward the ground 45°-60° and held in position for 1-2 minutes, which would free the debris from the utricular side of the cupula. The patient then sits up again with the head held toward the left shoulder until fully upright and then may be straightened. (Not pictured) In variation 2, move the patient's head, nose up, 45°-60° and hold in that position for 1-2 minutes, which would free the debris from the canal side of the cupula (or from being lodged in the anterior arm of the lateral semicircular canal).

consecutive patients in 10 nationwide dizziness clinics in Korea reported that after a maximum of 2 maneuvers on the initial visit day, both the barbeque roll and the Gufoni maneuver were better than sham maneuvers at both 1 hour and 1 month after treatment (69%, 61%, and only 35%, respectively).¹⁹⁶ In the Kim study for apogeotropic lateral semicircular canal BPPV, statistically significant results were also noted for specific CRPs (modified Gufoni and therapeutic head shaking) over sham maneuvers at 73%, 62%, and only 35%, respectively, for immediate and long-term outcomes.¹⁹⁸ A recent systematic review of the Gufoni maneuver for the treatment of geotropic form of lateral semicircular canal BPPV found that the Gufoni maneuver was easy to perform and more effective than a sham maneuver or vestibular suppressants.¹⁹⁷

Forced prolonged positioning, as mentioned in the previously discussed Casani study, is another treatment that has

been found to be effective for lateral semicircular canal BPPV. This involves lying for an entire night on the uninvolved side (for the geotropic form) or the involved side (for the apogeotropic form). It may be performed either alone or with other maneuvers.⁵⁸ The effectiveness based on case series ranged from 75% to 90%.^{192,195,199,200} Lesser-known maneuvers, such as the Vannucchi-Asprella LM, have also been reported as being effective in uncontrolled studies.^{194,199}

In summary, variations of the barbecue roll maneuver or Gufoni maneuver appear moderately effective for the geotropic form of lateral semicircular canal BPPV. Other methods are not supported by RCTs. For the apogeotropic form of lateral semicircular canal BPPV, there is only a single RCT providing insufficient evidence to recommend a preferred CRP.¹⁹⁶

Self-administered CRP. CRP (Epley) and the LM have both been modified for self-administration by patients for the treatment of BPPV.^{201,202} Self-administered CRP appears to be more effective (64% improved) than self-treatment with Brandt-Daroff exercises (23% improvement).²⁰¹ Another trial reported that self-administered CRP (Epley) resulted in 95% resolution of positional nystagmus 1 week after treatment, compared with 58% for patients self-administered LM (Semont) maneuver ($P < .001$).²⁰² No comparison studies have been published from which to make recommendations regarding self-treatment versus clinician-administered treatment of BPPV.

STATEMENT 4b. POSTPROCEDURAL RESTRICTIONS: Clinicians should not recommend postprocedural postural restrictions after CRP for posterior canal BPPV. *Strong recommendation against restrictions based on RCTs with minor limitations and a preponderance of benefit over harm.*

Action Statement Profile for Statement 4b

- Quality improvement opportunity: Avoidance of unnecessary interventions, engaging patients, decreasing use of ineffective treatments (National Quality Strategy domain: coordination of care)
- Aggregate evidence quality: Grade A
- Level of confidence in evidence: High
- Benefits: Faster return to normal lifestyle, reduced anxiety, less sleep or work interruption, reduced musculoskeletal discomfort, reduced cost (eg, of cervical collars)
- Risks, harms, costs: Potential risk for increased failure risk in a small subset of patients
- Benefits-harm assessment: Preponderance of benefit
- Value judgments: None
- Intentional vagueness: The generic term *restrictions* is used, but that can include sleeping upright, lying on the involved side, use of a cervical collar, or any type of restriction.
- Role of patient preferences: Small
- Exceptions: None
- Policy level: Strong recommendation against

- **Differences of opinion:** Several panel members had only medium confidence in the evidence.

Supporting Text. The purpose of this statement is to emphasize that clinicians should not routinely apply postural restrictions to patients following CRP for posterior semicircular canal BPPV.

As canalith repositioning maneuvers grew in acceptance as a favored treatment choice for BPPV, clinicians often advised patients regarding various postmaneuver restrictions. The rationale has been that mobile otoconial debris that returned to the vestibule during treatment may move back into the semicircular canal if patients do not carefully avoid certain movements and positions. The actual restrictions vary among clinicians and even among reports describing research in this area. Common restrictions include avoidance of the following: sleeping without elevation of the head, sleeping with the treated ear in a dependent position, vertical head movement, and so on. Soft cervical collars have been used to help remind patients to avoid certain head movements. Again, there is lack of clarity on exactly which positions and head movements should be avoided or for how long these limitations should be recommended. Some authors have reported that complications, including neck stiffness, are observed when patients are given these types of restrictions.²⁰³

Comparison of studies, in particular the treatment arms for RCTs, reveals similar response rates whether posttreatment postural or activity restrictions are observed.²⁰³⁻²⁰⁶ At least 9 investigations indicate no effect. Two investigations report statistically significant benefit from use of postmaneuver restrictions.^{11,207}

Devaiah and Andreoli conducted a meta-analysis based on data from 6 investigations with 523 patients meeting all inclusion criteria.²⁰⁸ With this analysis, they found no effect when outcomes from the 2 groups were compared (ie, restrictions vs no restrictions). The authors stated that their findings contradict recommendations that postmaneuver head restrictions are necessary to maintain the effectiveness of BPPV maneuvers. This finding contrasts with that from a more recent systematic review by Hunt et al, which identified 9 studies for further analysis of the effects of postural restrictions on BPPV treatment efficacy.²⁰⁹ They included data from 528 patients from the 9 trials. Their results indicated benefit from use of postural restrictions, which provided a statistically significant improvement in outcome when the pooled data were considered. Still, the authors noted a small effect size and stated that the statistically significant effect highlights only a small improvement in treatment efficacy. Since this report was published, there have been 2 additional investigations reporting no significant effect of postmaneuver restrictions on BPPV treatment outcome.^{206,210}

Overall, there is insufficient evidence to recommend postmaneuver restrictions for most patients with posterior semicircular canal BPPV who are treated with a CRP. The clinician must bear in mind that these published investigations specifically excluded patients with BPPV and concomitant vertiginous disorders, such as Ménière's disease, migraine, vestibular

neuritis, and so on. Patients with bilateral and/or multicanal involvement were also excluded. There is a small subset of patients with BPPV who will present with frequently recurring BPPV. That group was also not investigated in these reports. It is possible that some of these groups may benefit from postmaneuver restrictions, and this may be considered by the clinician in select cases.

STATEMENT 4c. OBSERVATION AS INITIAL THERAPY: Clinicians may offer observation with follow-up as initial management for patients with BPPV. *Option based on data from cohort and observational studies with heterogeneity and a relative balance of benefits and harms.*

Action Statement Profile for Statement 4c

- **Quality improvement opportunity:** Decreased costs due to less intervention and incorporating patient preferences (National Quality Strategy domains: engaging patients, affordable quality care)
- **Aggregate evidence quality:** Grade B based on control groups from RCTs and observational studies with heterogeneity in follow-up and outcomes measures
- **Level of confidence in evidence:** High
- **Benefits:** Symptom resolution in 15% to 85% at 1 month without intervention
- **Risks, harms, costs:** Prolonged symptoms compared with other interventions that may expose patients to increased risks for falls or lost days of work; indirect costs of delayed resolution compared with other measures
- **Benefits-harm assessment:** Relative balance of benefits and harms
- **Value judgments:** The panel felt strongly in favor of treatment with CRP rather than observation, particularly with respect to the value of an expedited time to symptom resolution. The panel felt that observation may not be suitable for older patients, patients with preexisting balance disorders, or individuals at high risks for falls.
- **Intentional vagueness:** Definition of follow-up is not explicitly specified.
- **Role of patient preferences:** Large
- **Exceptions:** None
- **Policy level:** Option
- **Differences of opinion:** Some panel members thought that this option was not the optimal choice for management, given the data for other interventions.

Supporting Text. The purpose of this statement is to provide evidence and rationale for the use of "observation" as a treatment option for patients with known BPPV, including the use of waiting times prior to CRP for acute episodes or recurrences of BPPV, especially when contraindications to treatments or a history of adverse consequences from prior treatments for BPPV is present or as per stated preferences by the patient. Delaying referrals for specialty evaluations and/or VR is also

included within the category of “observation,” until such time that it is mutually agreeable with all involved.

“Observation” may be defined as a “watchful waiting,” or not immediately utilizing specific therapeutic interventions for a given disease or medical condition. Observation is typically considered when the course of the disease or condition is self-limited and/or when it is likely to be benign, perhaps with limited sequelae as a result of no active intervention. In BPPV, observation implies that therapeutic interventions, such as VR and/or CRP, will also be withheld, thereby anticipating a natural and spontaneous improvement of the symptoms and severity of BPPV. With a course of observation, patients may still be instructed to avoid activities that may increase the risk of injury (eg, falls), either until symptoms resolve or until the patients are reassessed clinically for symptom resolution.

To consider observation as an option in the management of BPPV, the natural history of BPPV needs to be understood. BPPV is a common, often self-limiting condition, but it can be acute as a single episode, chronic, and/or persisting. Although BPPV can manifest along all ages of the life span, it is relatively rare in children, with a steady and dramatic increase after the age of 40 years. Prevalence in patients aged >60 years is 7 times greater than in those aged 18 to 39 years.²² The cumulative lifetime incidence of BPPV was almost 10% by age 80 years in 1 population-based survey from Germany, although the diagnoses were made by historical criteria alone, with no confirmation by the Dix-Hallpike maneuver.²² The natural history of BPPV is usually one of eventual resolution of symptoms in most patients. In several studies, the spontaneous rate of symptomatic resolution of BPPV ranged from 27% to 38%.¹⁷³ Similarly, review of a recent commentary in a Cochrane report stated that the “successful resolution of BPPV with no treatment except observation in 35%-50% of patients indicates the rate of spontaneous recovery as part of the natural history of this condition.”⁷⁷

Adverse effects associated with CRP may influence decisions to avoid or delay treatment for BPPV, in favor of observation. However, adverse effects from CRP are infrequently reported. There are usually no serious adverse effects of treatment reported, although the rates of nausea during the repositioning maneuver varied from 16.7% to 32%.¹⁷³ In addition, some patients were unable to tolerate CRP because of cervical spine problems, while others complained of headache or pain in the neck after treatments. Patients with any of the relative contraindications cited elsewhere in this report, including cervical spondylosis, known cervical disk disease, and/or unstable cardiac conditions, may be candidates for observation rather than active treatment.

There was no consensus present among the guideline panel members regarding the optimal duration of observation for patients with symptomatic BPPV. However, the panel strongly favored initial treatment with CRP, particularly in subsets of patients who either are at higher risk for falls or are reporting more disabling symptoms given the high success rates detailed in section 4a. For example, there is evidence for the elderly, the most common age group to experience BPPV, that BPPV has a significant impact on health-related quality of life that

improves with CRP and that unrecognized (or untreated) BPPV has significant associated morbidity (impaired capacity for activities of daily living or instrumental activities of daily living and fall prevalence; 78% vs 35%, $P = .026$; OR, 6.2; 95% CI, 1.2-31).^{19,136} Additionally, BPPV can be a triggering event for more chronic disabling dizziness in patients who are more distraught/anxious, for which timely treatment is indicated.²¹¹ Widespread adoption of CRP for treatment of BPPV has yet to be seen, despite CRP’s documented efficacy. Some authors are already citing the poor utilization of CRP as an indicator of suboptimal treatment quality patterns in primary care.⁸⁸ However, if cases of BPPV are not as severe among those patients seen in primary care settings, compared with those patients visiting subspecialty clinics or emergency departments (spectrum bias or selection bias), then observation may become a more suitable treatment option within primary care settings. Waiting for recurrence or persistence of what would be expected to be self-limited BPPV symptoms may be 1 possible option to make the routine use of CRP and VR services a more rational and cost-effective policy. More research is needed to resolve the influence of a potential spectrum bias and the possible impact upon clinical trials, especially in those that include observation as a viable option.

The natural history of lateral canal BPPV is less well defined than that of posterior canal BPPV. Some authors have commented that lateral canal BPPV may be prone to more rapid spontaneous resolution than posterior canal BPPV.^{72,184} One study of untreated patients ($n = 34$) with posterior canal BPPV determined a mean interval from onset of symptoms to spontaneous resolution to be about twice that of those patients with lateral canal BPPV (39 vs 16 days, respectively).⁷⁰ Although repositioning maneuvers have shown success in lateral canal BPPV, the available high-quality comparative data regarding treatment versus observation (eg, RCTs) are limited in this subtype of BPPV.¹⁸⁴ Thus, observation as a management strategy for patients with lateral canal BPPV remains a rational option. More research is needed for the interventional management of lateral canal BPPV.

In summary, observation is an option for the management of posterior canal semicircular canal BPPV and lateral semicircular canal BPPV in some patients. Observation offers the potential benefits of avoiding provocation of new symptoms and any discomfort associated with the repositioning maneuvers themselves or with VR. There may also be cost savings from decreased rates of referral for VR or CRP. Patients who elect observation should be informed about the possibility of longer duration of symptoms when compared with patients receiving active treatment maneuvers. There is also a potential for higher recurrence rates of another episode of BPPV with the observation option. Patient education materials may be offered to those electing the observation approach to BPPV.

STATEMENT 5. VESTIBULAR REHABILITATION:
The clinician may offer VR in the treatment of BPPV.
Option based on controlled observational studies and a balance of benefit and harm.

Action Statement Profile for Statement 5

- Quality improvement opportunity: Offer additional therapy for patients with additional impairments, who fail initial CRP attempts, who are not candidates for CRP, and/or who refuse CRP. Promoting effective therapy and increased patient safety (National Quality Strategy domains: safety, promoting effective prevention/treatment)
- Aggregate evidence quality: Grade B based on subset analysis of a systematic review and limited RCTs
- Level of confidence in evidence: Medium
- Benefits: Offer additional therapy for patients with additional impairments; prevention of falls, improved return of natural balance function
- Risks, harms, costs: No serious adverse events noted in published trials; transient provocation of BPPV symptoms during rehabilitation exercises; potential for delayed symptom resolution as compared with CRP as a sole intervention; need for repeated visits if done with clinician supervision; cost of therapy
- Benefits-harm assessment: Relative balance of benefits and harm
- Value judgments: The panel felt that VR, as defined in this guideline, may be better as an adjunctive therapy rather than a primary treatment modality. Subsets of patients with preexisting balance deficit, CNS disorders, or risk for falls may derive more benefit from VR than the patient with isolated BPPV.
- Intentional vagueness: Nonspecification of type of VR nor timing (initial vs adjunctive) of therapy
- Role of patient preferences: Large
- Exceptions: Patients with physical limitations such as cervical stenosis, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries
- Policy level: Option
- Differences of opinion: None

Supporting Text for Statement 5. The purpose of this statement is to define VR, clarify various components of VR, including the distinction between movement/habituation-based VR versus isolated CRP, and provide evidence for the most effective application of VR in patients with BPPV.

VR has been defined as physical maneuvers or exercise regimens to treat dizziness and balance disorders. VR has long been recognized as an effective method for managing peripheral vestibular deficits by promoting habituation, adaptation, central compensation mechanisms, and, more recently, mechanical repositioning.²¹²⁻²¹⁷ Thus, VR is not a single specific protocol, but it refers to a broad designation of therapies that include CRP itself, as well as habituation exercises, exercises for gaze stabilization, balance retraining and facilitation of sensory and motor integration, gait retraining, fall prevention, relaxation training, conditioning exercises, functional and occupational skills retraining, and patient and family

education.²¹⁷⁻²²¹ For the purpose of this key action statement, VR is being more narrowly defined as any additional therapy beyond isolated CRP for patients who fail initial CRP attempts, are not candidates for CRP, have additional impairments, and/or who refuse CRP.

Two movement/habituation-based VR treatment protocols with respect to BPPV deserve specific mention, as they are well defined in the literature and often adopted in clinical practice. These are the Cawthorne-Cooksey exercises and the Brandt-Daroff exercise. The Cawthorne and Cooksey exercises consist of a series of eye, head, and body movements in a hierarchy of increasing difficulty intended to provoke vestibular symptoms.²¹² Cawthorne-Cooksey-type exercises begin with simple head movement exercises performed in the sitting or supine position and progress to complex activities, including walking on slopes and steps with eyes open and closed and sports activities requiring eye-hand coordination. These exercises theoretically fatigue the vestibular response and force the CNS to compensate by habituation to the stimulus.²²² The Brandt and Daroff exercise was developed specifically for BPPV and involves a sequence of rapid lateral head/trunk tilts repeated serially to promote loosening and, ultimately, dispersion of debris toward the vestibule.^{223,224} In this exercise, the patient starts in a sitting position moving quickly to the right side-lying position with head rotated 45° facing upward. This position is maintained until the vertigo stops. The patient then moves rapidly to a left side-lying position with head rotated 45° facing upward.

Several studies have compared movement/habituation-based VR to CRP in the treatment of posterior canal BPPV. In an RCT of 124 patients randomized to CRP (Epley or modified LM), Brandt-Daroff exercises, vestibular habituation exercises, or sham, both habituation routines were more effective than sham.^{64,216} However, CRP was found to be more effective than both habituation routines.^{64,216} Soto Varela et al comparatively analyzed a total of 106 BPPV patients randomly assigned to receive Brandt-Daroff habituation exercises or 1 of 2 CRPs (LM or the Epley maneuver).¹⁷⁹ At the 1-week follow-up, patients treated with CRP (LM and Epley maneuvers) experienced resolution rates of 71% to 74%, compared with only 24% with the Brandt-Daroff exercise. More recently, Toledo et al found in 2000 that CRP (LM specifically) was superior to Cawthorne-Cooksey exercises both at 15 days and at 3 months.²²⁵ In the 2015 Cochrane review of VR for unilateral peripheral vestibular dysfunction, McDonnell and Hillier reported not only a significant effect of VR over control or no intervention (OR, 2.67; 95% CI, 1.85-3.86) but that CRP was found to be superior to movement/habituation-based VR (eg, Cawthorne-Cooksey, Brandt-Daroff) with an OR of 0.19 (95% CI, 0.07-0.49; OR for improvement with VR vs CRP).²¹⁷ Concluding statements from the Cochrane review support intuitive thought that the primary intervention for patients with BPPV should be maneuvers (CRP) that directly treat the condition (eg, mechanical repositioning) but that other aspects of movement/habituation-based VR may further aide and support long-term functional recovery.^{174,217}

Although there is evidence that movement/habituation VR should not be considered as a substitute for CRP in the initial treatment of BPPV, there is a role for VR as adjuvant therapy in the management of selected patients with BPPV. BPPV can result in significant residual complaints of more generalized dizziness (abnormal motion sensitivities not associated with provocation of nystagmus) and definable abnormal postural control with heightened fall risk even after CRP has successfully resolved paroxysmal positional nystagmus.^{226,227} There is a statistically significant increased risk for persistent postural abnormalities in the elderly in general where multifactorial comorbid impairments may be present.⁵³ An RCT found that individuals with BPPV who were treated with CRP and additional VR exercises (balance/habituation) had significantly improved measures of overall gait stability when compared with those who had received isolated CRP (Epley) for their BPPV.²²⁸ Additionally, this study documented that increased balance performance was achieved in patients only when additional movement/habituation-based VR was administered. BPPV has also been noted to trigger the development of more chronic disabling dizziness, which was originally described as phobic postural vertigo²²⁹ and, more recently, chronic subjective dizziness or persistent perceptual postural dizziness, for which VR appears to offer critical additional improvement.²³⁰ If balance and motion tolerance do not improve in a timely manner in patients treated successfully with CRP, then further clinical assessment and VR are often not only indicated but necessary to complete healing and optimal resolution of disability.

Historically, VR is offered as either a home exercise-based standardized progression or more specialized and individually tailored exercise, termed *customized VR*. Where home exercise-based VR programs (eg, Cawthorne-Cooksey exercises) are most often provided as a handout to a patient during initial consult with no anticipated follow-up and with limited education and instruction, customized VR is usually prescribed by a therapist who tailors the exercises according to patient-specific impairments/tolerance with the anticipation of follow-up to progress the routine. Evidence for the benefits of customized VR over home exercise-based VR have been shown in early studies.^{231,232} Although larger randomized controlled studies are needed, customized VR has the potential to improve outcomes of BPPV. When delivered by a VR specialist, customized VR can provide secondary assessment that can gather further diagnostic information and provide individualized modifications to the CRP (eg, more ideal positioning with use of a Trendelenburg table in patients with limited range of motion). In cases of resistive forms of BPPV or complicating comorbidities, customized VR can offer an exercise prescription that is more comprehensive—for example, combinations of liberatory, habituation, and more specific balance and gait-retraining techniques. Examples of comorbidities that can often require customization include cervical stenosis, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries. Additionally,

patients with BPPV but with other comorbid otologic or neurologic disorders may benefit from customized VR since they may have other vestibular, mechanical, or neurologic deficits that require more comprehensive and customized rehabilitation.

In summary, given the substantial evidence that movement/habituation-based VR is significantly less effective than CRP as an initial treatment for BPPV, VR should be considered an option in the treatment of BPPV rather than a recommended first-line treatment modality for BPPV. VR is, however, indicated for patients with BPPV who have persistent disability following CRP, refuse CRP, or are not candidates for CRP. VR is particularly indicated in patients with additional impairments where further therapy is needed to resolved more non-specific dizziness and those patients with heightened fall risk (eg, elderly).

STATEMENT 6. MEDICAL THERAPY: Clinicians should not routinely treat BPPV with vestibular suppressant medications such as antihistamines and/or benzodiazepines. *Recommendation against routine medication based on observational studies and a preponderance of benefit over harm.*

Action Statement Profile for Statement 6

- Quality improvement opportunity: Decreased use of unnecessary medications with potentially harmful side effects; reduced costs (National Quality Strategy domains: safety, promoting effective prevention/treatment, affordable quality care)
- Aggregate evidence quality: Grade C based on observational and cross-sectional studies.
- Level of confidence in evidence: Medium
- Benefits: Avoidance of adverse effects from, or medication interactions with, these medications; prevention of decreased diagnostic sensitivity from vestibular suppression during performance of the Dix-Hallpike maneuvers
- Risks, harms, costs: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: To avoid harm from ineffective treatments. The panel felt that data regarding harms and side effects from non-BPPV populations with vertigo would be applicable to the BPPV patient population.
- Intentional vagueness: The panel recognized that there most likely is a very small subgroup of patients with severe symptoms who may need vestibular suppression until more definitive treatment can be offered (eg, CRP) or immediately before and/or after treatment with CRP.
- Role of patient preferences: Small
- Exceptions: Severely symptomatic patients refusing other treatment options and patients requiring prophyllaxis for CRP

- Policy level: Recommendation against
- Differences of opinion: None

Supporting Text. The purpose of this statement is to dissuade the routine use of medication in the treatment of BPPV.

The symptoms of vertigo, due to many underlying etiologies, may commonly be treated with medications. Clinicians may prescribe pharmacologic management to (1) reduce the spinning sensations of vertigo specifically and/or (2) reduce the accompanying motion sickness symptoms—including a constellation of autonomic or vegetative symptoms such as nausea, vomiting, and diarrhea, which can accompany the vertigo. Such pharmacologic therapies for vertigo may be broadly termed *vestibular suppressant medications*.^{233,234}

Several categories of vestibular suppressant medications may be used to treat a variety of vestibular disorders in general. Among these, the most often considered are the benzodiazepine and antihistamine drug classes. Benzodiazepines, such as diazepam and clonazepam, have anxiolytic, sedative, muscle-relaxant, and anticonvulsant properties derived from potentiating the inhibitory effect of the gamma-amino butyric acid system. In prolonged dizziness, these medications can reduce the subjective sensation of spinning but also can interfere with central compensation in peripheral vestibular conditions. Antihistamines, however, appear to have a suppressive effect on the central emetic center to relieve the nausea and vomiting associated with motion sickness. Common examples of antihistamines used to treat symptoms of vertigo and/or associated motion sickness include meclizine and diphenhydramine. Other medications that are often used for motion sickness include promethazine, which is a phenothiazine with antihistamine properties, and ondansetron, which is a serotonin-5HT₃ antagonist. Last, anticholinergic medications, such as scopolamine block acetylcholine, a widespread CNS transmitter, can help with motion sickness by reducing neural mismatching.^{233,234}

Conversely, vestibular suppressant medications have the potential for significant harm. All of these medications may produce drowsiness, cognitive deficits, and interference with driving or operating machinery.²³⁵⁻²³⁹ Medications used for vestibular suppression, especially psychotropic medications such as benzodiazepines, are a significant independent risk factor for falls.²⁴⁰ The risk of falls increases in patients taking multiple medications and with the use of medications such as antidepressants.^{32,241} The potential for polypharmacy when adding vestibular suppressants further exposes the elderly to additional risk.²⁴² Educational programs to modify a practitioner's use of such medications can result in a reduction of falls.²⁴³

There are other potential harmful side effects of vestibular suppressants. Benzodiazepines and antihistamines interfere with central compensation for a vestibular injury.^{4,101} The use of vestibular suppressants may obscure the findings on the Dix-Hallpike maneuvers. In addition, there is evidence of additional potential harm from the antihistamine class of medications on cognitive functioning²³⁵ and on gastrointestinal motility, urinary retention, vision, and dry mouth in the elderly.²⁴⁴

There is no evidence in the literature to suggest that any of these vestibular suppressant medications are effective as a definitive, primary treatment for BPPV or effective as a substitute for repositioning maneuvers.^{135,233,245-247} Some studies show a resolution of BPPV over time with medications, but these studies follow patients for the period of time during which spontaneous resolution would typically occur.^{170,248-251} In 1 double-blind controlled trial comparing diazepam, lorazepam, and placebo, all groups showed a gradual decline in symptoms with no additional relief in the drug treatment arms.²⁵¹ A small study compared particle repositioning maneuvers to a medication-alone treatment arm and found that particle repositioning maneuvers had substantially higher treatment responses (78.6%-93.3% improvement) compared with medication alone (30.8% improvement) at 2-week follow-up.²⁵⁰ The data reinforced previous data that also indicated superiority of vestibular training for BPPV over medication use alone.²⁴⁷ Similar findings were noted when comparing canal repositioning maneuvers to betahistine where patients randomized to canal repositioning maneuvers had faster physical and mental recovery than their pharmacologic counterparts.²⁵² A more recent study showed that patients who underwent the Epley maneuver alone recovered faster than those who underwent the Epley maneuver and concurrently received a labyrinthine sedative.²⁵³ Also, the addition of an antihistamine to canal repositioning maneuvers demonstrated no change in the Dizziness Handicap Inventory score.¹⁴

However, more recent studies have shown that there may be some pharmacologic benefit in select patients. In 1 randomized study, the addition of a benzodiazepine to canal repositioning maneuvers significantly decreased the functional and emotional scores of the Dizziness Handicap Inventory but did not affect the physical score when compared with patients who were treated with canal repositioning maneuvers alone, thereby suggesting a role in treating psychological anxiety secondary to BPPV.²⁵⁴ In 1 trial, betahistine was shown to be effective in reducing symptoms in patients >50 years old with hypertension, with symptom onset <1 month, and with brief attacks <1 minute when used concurrently with canal repositioning maneuvers.²⁵⁵ A general lack of isolated benefit from vestibular suppressants and inferiority to particle repositioning maneuvers indicate that clinicians should not routinely substitute pharmacologic treatment of symptoms associated with BPPV in lieu of other, more effective treatment modalities. However, when used judiciously in conjunction with canal repositioning maneuvers, pharmacologic therapy may have a role.

In summary, vestibular suppressant medications are not routinely recommended for treatment of BPPV, other than for the short-term management of autonomic symptoms, such as nausea or vomiting, in a severely symptomatic patient. Examples of potential short-term uses include patients who are severely symptomatic yet refuse therapy or patients who become severely symptomatic after a CRP. Antiemetics may also be considered for prophylaxis for patients who have previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuvers and in whom a CRP is planned. If

prescribed for these very specific indications, clinicians should also provide counseling that the rates of cognitive dysfunction, falls, drug interactions, and machinery and driving accidents increase with use of vestibular suppressants.

STATEMENT 7a. OUTCOME ASSESSMENT: Clinicians should reassess patients within 1 month after an initial period of observation or treatment to document resolution or persistence of symptoms. *Recommendation based on observational outcomes studies and expert opinion and a preponderance of benefit over harm.*

Action Statement Profile for Statement 7a

- Quality improvement opportunity: Obtain outcomes data for treatment of BPPV; ability to assess treatment effectiveness (National Quality Strategy domains: safety, engaging patients, coordination of care, promoting effective prevention/treatment)
- Aggregate evidence quality: Grade C studies with known significant failure rates for an observation option and lower failure rates for CRP
- Level of confidence in evidence: Medium
- Benefits: Increased accuracy of BPPV diagnosis; identify patients initially treated with observation who have persistent symptoms and may benefit from CRP or VR to hasten symptom resolution
- Risks, harms, costs: Cost of reassessment
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Panel valued ensuring the accuracy of diagnosis that may be enhanced by follow-up and capturing patients who could benefit from treatment or retreatment to improve symptom resolution. Panel valued the potential importance of outcomes measures in the overall health care data environment.
- Intentional vagueness: The term *reassess* could represent various types of follow-up, including phone calls from office staff or other methods to document outcome.
- Role of patient preferences: Small
- Exceptions: None
- Policy level: Recommendation
- Differences of opinion: Some panel members felt that there is value in return visits to establish symptom resolution or to document objective improvement. Most other panel members felt that phone contact versus open-ended follow-up if symptoms persist or recur is sufficient.

Supporting Text. The purpose of this statement is to emphasize that clinicians should reassess patients within 1 month after an initial period of observation or treatment to document resolution or persistent symptoms.

Importance of Patient Reassessment. Patients with BPPV, regardless of the initial treatment option, will have variable responses to therapy.²⁵⁶ The response to therapy may depend

on several factors, including the accuracy of diagnosis, the duration of symptoms prior to the diagnosis, and patient compliance with the prescribed therapy.^{63,257} It is important to reassess patients because those who continue to have vestibular symptoms remain at risk for falls, have decreased quality of life, and other consequences of unresolved BPPV. Furthermore, patients with continued vestibular symptoms should be reassessed for an accurate diagnosis and evaluated for further treatment needs.

The most effective treatment for BPPV is CRP. Recent studies have shown that the majority of patients are adequately treated with 1 or 2 CRPs (79.4%-92.7%).^{175,258-260} However, 12.8% to 15.3% of patients will require a second CRP, and 5.1% will be classified as treatment failures after 2 CRPs.^{175,258-260}

If initial therapy fails, the patient should be reassessed for BPPV diagnosis accuracy. Symptoms of CNS disorders may mimic BPPV, and these conditions would not respond to BPPV treatments. In cohort studies, the rates of false-positive diagnosis for BPPV subsequently found to be CNS lesions *after failed treatment* with CRP range from 1.1% to 3%.^{257,261} Thus, persistence of symptoms after initial management requires clinicians to reassess and reevaluate patients for other etiologies of vertigo. Conversely, resolution of BPPV symptoms after BPPV-targeted initial therapy, such as CRP, would corroborate and provide further evidence about an accurate diagnosis.

Definition of Treatment Failure. To define a BPPV treatment failure, a failed outcome criterion as well as an appropriate time interval for reassessment needs to be defined. In clinical trials, successful BPPV treatment outcomes are traditionally defined as subjective symptom resolution and/or conversion to a negative Dix-Hallpike test.^{63,262,263}

Although conversion to a negative Dix-Hallpike test may have the advantage of being a more objective reassessment when compared with subjective symptom resolution, it also carries the disadvantage of requiring a repeat clinical visit, which is associated with direct and indirect costs. The alternative of a symptom-based reassessment allows practitioners to use clinical judgment regarding the most appropriate follow-up modality for individual patients, including telephone communication, electronic communication, or office-based reexamination. Symptom-based assessment of treatment resolution should be detailed enough to distinguish those patients whose symptoms have decreased or minimized because of positional avoidance (who may not be treatment successes) from those with true symptom resolution.²⁶² If the patient was initially diagnosed and treated in an acute care setting (eg, an emergency room or urgent care clinic), her or his primary care provider or specialist would be a suitable provider to reassess the patient.

Definition of Time Interval. There is no widely accepted time interval to assess patients for treatment failure. Therapeutic BPPV trials report follow-up assessments for treatment outcomes at 40 hours, 2 weeks, 1 month, and up to 6 months. However, the most common follow-up interval is within or at

1 month.^{63,262,263} Spontaneous symptom resolution at 1 month ranges from 20% to 80%.^{182,264-268} At the 1-month reassessment, patients should be evaluated for further interventional treatment for unresolved BPPV as well as reassessed for accurate diagnosis.^{182,264-268}

Of note, the panel was somewhat divided regarding the need for a method of assessment for treatment failure. The panel recognized that BPPV is often in and of itself a self-limiting condition and that CRP is a very effective maneuver for its treatment. Given that the majority of patients ultimately come to symptom resolution, the panel recognized that a requirement for reassessment would be tracking this majority of patients who do well. In contradistinction, however, the panel also felt that there was a need for documentation of symptom resolution to ensure an added layer of safety with respect to the accuracy of diagnosis of BPPV and to reduce the quality-of-life impact of unresolved BPPV, even though numerically this may be a small fraction of initial patients suffering from BPPV. This may be of greater importance as the management of BPPV may move to the primary care or emergency department setting rather than subspecialty settings. The panel also felt that assessment would allow for collection of longitudinal comparative effectiveness data in a real-world setting, which may be of future value from a research and health care quality perspective.

STATEMENT 7b. EVALUATION OF TREATMENT FAILURE: Clinicians should evaluate, or refer to a clinician who can evaluate, patients with persistent symptoms for unresolved BPPV and/or underlying peripheral vestibular or CNS disorders. *Recommendation based on observational studies of diagnostic outcomes in patients with BPPV and a preponderance of benefit over harm.*

Action Statement Profile for Statement 7b

- **Quality improvement opportunity:** Capture missed or erroneous diagnoses; offer retreatment to those patients with early recurrence of BPPV or failed initial CRP (National Quality Strategy domain: safety, promoting effective prevention/treatment)
- **Aggregate evidence quality:** Grade A for treatment of observation failure and Grade B for CRP failure based on RCT and systematic review examining treatment responses and failure rates
- **Level of confidence in evidence:** Medium
- **Benefits:** Expedite effective treatment of patients with persistent BPPV and associated comorbidities; decrease the potential for missed serious medical conditions that require a different treatment algorithm
- **Risks, harms, costs:** Costs of reevaluation and the additional testing incurred
- **Benefits-harm assessment:** Preponderance of benefit over harm
- **Value judgments:** Valued comprehensive treatment of not only BPPV but associated conditions that

affect balance and function. The panel also valued expeditiously treating cases of persistent BPPV following observation or VR with a CRP as more definitive therapy.

- **Intentional vagueness:** Characterization of persistent symptoms was intentionally vague to allow clinicians to determine the quality a degree of symptoms that should warrant further evaluation or retreatment.
- **Role of patient preferences:** Small
- **Exceptions:** None
- **Policy level:** Recommendation
- **Differences of opinion:** None

Supporting Text. The purpose of this statement recommending evaluation of patients with persistent symptoms after initial treatment of BPPV is to expeditiously identify treatment failures, promote the timely diagnosis and management of underlying peripheral or CNS disorders, and, by doing so, reduce the risk of secondary complications related to unresolved or unidentified disease.

Patients with persistent symptoms of vertigo, dizziness, or unsteadiness after initial therapy for BPPV are classified as treatment failures. Treatment failures require reevaluation for the following reasons: (1) persistent BPPV may be present and responsive to additional maneuvers; (2) coexisting vestibular conditions may be present that can be identified and treated; and (3) serious CNS disorders may simulate BPPV and need to be identified.^{45,269,270}

Persistent BPPV. Patients with BPPV who initially are treated with observation may fail to resolve spontaneously. Also, based on failure rates of VR or a single-session CRP ranging from 8% to 50%, a significant number of patients initially managed with VR or CRP will have persistent BPPV after initial therapy, also indicating a treatment failure.[†] As such, reevaluation of a treatment failure is advisable and should include obtaining a history of vertigo and determining if the vertigo is provoked by positional change relative to gravity (ie, lying down in bed, rolling over, bending down, or tilting the head back), which then suggests persistent BPPV. As with the original diagnostic criteria, the Dix-Hallpike test should be repeated to confirm the diagnosis of BPPV. If the Dix-Hallpike maneuver is still positive, repeat canalith repositioning maneuvers can then be performed as a preferred treatment. The rate of successful treatment of BPPV reaches 90% to 98% when additional repositioning maneuvers are subsequently performed.^{186,274,275} Therefore, the CRPs are the treatment of choice for initial BPPV treatment failures deemed to be due to persistent BPPV. For treatment failures refractory to multiple CRP, surgical plugging of the involved posterior semicircular canal or singular neurectomy has a >96% success rate; however, the quality of data supporting these interventions precludes the ability to make definitive recommendations for their utilization.⁶²

[†]References 45, 63, 64, 171-175, 216, 217, 271-273

A similar approach may be adopted for the reevaluation of persistent symptoms of vertigo after an initial diagnosis of lateral canal BPPV. The supine roll test should be repeated, and if characteristic nystagmus is elicited, a CRP appropriate for lateral canal BPPV may be repeated as well. There are limited data regarding the management of treatment failures after CRP for lateral canal BPPV, since this condition seems to respond more consistently to CRP and also has a higher spontaneous resolution rate.^{79,184,190,194,197} Some studies indicate cure rates of 86% to 100% with up to 4 CRP treatments in lateral canal BPPV.^{192,195} Further subanalysis suggests that the apogeotropic variant of lateral canal BPPV may be more refractory to therapy.^{169,192,197}

A small percentage of patients initially diagnosed and treated for lateral canal BPPV may experience a “canal conversion.” In these cases, initially lateral canal BPPV may transform into posterior canal BPPV in up to 6% of cases.^{78,79} Similarly, a small fraction of patients (also approximating 6%) initially presenting with posterior canal BPPV may, after treatment, transition to lateral canal BPPV.^{182,187} A small subset of patients who do not respond to treatment for posterior canal and/or lateral canal BPPV may suffer from anterior canal BPPV and may need to be evaluated accordingly.¹⁵ In addition, although rare, 2 semicircular canals may be simultaneously involved. The second canal’s involvement may become evident at the time of reassessment if 1 of the involved canals was appropriately treated.²⁶⁹ Finally, it is possible that initial treatment was not properly directed to the involved canal, thereby increasing the chance of persistent symptoms. Thus, reassessment of persistent positional vertigo in BPPV should include examination for involvement of semicircular canals other than that which was originally diagnosed.

Coexisting Vestibular System Dysfunction. A BPPV treatment failure may be subsequently found to be a case manifesting vertiginous symptoms that are provoked by head and body movements in general (ie, not primarily provoked by positional changes relative to gravity), unprovoked (ie, spontaneous) episodes of vertigo occurring while not moving, or, in fact, a constant unsteadiness. These specific findings should be identified by clinicians, as such findings suggest the presence of vestibular system dysfunction associated with, or in addition to, the initially treated BPPV.

In a study by Monobe et al, treatment failure of the CRP was most commonly seen in patients with BPPV secondary to head trauma or vestibular neuritis.²⁷⁶ Since vestibular neuritis and head trauma are both frequently associated with vestibular dysfunction, the cause of persistent symptoms following treatment of BPPV is likely related to widespread dysfunction within the vestibular system in this setting.²⁷⁷ Because BPPV is more common in patients with Ménière’s disease and migraine, vestibular system dysfunction associated with these disorders can lead to prolonged symptoms of BPPV, greater chance for recurrence BPPV, and increased risk for falls, particularly in older persons.^{132,151,157,278,279} In addition, BPPV not associated with other otologic or neurologic disease can still be associated with an underlying impaired vestibular function,

and affected individuals are more likely to have incomplete resolution of symptoms even if their Dix-Hallpike testing normalizes with CRP.¹⁵⁸ Finally, transient vestibular dysfunction can also occur following repositioning maneuvers. Evidence suggests that balance function continues to be affected between 1 and 3 months after repositioning maneuvers and that some patients may need additional balance therapy (ie, counseling, VR) to prevent falls and decrease their fear of falling after the vertigo from BPPV has resolved.^{53,227,280,281} Thus, reevaluation of BPPV treatment failures should include a search for these associated conditions.

When coexisting vestibular system dysfunction is suspected, additional testing should be considered. This may include audiometric testing to screen for Ménière’s disease and eighth nerve pathology, such as acoustic neuroma, vestibular function testing to detect central and peripheral vestibular dysfunction, and CNS imaging to detect CNS pathology. Such subsequent testing will need to be tailored to the clinical presentation, and clinicians should exercise their clinical judgment. VR has been shown to be an effective treatment for vestibular symptoms due to the potentially persistent vestibular dysfunction associated with BPPV and may reduce fall risk.¹⁶⁷

CNS Disorders Masquerading as BPPV. While vertigo of central origin is frequently associated with neurologic symptoms, such as gait, speech, and autonomic dysfunction, it is important to recognize that, rarely, CNS disorders can masquerade as BPPV.²⁸² Many of these are discussed in the section on differential diagnosis, but the relative likelihood of their diagnosis increases in the face of initial treatment failure. In 1 study, a CNS disorder explaining BPPV treatment failure was found in 3% of patients.²⁸³

Whenever the signs and symptoms of BPPV are atypical or refractory to treatment, additional history and physical examination should be obtained to address the possibility of undiagnosed CNS disease.²⁸⁴ Patients with symptoms consistent with those of BPPV who do not show improvement or resolution after undergoing the CRP, especially after 2 or 3 attempted maneuvers, or those who describe associated auditory or neurologic symptoms should be evaluated with a thorough neurologic examination, additional CNS testing, and/or magnetic resonance imaging of the brain and posterior fossa to identify possible intracranial pathologic conditions.^{102,285}

STATEMENT 8. EDUCATION: Clinicians should educate patients regarding the impact of BPPV on their safety, the potential for disease recurrence, and the importance of follow-up. *Recommendation based on observational studies of diagnostic outcomes and recurrence in patients with BPPV and a preponderance of benefit over harm.*

Action Statement Profile for Statement 8

- **Quality improvement opportunity:** Education allows patients to understand the implications of BPPV on quality of life and patient safety, especially falls (National Quality Strategy domains: safety, engaging patients, promoting effective prevention/treatment)

- Aggregate evidence quality: Grade C based on observational and cross-sectional studies of recurrence and fall risk
- Level of confidence in evidence: Medium
- Benefits: Increased awareness of fall risk potentially decreasing injuries related to falls; increased patient awareness of BPPV recurrence, which allows prompt intervention
- Risks, harms, costs: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: None
- Exceptions: None
- Policy level: Recommendation
- Differences of opinion: None

Supporting Text. The purpose of this statement is to discuss the importance of patient education with respect to the impact of BPPV on the daily lives of patients with this diagnosis and to emphasize the importance of education as part of the plan of care for clinicians managing these patients. BPPV has multiple treatment options, is not always cured with the first treatment, and can reoccur, so it becomes a safety issue especially with respect to an increased risk of falling. The socioeconomic impact of the patient's inability to meet family and work responsibilities can be an added burden. Patient education should include a discussion of factors that might predispose to BPPV, diagnosis and treatment options, and risk for recurrence. This information can be reassuring to patients and help with their understanding of appropriate diagnostic testing and management. Written handouts can provide this information (**Table 16**). Patients can also be directed to numerous support groups through social media or by searching www.vestibular.org.

One of the most important goals of education is an understanding of what BPPV is. The acute onset of vertiginous symptoms can mimic those of a stroke or other neurologic problems and is very frightening for patients and their families. A thorough neurologic examination and a simple Dix-Hallpike test can reliably identify BPPV, making medications and expensive radiologic testing unnecessary. Explaining this to patients will help to put them at ease regarding their diagnosis.

Although BPPV generally responds well to treatment, there is a significant rate of BPPV recurrence after initial resolution or clinical cure. Most trials of BPPV maintain limited follow-up, rarely beyond 3 months. In the few trials of BPPV with longer-term follow-up, the rate of recurrent BPPV (ie, BPPV symptoms manifesting again after a symptom-free period) is reported to be 5% to 13.5% at 6-month follow-up.^{50,188} At 1 year after treatment, the rate of recurrence has been reported at a slightly higher rate of 10% to 18%.^{185,286} The recurrence rate continues to increase over time and may be as high as 36%.¹⁷³ Patients with BPPV after trauma are likely to demonstrate an even higher recurrence rate of their BPPV.¹³²

Thus, clinicians should be aware of the recurrence risk of BPPV and should counsel patients accordingly. Counseling will likely have several benefits. These include earlier recognition by patients of recurrent BPPV, allowing earlier return for CRP or VR. Also, counseling regarding recurrence will offset the potential anxiety that patients may feel when BPPV recurs and allow them to make corresponding adjustments in their daily routine to minimize the impact of BPPV symptomatology.

As with any balance or vestibular disorder, patients with BPPV should be counseled that BPPV places them at greater risk for falls.²⁸⁷ This may be particularly applicable for patients with preexisting balance disorders or vestibular deficits and a separate onset of BPPV. The propensity for falling may actually be a significant motivating factor for patients to be referred for evaluation and management of BPPV.³² The risk of falls and the fear of falls are significant considerations in the management of the elderly who suffer from chronic dizziness.¹¹⁷ In study of 120 elderly patients with chronic vestibular disorders, 36.7% carried the diagnosis of BPPV. Fifty-three percent of subjects had fallen at least once in the past year, and 29.2% had recurrent falls.¹¹⁷ Other authors have confirmed a relatively high rate of BPPV and associated falling tendencies in the elderly.^{19,288}

Practically speaking, clinicians should counsel patients and their families regarding the risk of falls associated with BPPV. This is particularly important in the elderly and frail, who may be more susceptible to serious injury as a result of falling. Such counseling could include assessment of home safety, activity restrictions, and the need for home supervision until BPPV is resolved.¹²² Patients may be particularly vulnerable in the time interval between initial diagnosis of BPPV and definitive treatment when they are referred to another clinician for CRP or VR. Counseling should therefore occur at the time of initial diagnosis. The direct costs of such counseling are anticipated to be minimal and will enhance patient and public safety and avoid potential posttraumatic sequelae.

Finally, patients should be counseled regarding the importance of follow-up after the diagnosis of BPPV. Patients initially treated with observation should be counseled that if BPPV fails to resolve spontaneously, effective therapies such as the CRP may then be undertaken, particularly if an observation option is initially elected. Also, patients should be educated about atypical symptoms (subjective hearing loss, gait disturbance, nonpositional vertigo, nausea, vomiting, etc) whose occurrence or persistence after resolution of the primary symptoms of BPPV warrants further clinical evaluation.²⁶⁹ As noted, such symptoms, particularly when unmasked by the resolution of BPPV may indicate an underlying or concurrent vestibular or CNS disorder.

Implementation Considerations

The complete guideline is published as a supplement to *Otolaryngology–Head and Neck Surgery*, which will facilitate reference and distribution. An executive summary will be published highlighting key recommendations from the guideline to facilitate information dissemination. Portions of the

Table 16. Patient Information: Frequently Asked Questions.

Question	Answer
What is BPPV?	Benign paroxysmal position vertigo (BPPV) is the most common inner ear problem and cause of vertigo, or false sense of spinning. BPPV is a specific diagnosis, and each word describes the condition: Benign —it is not life-threatening, even though the symptoms can be very intense and upsetting Paroxysmal (par-ek-siz-muhl)—it comes in sudden, short spells Positional —certain head positions or movements can trigger a spell Vertigo —feeling like you are spinning or the world around you is spinning
What causes BPPV?	There are crystals of calcium carbonate that are a normal part of our inner ear and help us with our balance and body motion. These tiny rocklike crystals, or “otoconia” (oh-toe-cone-ee-uh), are settled in the center “pouch” of the inner ear. BPPV is caused by the crystals becoming “unglued” from their normal place. They begin to float around and/or get stuck on sensors in the wrong or canal part of the inner ear. The most intense part of your BPPV symptoms have to do with how long it takes the crystal/sensor to settle after you move or change your head or body position. As the crystals move and settle, your brain is getting powerful (false) messages that you are violently spinning when all you may have done is lay down or rolled over in bed.
What are the common symptoms, and how can BPPV affect me?	Everyone will experience BPPV differently, but there are common symptoms. The most common symptoms are distinct <i>triggered</i> spells of vertigo or spinning sensations. You may experience nausea (sometimes vomiting) and/or a severe sense of disorientation in space. You may also feel unstable or like you are losing your balance. These symptoms will be intense for seconds to minutes. You can have lasting feelings of dizziness and instability, though at a lesser level, once the episode has passed. In some people, especially seniors, BPPV can appear as an isolated sense of instability brought on by position change, such as sitting up, looking up, bending over, and reaching. BPPV does not cause constant severe dizziness that is not affected by position or movement. BPPV does not affect your hearing or cause you to faint. The natural course of BPPV is to become less severe over time. People will often report that their very first BPPV spinning episode was the worst and the following episodes were not as bad.
How common is BPPV?	BPPV is very common. It is more common in older people. Many of us will experience it at some time in our lives.
What caused my BPPV?	Most cases of BPPV happen for no reason. It can sometimes be associated with trauma, migraine, other inner ear problems, diabetes, osteoporosis, and lying in bed for long periods (preferred sleep side, surgical procedures, illness).
How is BPPV diagnosed?	Normal medical imaging, such as scans and x-rays, or medical laboratory testing cannot confirm BPPV. Your health care provider or examiner will complete simple bedside testing to help to confirm your diagnosis. The bedside testing requires the examiner to move your head into a position that makes the crystal move. The testing may include hanging your head a little off the edge of the bed or rolling your head left and right while lying in bed. The examiner will be watching you for a certain eye movement to confirm your diagnosis. The most common tests are called either the Dix-Hallpike test or supine roll test.
Can BPPV be treated?	Yes. Although medications are not used other than for relief of immediate distress, such as nausea, most BPPV cases can be corrected with bedside repositioning exercises that take only a few minutes to complete. They have high success rates (around 80%) with only 1-3 treatments. These maneuvers are designed to guide the crystals back to their original location in your inner ear. They can be done at the same time that the bedside testing for diagnosis is being performed. You might be sent to a health professional (medical provider, audiologist, or therapist) who can perform these maneuvers, especially if any of the following apply: <ul style="list-style-type: none"> • You have severe disabling symptoms. • You are a senior with history of past falls or fear of falling. • You have difficulty moving around, such as joint stiffness especially in your neck and back and/or weakness. You can also be taught to perform these maneuvers by yourself with supervision, which is called “self-repositioning.”
Is there any downside to BPPV repositioning treatments?	During the actual BPPV treatment, there can be some brief distress from vertigo, nausea, and feelings of disorientation like you usually have with your BPPV episodes. Following the treatment, some people report that their symptoms start to clear right away. Many times, others report that they have continuing motion sickness-type symptoms and mild instability. These symptoms can take a few hours or a few days to go away.
Can BPPV go away on its own?	There is evidence that if BPPV is left untreated, it can go away within weeks. However, remember that while the crystal is out of place, in addition to feeling sick and sensitive to motion, your unsteadiness can increase your risk for falling. You will need to take precautions not to fall. You are at a higher risk for injury if you are a senior or have another balance issue. Seniors are encouraged to seek professional help quickly to help to resolve symptoms.
How do I know that my BPPV has gone away?	The strong spinning sensations that have been triggered by position changes should be greatly reduced, if not completely gone.

(continued)

Table 16. (continued)

Question	Answer
How long will it take before I feel better?	You can still feel a little bit sensitive to movement even after successful treatments for BPPV. You can also feel unsteady at times. These mild symptoms can take a few days to a few weeks to slowly go away. You should follow up with your medical provider or therapist if your symptoms of dizziness or instability do not get better in a few days to a couple of weeks. Seniors with a history of falls or fear of falling may need further exercises or balance therapy to clear BPPV completely.
Is there anything that I should or should not do to help my BPPV?	Yes. Your balance will be “off,” so you will need to take precautions that you do not fall. You will feel more sensitive to movement until the BPPV has been successfully treated and healed. After your symptoms are slowly going away, it is important to return to normal activities that you can do safely. Exposure to motion and movement will help to speed your healing.
Can BPPV come back, and/or can I prevent it?	Unfortunately, BPPV is a condition that can sometimes return. Your risk for BPPV returning can shift from low risk (few experiences in your lifetime) to a higher risk, which is often caused by some other factor, such as trauma (physical injury), other inner ear or medical conditions, or aging. Medical research has not found any way to stop BPPV from coming back, but it can be treated with a high rate of success.
What happens if I still have symptoms following my initial treatments?	There are a number of reasons why your initial treatment could have failed: <ol style="list-style-type: none"> 1. It is not uncommon to need more than 1 repositioning session to get the crystals back in their proper place. You may only need a few more treatments. 2. There are a number of different forms or types of BPPV, which can require special treatment. The self-treatment is designed for the most common form of BPPV. There are a number of other treatments available that depend on the different types of BPPV. 3. BPPV can sometimes be in more than 1 canal and/or side at the same time. This would require multiple treatments to resolve. 4. If your initial tries at repositioning have failed, mainly if you have tried only self-repositioning, seek a health professional who specializes in BPPV. It can be difficult to complete correct positioning by yourself. A professional may be able to complete better positioning and/or use helpful equipment. 5. There can be some significant leftover dizziness even after the BPPV crystals have been correctly repositioned. This dizziness may require more time (few days to couple of weeks), or it may be appropriate for a different exercise/movement routine. It is very important to follow-up with your health care provider if you continue to have symptoms. You may be sent for further testing to confirm your diagnosis and/or discuss further treatment options.
Resources	Vestibular Disorders Association, 5018 NE 15th Ave, Portland, OR 97211; (800) 837-8428; INFO@vestibular.org.

guideline will be presented at various clinical meetings, including planned presentation in a miniseminar at the annual meeting of the AAO-HNSF. Existing brochures and publications by the AAO-HNSF will be updated to reflect the guideline recommendations. A visual depiction of the anticipated diagnostic and therapeutic treatment algorithm that arises from the current guideline's recommendations is presented in **Figure 8**. This treatment algorithm emphasizes the diagnosis and evidence-based treatment of BPPV with CRPs. Members of the panel will be representing the guideline at their specialty societies for possible presentation and endorsement.

Because the guideline presents recommendations for an office-based diagnosis of BPPV based on positional maneuvers, an anticipated barrier to implementation is clinician unfamiliarity with the Dix-Hallpike maneuver and with the supine roll test. In addition to the descriptive and diagrammatic representations of the diagnostic tests provided in the guideline, a video is available at <https://youtu.be/KLt2LtISPmQ>, illustrating performance of these maneuvers as well as representations of the expected diagnostic nystagmus findings, especially in the case of lateral canal BPPV. It will be important to incorporate guideline recommendations into the development of point-of-care decision support tools

to encourage point-of-service adherence to the guidelines and to facilitate rapid clinical decision making in a busy office environment.

Another barrier to implementation of this guideline is potential clinician or patient preference for the ordering of diagnostic tests to evaluate vertigo. Because the differential diagnosis of vertigo may be vast and at times complex, clinicians may feel obligated to order diagnostic testing such as CNS imaging or vestibular testing to rule out other causes of vertigo even when diagnostic criteria for BPPV are met. In addition, patients may expect imaging or additional testing based on the perception that such testing is required or a safer course of action in the routine management of vertigo. The guideline's current strong recommendation for CRP with its anticipated high, almost immediate symptom resolution rate is anticipated to decrease such expectations and tendencies. Informational pamphlets for patients regarding their diagnosis and expectations regarding the natural history of BPPV may ease this difficulty. Specialty clinicians may exhibit a tendency for ordering additional diagnostic testing due to a variety of factors. Clinician and patient education regarding outcomes expectations and counseling on proper follow-up may offset these issues.

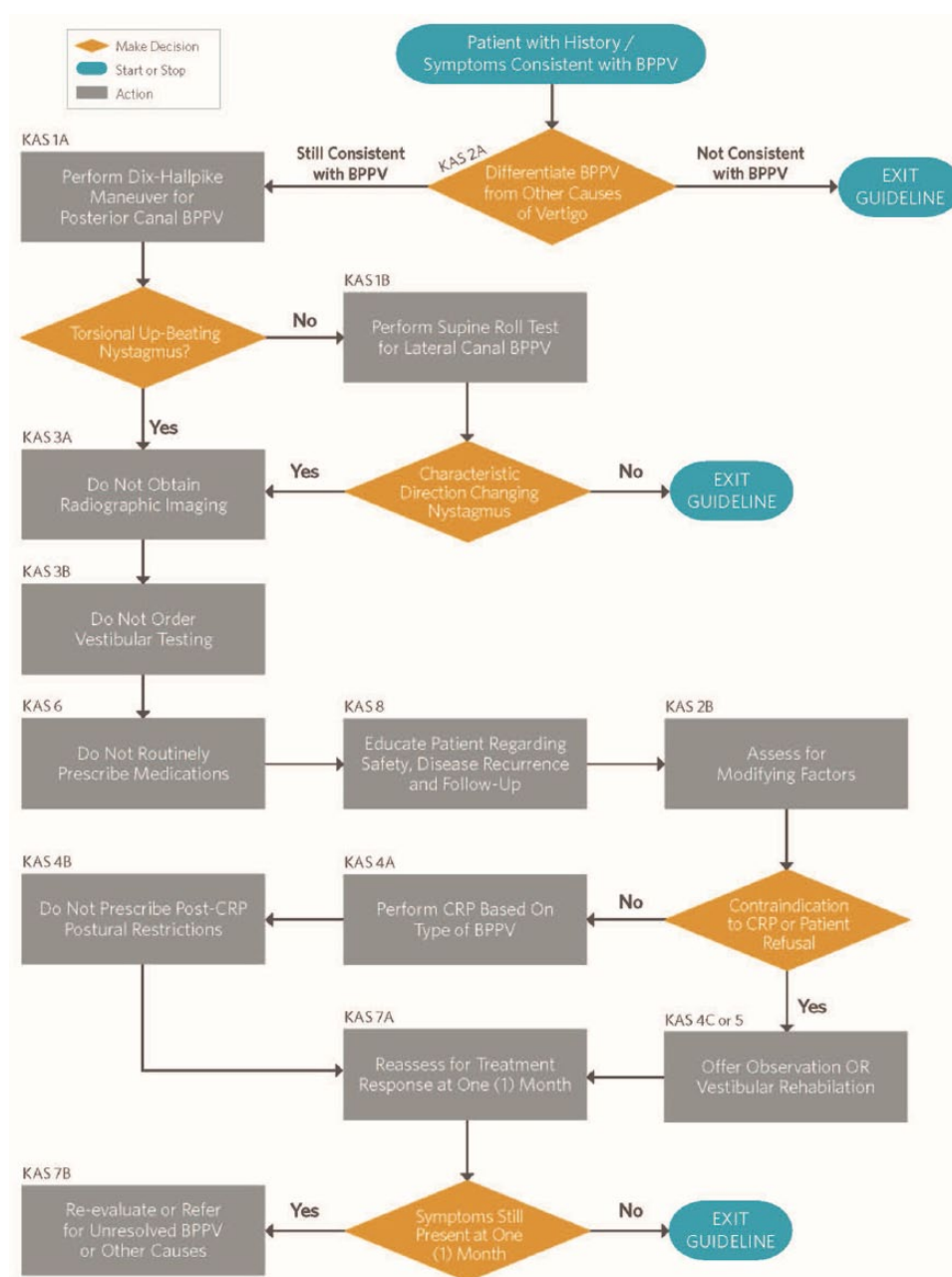


Figure 8. Algorithm showing the relationship of guideline key action statements. BPPV, benign paroxysmal positional vertigo; CRP, canalith repositioning procedure.

With respect to treatment with CRP, several barriers may still need to be overcome. First, many clinicians are likely to be unfamiliar with the CRP or other treatment maneuvers. In a busy clinical setting, diagnosing physicians may be unable or unwilling to take additional time to treat BPPV at the same office visit as diagnosis. In such cases, increasing familiarity with CRP or additional training of clinicians such as audiologists, physical therapists, and other providers may facilitate patients' access to CRP. Training courses on performance of the CRP offered at clinical education meetings will also help overcome this barrier.

Finally, patients may seek what are perceived to be simpler solutions such as medication therapy for BPPV. Given that medication therapy has not been shown effective in the treatment of BPPV, clinicians will need to educate patients that these medications offer more harm than benefit. Additional education of patients will be required in the form of handouts or brochures that inform patients of the risks associated with symptomatic BPPV, including risks for falls, recurrence of BPPV, and treatment options. Algorithms for fall assessment and home safety assessment will allow clinicians to stratify patients about these risks.¹²²

Research Needs

As determined by the panel's review of the literature, assessment of current clinical practices, and evidence gaps, research needs were determined as follows:

1. Conduct diagnostic and cost-effectiveness studies to identify which subsets of patients, based on specific history or physical examination findings, should be submitted for additional vestibular testing and/or radiographic imaging in the setting of presumed BPPV.
2. Diagnostic and cost-effectiveness studies evaluating the utility and costs of audiometry in the diagnostic evaluation of BPPV are needed.
3. Determine whether education and application of clinical diagnostic criteria for BPPV will change physician behavior in terms of anticipated decreases in ordering of diagnostic tests.
4. Determine the optimal number of CRPs and the time interval between performance of CRPs for patients with posterior canal BPPV.
5. Cost-effectiveness studies for the potential advantages of earlier intervention based on earlier diagnosis and earlier symptom resolution with expedient CRPs for BPPV are needed. Both direct health care and global economic costs require assessment.
6. Extended cohort studies with longer follow-up to determine if measures such as self-performance of CRP or longitudinal VR decrease recurrence rates for BPPV or complications from BPPV such as falls.
7. Determine whether vestibular therapy after the CRP offers additional benefits over CRP alone in select patient populations.
8. Studies on the functional impact of BPPV as they relate to home safety, work safety and absences, and driving risks.
9. Epidemiologic studies on the rates of falls with BPPV as an underlying cause/diagnosis.
10. Assess the impact of BPPV on quality of life for those affected with general quality-of-life and/or dizziness-specific quality-of-life metrics.
11. Develop and validate a disease-specific quality-of-life measure for BPPV to assess treatment outcomes.
12. Perform studies to evaluate the effect of structured versus "as needed" follow-up regimens on the outcomes of patients with BPPV.
13. Clarify and standardize the terms used to describe repositioning maneuvers for BPPV of the lateral canal to enable meaningful comparison of their efficacy.
14. Perform studies to evaluate the effectiveness of mastoid vibration in the treatment of BPPV.
15. Epidemiologic studies to characterize the relative risk of factors associated with the development of

BPPV, such as osteoporosis, dental procedures, and other devices that deliver cranial vibrations (massage devices, motorized toothbrushes, etc).

16. Identify patient- and treatment-related risk factors for the development of recalcitrant BPPV.
17. Perform studies to evaluate the sensitivity, specificity, and predictive values of the available examination maneuvers to determine the presence and laterality of BPPV affecting the anterior semicircular canal.
18. Perform studies to characterize the accuracy of diagnostic maneuvers for posterior and lateral canal BPPV and to evaluate the treatment outcomes for patients with BPPV seen in nonspecialty settings.

Disclaimer

The clinical practice guideline is provided for information and educational purposes only. It is not intended as a sole source of guidance in managing BPPV. Rather, it is designed to assist clinicians by providing an evidence-based framework for decision-making strategies. The guideline is not intended to replace clinical judgment or establish a protocol for all individuals with this condition and may not provide the only appropriate approach to diagnosing and managing this program of care. As medical knowledge expands and technology advances, clinical indicators and guidelines are promoted as conditional and provisional proposals of what is recommended under specific conditions but are not absolute. Guidelines are not mandates; these do not and should not purport to be a legal standard of care. The responsible provider, in light of all circumstances presented by the individual patient, must determine the appropriate treatment. Adherence to these guidelines will not ensure successful patient outcomes in every situation. The AAO-HNSF emphasizes that these clinical guidelines should not be deemed to include all proper treatment decisions or methods of care or to exclude other treatment decisions or methods of care reasonably directed to obtaining the same results.

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References

- Bhattacharyya N, Baugh RF, Orvidas L. Clinical practice guideline: benign paroxysmal positional vertigo. *Otol Head Neck Surg*. 2008;129:S47-S81.
- Schappert SM. National Ambulatory Medical Care Survey: 1989 summary. *Vital Health Stat* 13. 1992;(110):1-80.
- Katsarkas A. Benign paroxysmal positional vertigo (BPPV): idiopathic versus post-traumatic. *Acta Otolaryngol*. 1999;119:745-749.
- Hanley K, O'Dowd T, Considine N. A systematic review of vertigo in primary care. *Br J Gen Pract*. 2001;51:666-671.
- Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology*. 1987;37:371-378.
- Lynn S, Pool A, Rose D, Brey R, Suman V. Randomized trial of the canalith repositioning procedure. *Otolaryngol Head Neck Surg*. 1995;113:712-720.
- Burton MJ, Eby TL, Rosenfeld RM. Extracts from the Cochrane Library: modifications of the Epley (canalith repositioning) maneuver for posterior canal benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2012;147:407-411.
- Lopez-Escamez JA, Gamiz MJ, Fernandez-Perez A, et al. Long-term outcome and health-related quality of life in benign paroxysmal positional vertigo. *Eur Arch Otorhinolaryngol*. 2005;262:507-511.
- Roberts RA, Abrams H, Sembach MK, Lister JJ, Gans RE, Chisolm TH. Utility measures of health-related quality of life in patients treated for benign paroxysmal positional vertigo. *Ear Hear*. 2009;30:369-376.
- White JA, Coale KD, Catalano PJ, et al. Diagnosis and management of lateral semicircular canal benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2005;133:278-284.
- Cakir BO, Ercan I, Cakir ZA, et al. What is the true incidence of horizontal semicircular canal benign paroxysmal positional vertigo? *Otolaryngol Head Neck Surg*. 2006;134:451-454.
- Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). *CMAJ*. 2003;169:681-693.
- Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. *Laryngoscope*. 1992;102:988-992.
- Kim J-S, Zee DS. Clinical practice: benign paroxysmal positional vertigo. *N Engl J Med*. 2014;370:1138-1147.
- Jackson LE, Morgan B, Fletcher JC, et al. Anterior canal benign paroxysmal positional vertigo: an underappreciated entity. *Otol Neurotol*. 2007;28:218-222.
- Mizukoshi K, Kobayashi H, Ohashi N, et al. Quantitative analysis of the visual vestibulo-ocular reflex using sinusoidal rotation in patients with peripheral vestibular disorders. *Acta Otolaryngol Suppl*. 1984;406:178-181.
- Froehling DA, Silverstein MD, Mohr DN, et al. Benign positional vertigo: incidence and prognosis in a population-based study in Olmsted County, Minnesota. *Mayo Clin Proc*. 1991;66:596-601.
- Van der Zaag-Loonen HJ, van Leeuwen RB, Buntjes TD, van Munster BC. Prevalence of unrecognized benign paroxysmal positional vertigo in older patients. *Eur Arch Otorhinolaryngol*. 2015;272:1521-1524.
- Oghalai JS, Manolidis S, Barth JL, et al. Unrecognized benign paroxysmal positional vertigo in elderly patients. *Otolaryngol Head Neck Surg*. 2000;122:630-634.
- Kollén L, Frändin K, Möller M, Fagdevik Olsén M, Möller C. Benign paroxysmal positional vertigo is a common cause of dizziness and unsteadiness in a large population of 75-year-olds. *Aging Clin Exp Res*. 2012;24:317-323.
- Kerrigan MA, Costigan MF, Blatt KJ, Mathiason MA, Domroese ME. Prevalence of benign paroxysmal positional vertigo in the young adult population. *Phys Med Rehab*. 2013;5:778-785.
- von Brevern M, Radtke A, Lezius F, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry*. 2007;78:710-715.
- Neuhauser HK, Lempert T. Vertigo: epidemiologic aspects. *Semin Neurol*. 2009;29:473-481.
- Nedzelski JM, Barber HO, McIlmoyl L. Diagnoses in a dizziness unit. *J Otolaryngol*. 1986;15:101-104.
- Neuhauser HK. Epidemiology of vertigo. *Curr Opin Neurol*. 2007;20:40-46.
- Wang H, Yu D, Song N, Yin S. Delayed diagnosis and treatment of benign paroxysmal positional vertigo associated with current practice. *Eur Arch Otorhinolaryngol*. 2014;271:261-264.
- Li JC, Li CJ, Epley J, et al. Cost-effective management of benign positional vertigo using canalith repositioning. *Otolaryngol Head Neck Surg*. 2000;122:334-339.
- Benecke H, Agus S, Kuessner D, Goodall G, Strupp M. The burden and impact of vertigo: findings from the REVERT patient registry. *Front Neurol*. 2013;4:136.
- Ekvall Hansson E, Mansson NO, Hakansson A. Benign paroxysmal positional vertigo among elderly patients in primary health care. *Gerontology*. 2001;51:386-389.

30. Lin HW, Bhattacharyya N. Balance disorders in the elderly: epidemiology and functional impact. *Laryngoscope*. 2012;122:1858-1861.
31. Lin HW, Bhattacharyya N. Otologic diagnoses in the elderly: current utilization and predicted workload increase. *Laryngoscope*. 2011;121:1504-1507.
32. Lawson J, Johnson I, Bamio DE, et al. Benign paroxysmal positional vertigo: clinical characteristics of dizzy patients referred to a Falls and Syncope Unit. *QJM*. 2005;98:357-364.
33. Fife D, FitzGerald JE. Do patients with benign paroxysmal positional vertigo receive prompt treatment? Analysis of waiting times and human and financial costs associated with current practice. *Int J Audiol*. 2005;44:50-57.
34. von Brevern M, Lezius F, Tiel-Wilck K, et al. Benign paroxysmal positional vertigo: current status of medical management. *Otolaryngol Head Neck Surg*. 2004;130:381-382.
35. Grill E, Strupp M, Müller M, Klaus J. Health services utilization of patients with vertigo in primary care: a retrospective cohort study. *J Neurol*. 2014;261:1492-1498.
36. Rosenfeld RM, Shiffman RN, Robertson P. Clinical practice guideline development manual, third edition: a quality-driven approach for translating evidence into action. *Otolaryngol Head Neck Surg*. 2013;148(1):S1-S55.
37. Shiffman RN, Michel G, Rosenfeld RM, Davidson C. Building better guidelines with BRIDGE-Wiz: development and evaluation of a software assistant to promote clarity, transparency, and implementability. *J Amer Med Inform Assoc*. 2012;19:94-101.
38. Shiffman RN, Dixon J, Brandt C, et al. The guideline implementability appraisal (GLIA): development of an instrument to identify obstacles to guideline implementation. *BMC Med Inform Decis*. 2005;5:23.
39. Eddy DM. *A Manual for Assessing Health Practices and Designing Practice Policies: The Explicit Approach*. Philadelphia, PA: American College of Physicians; 1992.
40. American Academy of Pediatrics Steering Committee on Quality Improvement and Management. Classifying recommendations for clinical practice guidelines. *Pediatrics*. 2004;114:874-877.
41. Choudhry NK, Stelfox HT, Detsky AS. Relationships between authors of clinical practice guidelines and the pharmaceutical industry. *JAMA*. 2002;287:612-617.
42. Detsky AS. Sources of bias for authors of clinical practice guidelines. *CMAJ*. 2006;175:1033, 1035.
43. von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: diagnostic criteria. *J Vestib Res*. 2015;25:105-117.
44. Blakley BW, Goebel J. The meaning of the word “vertigo.” *Otolaryngol Head Neck Surg*. 2001;125:147-150.
45. Furman JM, Cass SP. Benign paroxysmal positional vertigo. *N Engl J Med*. 1999;341:1590-1596.
46. Dix MR, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Ann Otol Rhinol Laryngol*. 1952;61:987-1016.
47. Whitney SL, Marchetti GF, Morris LO. Usefulness of the Dizziness Handicap Inventory in the screening for benign paroxysmal positional vertigo. *Otol Neurotol*. 2005;26:1027-1033.
48. Ruckenstein MJ, Shepard NT. The canalith repositioning procedure with and without mastoid oscillation for the treatment of benign paroxysmal positional vertigo. *ORL J Otorhinolaryngol Relat Spec*. 2007;69:295-298.
49. Herdman SJ. Advances in the treatment of vestibular disorders. *Phys Ther*. 1997;77:602-618.
50. Macias JD, Lambert KM, Massingale S, et al. Variables affecting treatment in benign paroxysmal positional vertigo. *Laryngoscope*. 2000;110:1921-1924.
51. Cohen HS, Kimball KT, Stewart MG. Benign paroxysmal positional vertigo and comorbid conditions. *ORL J Otorhinolaryngol Relat Spec*. 2004;66:11-15.
52. Haynes DS, Resser JR, Labadie RF, et al. Treatment of benign positional vertigo using the semont maneuver: efficacy in patients presenting without nystagmus. *Laryngoscope*. 2002;112:796-801.
53. Blatt PJ, Georgakakis GA, Herdman SJ, et al. The effect of the canalith repositioning maneuver on resolving postural instability in patients with benign paroxysmal positional vertigo. *Am J Otol*. 2000;21:356-363.
54. Norre ME. Reliability of examination data in the diagnosis of benign paroxysmal positional vertigo. *Am J Otol*. 1995;16:806-810.
55. Nunez RA, Cass SP, Furman JM. Short- and long-term outcomes of canalith repositioning for benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2000;122:647-652.
56. Honrubia V, Baloh RW, Harris MR, et al. Paroxysmal positional vertigo syndrome. *Am J Otol*. 1999;20:465-470.
57. Heidenreich KD, Kerber KA, Carender WJ, et al. Persistent positional nystagmus: a case of superior semicircular canal benign paroxysmal positional vertigo? *Laryngoscope*. 2011;121:1818-1820.
58. Casani AP, Nacci A, Dallan I, et al. Horizontal semicircular canal benign paroxysmal positional vertigo: effectiveness of two different methods of treatment. *Audiol Neurotol*. 2011;16:175-184.
59. Lopez-Escamez JA, Molina MI, Gamiz MJ. Anterior semicircular canal benign paroxysmal positional vertigo and positional downbeating nystagmus. *Am J Otolaryngol*. 2006;27:173-178.
60. Fife TD. Benign paroxysmal positional vertigo. *Semin Neurol*. 2009;29:500-508.
61. Norre ME, Beckers A. Benign paroxysmal positional vertigo in the elderly: treatment by habituation exercises. *J Am Geriatr Soc*. 1988;36:425-429.
62. Fife TD, Iverson DJ, Lempert T, et al. Practice parameter: therapies for benign paroxysmal positional vertigo (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*. 2008;70:2067-2074.
63. Hilton M, Pinder D. The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. *Cochrane Database Syst Rev*. 2004;(2):CD003162.
64. Cohen HS, Kimball KT. Effectiveness of treatments for benign paroxysmal positional vertigo of the posterior canal. *Otol Neurotol*. 2005;26:1034-1040.

65. Lopez-Escamez JA, Lopez-Nevot A, Gamiz MJ, et al. Diagnosis of common causes of vertigo using a structured clinical history. *Acta Otorrinolaringol Esp*. 2000;51:25-30.
66. Hanley K, O'Dowd T. Symptoms of vertigo in general practice: a prospective study of diagnosis. *Br J Gen Pract*. 2002;52:809-812.
67. Viirre E, Purcell I, Baloh RW. The Dix-Hallpike test and the canalith repositioning maneuver. *Laryngoscope*. 2005;115:184-187.
68. Norre ME. Diagnostic problems in patients with benign paroxysmal positional vertigo. *Laryngoscope*. 1994;104:1385-1388.
69. Whitney SL, Morris LO, Calhoun KH, et al. Multisensory impairment in older adults: evaluation and intervention. In: Calhoun KH, Eibling DE, eds. *Geriatric Otolaryngology*. New York, NY: Taylor & Francis; 2006:115.
70. Imai T, Ito M, Takeda N, et al. Natural course of the remission of vertigo in patients with benign paroxysmal positional vertigo. *Neurology*. 2005;64:920-921.
71. Steenerson RL, Cronin GW, Marbach PM. Effectiveness of treatment techniques in 923 cases of benign paroxysmal positional vertigo. *Laryngoscope*. 2005;115:226-231.
72. Moon SY, Kim JS, Kim BK, et al. Clinical characteristics of benign paroxysmal positional vertigo in Korea: a multicenter study. *J Korean Med Sci*. 2006;21:539-543.
73. De La Meilleure G, Dehaene I, Depondt M, Damman W, Crevits L, Vanhooren G. Benign paroxysmal positional vertigo of the horizontal canal. *J Neurol Neurosurg Psychiatry*. 1996;60:68-71.
74. Hornibrook J. Horizontal canal benign positional vertigo. *Ann Otol Rhinol Laryngol*. 2004;113:721-725.
75. Han BI, Oh HJ, Kim JS. Nystagmus while recumbent in horizontal canal benign paroxysmal positional vertigo. *Neurology*. 2006;66:706-710.
76. Caruso G, Nuti D. Epidemiological data from 2270 PPV patients. *Audiological Med*. 2005;3:7-11.
77. Fife TD. Positional dizziness. *Continuum (Minneapolis, Minn)*. 2012;18(5, neuro-otology):1060-1085.
78. Nuti D, Agus G, Barbieri MT, et al. The management of horizontal-canal paroxysmal positional vertigo. *Acta Otolaryngol*. 1998;118:455-460.
79. Tirelli G, Russolo M. 360-Degree canalith repositioning procedure for the horizontal canal. *Otolaryngol Head Neck Surg*. 2004;131:740-746.
80. Baloh RW, Jacobson K, Honrubia V. Horizontal semicircular canal variant of benign positional vertigo. *Neurology*. 1993;43:2542-2549.
81. Lee S-H, Choi K-D, Jeong S-H, et al. Nystagmus during neck flexion in the pitch plane in benign paroxysmal positional vertigo involving the horizontal canal. *J Neurol Sci*. 2007;256:75-80.
82. Mandalà M, Pepponi E, Santoro GP, et al. Double-blind randomized trial on the efficacy of the Gufoni maneuver for treatment of lateral canal BPPV. *Laryngoscope*. 2013;123:1782-1786.
83. Hwang M, Kim S-H, Kang K-W, et al. Canalith repositioning in apogeotropic horizontal canal benign paroxysmal positional vertigo: do we need faster maneuvering? *J Neurol Sci*. 2015;358:183-187.
84. Froehling DA, Bowen JM, Mohr DN, et al. The canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo: a randomized controlled trial. *Mayo Clin Proc*. 2000;75:695-700.
85. Newman-Toker DE, Hsieh Y-H, Camargo CA, et al. Spectrum of dizziness visits to US emergency departments: cross-sectional analysis from a nationally representative sample. *Mayo Clin Proc*. 2008;83:765-775.
86. Lüscher, M, Theilgaard, S, Edholm, B. Prevalence and characteristics of diagnostic groups amongst 1034 patients seen in ENT practices for dizziness. *J Laryngol Otol*. 2014;128:128-133.
87. Karlberg M, Hall K, Quickert N, et al. What inner ear diseases cause benign paroxysmal positional vertigo? *Acta Otolaryngol*. 2000;120:380-385.
88. Kerber KA. Benign paroxysmal positional vertigo: opportunities squandered. *Ann N Y Acad Sci*. 2015;1343:106-112.
89. Newman-Toker, DE, Edlow, JA. TiTrATE: a novel, evidence-based approach to diagnosing acute dizziness and vertigo. *Neurol Clin*. 2015;33:577-599.
90. Kentala E, Rauch SD. A practical assessment algorithm for diagnosis of dizziness. *Otolaryngol Head Neck Surg*. 2003;128:54-59.
91. Thorp MA, Shehab ZP, Bance ML, et al. The AAO-HNS Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Ménière's disease: have they been applied in the published literature of the last decade? *Clin Otolaryngol Allied Sci*. 2003;28:173-176.
92. Baloh RW. Clinical practice: vestibular neuritis. *N Engl J Med*. 2003;348(11):1027-1032.
93. Kentala E. Characteristics of six otologic diseases involving vertigo. *Am J Otol*. 1996;17:883-892.
94. Kentala E, Laurikkala J, Pyykkö I, et al. Discovering diagnostic rules from a neurotologic database with genetic algorithms. *Ann Otol Rhinol Laryngol*. 1999;108:948-954.
95. Minor LB, Cremer PD, Carey JP, et al. Symptoms and signs in superior canal dehiscence syndrome. *Ann N Y Acad Sci*. 2001;942:259-273.
96. Rosowski JJ, Songer JE, Nakajima HH, et al. Clinical, experimental, and theoretical investigations of the effect of superior semicircular canal dehiscence on hearing mechanisms. *Otol Neurotol*. 2004;25:323-332.
97. Marzo SJ, Leonetti JP, Raffin MJ, et al. Diagnosis and management of post-traumatic vertigo. *Laryngoscope*. 2004;114:1720-1723.
98. Hoffer ME, Gottshall KR, Moore R, Balough BJ, Wester D. Characterizing and treating dizziness after mild head trauma. *Otol Neurotol*. 2004;25:135-138.
99. Davies RA, Luxon LM. Dizziness following head injury: a neuro-otological study. *J Neurol*. 1995;242:222-230.
100. Labuguen RH. Initial evaluation of vertigo. *Am Fam Physician*. 2006;73:244-251.
101. Baloh RW. Dizziness: neurological emergencies. *Neurol Clin*. 1998;16:305-321.
102. Dunnaway HM, Welling DB. Intracranial tumors mimicking benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 1998;118:429-436.
103. Lempert T, Neuhauser H. Epidemiology of vertigo, migraine and vestibular migraine. *J Neurol*. 2009;256:333-338.
104. Seemungal B, Kaski D, Lopez-Escamez JA. Early diagnosis and management of acute vertigo from vestibular migraine and Ménière's disease. *Neurol Clin*. 2015;33:619-628.

105. Kerber KA. Acute continuous vertigo. *Semin Neurol.* 2013;33:173-178.
106. Lee H, Sohn SI, Cho YW, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular topographical patterns. *Neurology.* 2006;67:1178-1183.
107. Blum CA, Kasner SE. Transient ischemic attacks presenting with dizziness or vertigo. *Neurol Clin.* 2015;33:629-642.
108. Paul NLM, Simoni M, Rothwell PM, et al. Transient isolated brainstem symptoms preceding posterior circulation stroke: a population-based study. *Lancet Neurol.* 2013;12:65-71.
109. Soto-Varela A, Rossi-Izquierdo M, Sánchez-Sellero I, et al. Revised criteria for suspicion of non-benign positional vertigo. *QJM.* 2013;106:317-321.
110. Pula JH, Newman-Toker DE, Kattah JC. Multiple sclerosis as a cause of the acute vestibular syndrome. *J Neurol.* 2013;260:1649-1654.
111. Frohman EM, Zhang H, Dewey RB, et al. Vertigo in MS: utility of positional and particle repositioning maneuvers. *Neurology.* 2000;55:1566-1569.
112. Jacob RG, Furman JM, Durrant JD, et al. Panic, agoraphobia, and vestibular dysfunction. *Am J Psychiatry.* 1996;153:503-512.
113. Furman JM, Redfern MS, Jacob RG. Vestibulo-ocular function in anxiety disorders. *J Vestib Res.* 2006;16:209-215.
114. Bracher ES, Almeida CI, Almeida RR, et al. A combined approach for the treatment of cervical vertigo. *J Manipulative Physiol Ther.* 2000;23:96-100.
115. Padoan S, Karlberg M, Fransson PA, et al. Passive sustained turning of the head induces asymmetric gain of the vestibulo-ocular reflex in healthy subjects. *Acta Otolaryngol.* 1998;118:778-782.
116. Rubenstein LZ, Powers CM, MacLean CH. Quality indicators for the management and prevention of falls and mobility problems in vulnerable elders. *Ann Intern Med.* 2001;135:686-693.
117. Gazzola JM, Gananca FF, Aratani MC, et al. Circumstances and consequences of falls in elderly people with vestibular disorder. *Rev Bras Otorrinolaringol (Engl Ed).* 2006;72:388-392.
118. Agrawal Y, Carey JP, Della Santina CC, et al. Disorders of balance and vestibular function in US adults: data from the National Health and Nutrition Examination Survey, 2001-2004. *Arch Intern Med.* 2009;169:938-944.
119. Murdin L, Schilder AGM. Epidemiology of balance symptoms and disorders in the community: a systematic review. *Otol Neurotol.* 2015;36:387-392.
120. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. *N Engl J Med.* 1988;319:1701-1707.
121. Agrawal Y, Ward BK, Minor LB. Vestibular dysfunction: prevalence, impact, and need for targeted treatment. *J Vestib Res.* 2013;23:113-117.
122. Rubenstein LZ. Falls in older people: epidemiology, risk factors and strategies for prevention. *Age Ageing.* 2006;35(suppl 2):ii37-ii41.
123. Mathias S, Nayak USL, Isaacs B, et al. Balance in elderly patients: the “get-up and go” test. *Arch Phys Med Rehabil.* 1986;67:387-389.
124. Tinetti ME, Williams TF, Mayewski R. Fall risk index for elderly patients based on number of chronic disabilities. *Am J Med.* 1986;80:429-434.
125. Berg K, Wood-Dauphinee S, Williams JI, Maki B. Measuring balance in the elderly: validation of an instrument. *Can J Pub Health.* 1992;2:S7-S11.
126. Casellini CM, Vinik AI. Clinical manifestations and current treatment options for diabetic neuropathies. *Endocr Pract.* 2007;13:550-566.
127. Richardson JK. Factors associated with falls in older patients with diffuse polyneuropathy. *J Am Geriatr Soc.* 2002;50:1767-1773.
128. Tilling LM, Darawil K, Britton M. Falls as a complication of diabetes mellitus in older people. *J Diabetes Complications.* 2006;20:158-162.
129. Yu S, Liu F, Cheng Z, Wang Q. Association between osteoporosis and benign paroxysmal positional vertigo: a systematic review. *BMC Neurology.* 2014;14:110.
130. Jönsson R, Sixt E, Landahl S, et al. Prevalence of dizziness and vertigo in an urban elderly population. *J Vestib Res.* 2004;14:47-52.
131. Motin M, Keren O, Groswasser Z, et al. Benign paroxysmal positional vertigo as the cause of dizziness in patients after severe traumatic brain injury: diagnosis and treatment. *Brain Inj.* 2005;19:693-697.
132. Gordon CR, Levite R, Joffe V, et al. Is posttraumatic benign paroxysmal positional vertigo different from the idiopathic form? *Arch Neurol.* 2004;61:1590-1593.
133. Aron M, Lea J, Nakku D, et al. Symptom resolution rates of posttraumatic versus nontraumatic benign paroxysmal positional vertigo: a systematic review. *Otolaryngol Head Neck Surg.* 2015;153:721-730.
134. Ahn S-K, Jeon S-Y, Kim J-P, et al. Clinical characteristics and treatment of benign paroxysmal positional vertigo after traumatic brain injury. *J Trauma.* 2011;70:442-446.
135. Frohman EM, Kramer PD, Dewey RB, et al. Benign paroxysmal positioning vertigo in multiple sclerosis: diagnosis, pathophysiology and therapeutic techniques. *Mult Scler.* 2003;9:250-255.
136. Gamiz MJ, Lopez-Escamez JA. Health-related quality of life in patients over sixty years old with benign paroxysmal positional vertigo. *Gerontology.* 2004;50:82-86.
137. Lopez-Escamez JA, Gamiz MJ, Fernandez-Perez A, et al. Impact of treatment on health-related quality of life in patients with posterior canal benign paroxysmal positional vertigo. *Otol Neurotol.* 2003;24:637-641.
138. Turski P, Seidenwurm D, Davis P, et al; American College of Radiology. *ACR Appropriateness Criteria: Vertigo and Hearing Loss.* Reston, VA: American College of Radiology; 1996.
139. Turski P, Seidenwurm D, Davis P; American College of Radiology. *Expert Panel on Neuroimaging: Vertigo and Hearing Loss.* Reston, VA: American College of Radiology; 2006.
140. Colledge NR, Barr-Hamilton RM, Lewis SJ, et al. Evaluation of investigations to diagnose the cause of dizziness in elderly people: a community based controlled study. *BMJ.* 1996;313:788-792.

141. Day JJ, Freer CE, Dixon AK, et al. Magnetic resonance imaging of the brain and brain-stem in elderly patients with dizziness. *Age Ageing*. 1990;19:144-150.
142. Brandt T, Dieterich M. VIIIth nerve vascular compression syndrome: vestibular paroxysmia. *Baillieres Clin Neurol*. 1994;3:565-575.
143. Jacobson GP, Butcher JA, Newman CW, et al. When paroxysmal positioning vertigo isn't benign. *J Am Acad Audiol*. 1995;6:346-349.
144. Kumar A, Patni AH, Charbel F. The Chiari I malformation and the neurotologist. *Otol Neurotol*. 2002;23:727-735.
145. Gizzi M, Riley E, Molinari S. The diagnostic value of imaging the patient with dizziness: a Bayesian approach. *Arch Neurol*. 1996;53:1299-1304.
146. Fife TD, Tusa RJ, Furman JM, et al. Assessment: vestibular testing techniques in adults and children: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology*. 2000;55:1431-1441.
147. Gordon CR, Shupak A, Spitzer O, et al. Nonspecific vertigo with normal otoneurological examination: the role of vestibular laboratory tests. *J Laryngol Otol*. 1996;110:1133-1137.
148. Phillips JS, FitzGerald JE, Bath AP. The role of the vestibular assessment. *J Laryngol Otol*. 2009;123:1212-1215.
149. Kentala E, Pyykkö I. Vertigo in patients with benign paroxysmal positional vertigo. *Acta Otolaryngol Suppl*. 2000;543:20-22.
150. Bath AP, Walsh RM, Ranalli P, et al. Experience from a multi-disciplinary "dizzy" clinic. *Am J Otol*. 2000;21:92-97.
151. Roberts RA, Gans RE, Kastner AH, et al. Prevalence of vestibulopathy in benign paroxysmal positional vertigo patients with and without prior otologic history. *Int J Audiol*. 2005;44:191-196.
152. Korres SG, Balatsouras DG. Diagnostic, pathophysiologic, and therapeutic aspects of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2004;131:438-444.
153. Hong SM, Yeo SG, Kim SW, et al. The results of vestibular evoked myogenic potentials, with consideration of age-related changes, in vestibular neuritis, benign paroxysmal positional vertigo, and Ménière's disease. *Acta Otolaryngol*. 2008;128:861-865.
154. Longo G, Onofri M, Pellicciari T, et al. Benign paroxysmal positional vertigo: is vestibular evoked myogenic potential testing useful? *Acta Otolaryngol*. 2012;132:39-43.
155. Lee JD, Park MK, Lee BD, Lee TK, Sung KB, Park JY. Abnormality of cervical vestibular-evoked myogenic potentials and ocular vestibular-evoked myogenic potentials in patients with recurrent benign paroxysmal positional vertigo. *Acta Otolaryngol*. 2013;133:150-153.
156. Hoseinabadi R, Pourbakht A, Yazdani N. The effects of abnormality of cVEMP and oVEMP on rehabilitation outcomes in patients with idiopathic benign paroxysmal positional vertigo. *Eur Arch Otorhinolaryngol*. 2016;273:643-648.
157. Hughes CA, Proctor L. Benign paroxysmal positional vertigo. *Laryngoscope*. 1997;107:607-613.
158. Pollak L, Davies RA, Luxon LL. Effectiveness of the particle repositioning maneuver in benign paroxysmal positional vertigo with and without additional vestibular pathology. *Otol Neurotol*. 2002;23:79-83.
159. Del Rio M, Arriaga MA. Benign positional vertigo: prognostic factors. *Otolaryngol Head Neck Surg*. 2004;130:426-429.
160. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 1992;107:399-404.
161. Li JC. Mastoid oscillation: a critical factor for success in canalith repositioning procedure. *Otolaryngol Head Neck Surg*. 1995;112:670-675.
162. Lempert T, Wolsley C, Davies R, et al. Three hundred sixty-degree rotation of the posterior semicircular canal for treatment of benign positional vertigo: a placebo-controlled trial. *Neurology*. 1997;49:729-733.
163. Wolf M, Hertanu T, Novikov I, et al. Epley's manoeuvre for benign paroxysmal positional vertigo: a prospective study. *Clin Otolaryngol Allied Sci*. 1999;24:43-46.
164. Lopez-Escamez J, Gonzalez-Sanchez M, Salinero J. Meta-analysis of the treatment of benign paroxysmal positional vertigo by Epley and Semont maneuvers. *Acta Otorrinolaringol Esp*. 1999;50:366-370.
165. Asawavichianginda S, Isipradit P, Snidvongs K, et al. Canalith repositioning for benign paroxysmal positional vertigo: a randomized, controlled trial. *Ear Nose Throat J*. 2000;79:732-734, 736.
166. Sherman D, Massoud EA. Treatment outcomes of benign paroxysmal positional vertigo. *J Otolaryngol*. 2001;30:295-299.
167. Angeli SI, Hawley R, Gomez O. Systematic approach to benign paroxysmal positional vertigo in the elderly. *Otolaryngol Head Neck Surg*. 2003;128:719-725.
168. Chang AK, Schoeman G, Hill M. A randomized clinical trial to assess the efficacy of the Epley maneuver in the treatment of acute benign positional vertigo. *Acad Emerg Med*. 2004;11:918-924.
169. White J, Savvides P, Cherian N, et al. Canalith repositioning for benign paroxysmal positional vertigo. *Otol Neurotol*. 2005;26:704-710.
170. Woodworth BA, Gillespie MB, Lambert PR. The canalith repositioning procedure for benign positional vertigo: a meta-analysis. *Laryngoscope*. 2004;114:1143-1146.
171. Teixeira LJ, Machado JN. Maneuvers for the treatment of benign positional paroxysmal vertigo: a systematic review. *Rev Bras Otorrinolaringol (Engl Ed)*. 2006;72:130-139.
172. Prim-Espada MP, De Diego-Sastre JJ, Pérez-Fernández E. Meta-analysis on the efficacy of Epley's manoeuvre in benign paroxysmal positional vertigo. *Neurologia*. 2010;25:295-299.
173. Hilton MP, Pinder DK. The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. *Cochrane Database Syst Rev*. 2014;(12):CD003162.
174. Amor-Dorado JC, Barreira-Fernández MP, Aran-Gonzalez I, et al. Particle repositioning maneuver versus Brandt-Daroff exercise for treatment of unilateral idiopathic BPPV of the posterior semicircular canal: a randomized prospective clinical trial with short- and long-term outcome. *Otol Neurotol*. 2012;33:1401-1407.
175. Brintjes TD, Companjen J, van der Zaag-Loonen HJ, et al. A randomised sham-controlled trial to assess the long-term effect of the Epley manoeuvre for treatment of posterior canal benign paroxysmal positional vertigo. *Clin Otolaryngol*. 2014;39:39-44.

176. Uneri A. Falling sensation in patients who undergo the Epley maneuver: a retrospective study. *Ear Nose Throat J*. 2005;84:82, 84-85.
177. Semont A, Freyss G, Vitte E. Curing the BPPV with a liberatory maneuver. *Adv Otorhinolaryngol*. 1988;42:290-293.
178. Salvinelli F, Casale M, Trivelli M, et al. Benign paroxysmal positional vertigo: a comparative prospective study on the efficacy of Semont's maneuver and no treatment strategy. *Clin Ter*. 2003;154:7-11.
179. Soto Varela A, Bartual Magro J, Santos Perez S, et al. Benign paroxysmal vertigo: a comparative prospective study of the efficacy of Brandt and Daroff exercises, Semont and Epley maneuver. *Rev Laryngol Otol Rhinol (Bord)*. 2001;122:179-183.
180. Chen Y, Zhuang J, Zhang L, et al. Short-term efficacy of Semont maneuver for benign paroxysmal positional vertigo: a double-blind randomized trial. *Otol Neurotol*. 2012;33:1127-1130.
181. Munoz JE, Miklea JT, Howard M, et al. Canalith repositioning maneuver for benign paroxysmal positional vertigo: randomized controlled trial in family practice. *Can Fam Physician*. 2007;53:1049-1053, 1048.
182. Yimtae K, Srirompotong S, Sae-Seaw P. A randomized trial of the canalith repositioning procedure. *Laryngoscope*. 2003;113:828-832.
183. Ruckenstein MJ. Therapeutic efficacy of the Epley canalith repositioning maneuver. *Laryngoscope*. 2001;111:940-945.
184. Sekine K, Imai T, Sato G, et al. Natural history of benign paroxysmal positional vertigo and efficacy of Epley and Lempert maneuvers. *Otolaryngol Head Neck Surg*. 2006;135:529-533.
185. Prokopakis EP, Chimona T, Tsagournisakis M, et al. Benign paroxysmal positional vertigo: 10-year experience in treating 592 patients with canalith repositioning procedure. *Laryngoscope*. 2005;115:1667-1671.
186. Reinink H, Wegner I, Stegeman I, et al. Rapid systematic review of repeated application of the epley maneuver for treating posterior BPPV. *Otolaryngol Head Neck Surg*. 2014;151:399-406.
187. Herdman SJ, Tusa RJ. Complications of the canalith repositioning procedure. *Arch Otolaryngol Head Neck Surg*. 1996;122:281-286.
188. Sridhar S, Panda N. Particle repositioning manoeuvre in benign paroxysmal positional vertigo: is it really safe? *J Otolaryngol*. 2005;34:41-45.
189. Lempert T, Tiel-Wilck K. A positional maneuver for treatment of horizontal-canal benign positional vertigo. *Laryngoscope*. 1996;106:476-478.
190. Fife TD. Recognition and management of horizontal canal benign positional vertigo. *Am J Otol*. 1998;19:345-351.
191. Appiani GC, Catania G, Gagliardi M. A liberatory maneuver for the treatment of horizontal canal paroxysmal positional vertigo. *Otol Neurotol*. 2001;22:66.
192. Casani AP, Vannucci G, Fattori B, et al. The treatment of horizontal canal positional vertigo: our experience in 66 cases. *Laryngoscope*. 2002;112:172-178.
193. Appiani GC, Gagliardi G, Magliulo G. Physical treatment of horizontal canal benign positional vertigo. *Eur Arch Otorhinolaryngol*. 1997;254:326-328.
194. Asprella Libonati G. Diagnostic and treatment strategy of lateral semicircular canal canalolithiasis. *Acta Otorhinolaryngol Ital*. 2005;25:277-283.
195. Chiou W-Y, Lee H-L, Tsai S-C, et al. A single therapy for all subtypes of horizontal canal positional vertigo. *Laryngoscope*. 2005;115:1432-1435.
196. Kim JS, Oh S-Y, Lee S-H, et al. Randomized clinical trial for geotropic horizontal canal benign paroxysmal positional vertigo. *Neurology*. 2012;79:700-707.
197. van den Broek EM, van der Zaag-Loonen HJ, Bruintjes TD. Systematic review: efficacy of Gufoni maneuver for treatment of lateral canal benign paroxysmal positional vertigo with geotropic nystagmus. *Otolaryngol Head Neck Surg*. 2014;150:933-938.
198. Kim JS, Oh SY, Lee SH, et al. Randomized clinical trial for apogeotropic horizontal canal benign paroxysmal positional vertigo. *Neurology*. 2012;28:159-166.
199. Appiani GC, Catania G, Gagliardi M. Repositioning maneuver for the treatment of the apogeotropic variant of horizontal canal benign paroxysmal positional vertigo. *Otol Neurotol*. 2005;26:257-260.
200. Vannucchi P, Giannoni B, Pagnini P. Treatment of horizontal semicircular canal benign paroxysmal positional vertigo. *J Vestib Res*. 1997;7:1-6.
201. Radtke A, Neuhauser H, von Brevern M, et al. A modified Epley's procedure for self-treatment of benign paroxysmal positional vertigo. *Neurology*. 1999;53:1358-1360.
202. Radtke A, von Brevern M, Tiel-Wilck K, et al. Self-treatment of benign paroxysmal positional vertigo: Semont maneuver vs Epley procedure. *Neurology*. 2004;63:150-152.
203. De Stefano A, Dispenza F, Citraro L, et al. Are postural restrictions necessary for management of posterior canal benign paroxysmal positional vertigo? *Ann Otol Rhinol Laryngol*. 2011;120:460-464.
204. Massoud EA, Ireland DJ. Post-treatment instructions in the nonsurgical management of benign paroxysmal positional vertigo. *J Otolaryngol*. 1996;25:121-125.
205. Roberts RA, Gans RE, DeBoodt JL, et al. Treatment of benign paroxysmal positional vertigo: necessity of postmaneuver patient restrictions. *J Am Acad Audiol*. 2005;16:357-366.
206. Balicki HH, Ozbay I. Effects of postural restriction after modified Epley maneuver on recurrence of benign paroxysmal positional vertigo. *Auris Nasus Larynx*. 2014;41:428-431.
207. Cohen HS, Kimball KT. Treatment variations on the Epley maneuver for benign paroxysmal positional vertigo. *Am J Otolaryngol*. 2004;25:33-37.
208. Devaiah AK, Andreoli S. Postmaneuver restrictions in benign paroxysmal positional vertigo: an individual patient data meta-analysis. *Otolaryngol Head Neck Surg*. 2010;142:155-159.
209. Hunt WT, Zimmermann EF, Hilton MP. Modifications of the Epley (canalith repositioning) manoeuvre for posterior canal benign paroxysmal positional vertigo (BPPV). *Cochrane Database Sys Rev*. 2012;(4):CD008675.
210. Toupet M, Ferrary E, Bozorg Grayeli A. Effect of repositioning maneuver type and postmaneuver restrictions on vertigo and dizziness in benign positional paroxysmal vertigo. *Scientific-WorldJournal*. 2012;2012:162123.
211. Heinrichs M, Gaab J. Neuroendocrine mechanisms of stress and social interaction: implications for mental disorders. *Curr Opin Psych*. 2007;20:158-162.
212. Cawthorne T. The physiologic basis for head exercises. *J Chart Soc Physiother*. 1944;30:106-107.

213. Cooksey FS. Rehabilitation in vestibular injuries. *Proc R Soc Med.* 1946;39:273-278.
214. Dix MR. The rationale and technique of head exercises in the treatment of vertigo. *Acta Otorhinolaryngol Bel.* 1979;33:370-384.
215. Whitney SL, Sparto PJ. Principles of vestibular physical therapy rehabilitation. *NeuroRehabilitation.* 2011;29:157-166.
216. Hillier SL, Hollohan V. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. *Cochrane Database Syst Rev.* 2007;(4):CD005397.
217. McDonnell MN, Hillier SL. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. *Cochrane Database Syst Rev.* 2015;(1):CD005397.
218. Herdman SJ, Blatt PJ, Schubert MC. Vestibular rehabilitation of patients with vestibular hypofunction or with benign paroxysmal positional vertigo. *Curr Opin Neurol.* 2000;13:39-43.
219. Telian SA, Shepard NT. Update on vestibular rehabilitation therapy. *Otolaryngol Clin North Am.* 1996;29:359-371.
220. Whitney SL, Rossi MM. Efficacy of vestibular rehabilitation. *Otolaryngol Clin North Am.* 2000;33:659-672.
221. Hall CD, Herdman SJ, Whitney SL, et al. Vestibular rehabilitation for peripheral vestibular hypofunction: an evidence-based clinical practice guideline. *J Neurologic Phys Ther.* 2016;40:124-155.
222. Han BI, Song HS, Kim JS. Vestibular rehabilitation therapy: review of indications, mechanisms, and key exercises. *J Clin Neurol.* 2011;7:184-196.
223. Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. *Arch Otolaryngol.* 1980;106:484-485.
224. Brandt T, Steddin S, Daroff RB. Therapy for benign paroxysmal positioning vertigo, revisited. *Neurology.* 1994;44:796-800.
225. Toledo H, Cortés ML, Pane C, et al. Semont maneuver and vestibular rehabilitation exercises in the treatment of benign paroxysmal postural vertigo: a comparative study. *Neurologia.* 2000;15:152-157.
226. Di Girolamo S, Paludetti G, Briglia G, et al. Postural control in benign paroxysmal positional vertigo before and after recovery. *Acta Otolaryngol.* 1998;118:289-293.
227. Giacomini PG, Alessandrini M, Magrini A. Long-term postural abnormalities in benign paroxysmal positional vertigo. *ORL J Otorhinolaryngol Relat Spec.* 2002;64:237-241.
228. Chang W-C, Yang Y-R, Hsu L-C, Chern CM, Wang RY. Balance improvement in patients with benign paroxysmal positional vertigo. *Clin Rehabil.* 2008;22:338-347.
229. Brandt T. Phobic postural vertigo. *Neurology.* 1996;46:1515-1519.
230. Staab JP. Chronic subjective dizziness. *Continuum Lifelong Learning Neurol.* 2012;18:1118-1141.
231. Horak FB, Jones-Rycewicz C, Black FO, Shumway-Cook A. Effects of vestibular rehabilitation on dizziness and imbalance. *Otolaryngol Head Neck Surg.* 1992;106:175-180.
232. Shepard NT, Telian SA. Programmatic vestibular rehabilitation. *Otolaryngol Head Neck Surg.* 1995;112:173-182.
233. Hain TC, Uddin M. Pharmacological treatment of vertigo. *CNS Drugs.* 2003;17:85-100.
234. Hain TC, Yacovino D. Pharmacologic treatment of persons with dizziness. *Neurol Clin.* 2005;23:831-853.
235. Ancelin ML, Artero S, Portet F, et al. Non-degenerative mild cognitive impairment in elderly people and use of anticholinergic drugs: longitudinal cohort study. *BMJ.* 2006;332:455-459.
236. Hebert C, Delaney JAC, Hemmelgarn B, et al. Benzodiazepines and elderly drivers: a comparison of pharmacoepidemiological study designs. *Pharmacoepidemiol Drug Saf.* 2007;16:845-849.
237. Barbone F, McMahon AD, Davey PG, et al. Association of road-traffic accidents with benzodiazepine use. *Lancet.* 1998;352:1331-1336.
238. Engeland A, Skurtveit S, Mørland J. Risk of road traffic accidents associated with the prescription of drugs: a registry-based cohort study. *Ann Epidemiol.* 2007;17:597-602.
239. Jauregui I, Mullol J, Bartra J, et al. H1 antihistamines: psychomotor performance and driving. *J Investig Allergol Clin Immunol.* 2006;16(suppl 1):37-44.
240. Hartikainen S, Lönnroos E, Louhivuori K. Medication as a risk factor for falls: critical systematic review. *J Gerontol A Biol Sci Med Sci.* 2007;62:1172-1181.
241. Hien LTT, Cumming RG, Cameron ID, et al. Atypical antipsychotic medications and risk of falls in residents of aged care facilities. *J Am Geriatr Soc.* 2005;53:1290-1295.
242. Landi F, Russo A, Liperoti R, et al. Anticholinergic drugs and physical function among frail elderly population. *Clin Pharmacol Ther.* 2007;81:235-241.
243. Pit SW, Byles JE, Henry DA, et al. A Quality Use of Medicines program for general practitioners and older people: a cluster randomised controlled trial. *Med J Aust.* 2007;187:23-30.
244. Rudolph JL, Salow MJ, Angelini MC, et al. The anticholinergic risk scale and anticholinergic adverse effects in older persons. *Arch Intern Med.* 2008;168:508-513.
245. Carlow TJ. Medical treatment of nystagmus and ocular motor disorders. *Int Ophthalmol Clin.* 1986;26:251-264.
246. Cesarani A, Alpin D, Monti B, et al. The treatment of acute vertigo. *Neurol Sci.* 2004;25(suppl 1):S26-S30.
247. Fujino A, Tokumasu K, Yosio S, et al. Vestibular training for benign paroxysmal positional vertigo: its efficacy in comparison with antivertigo drugs. *Arch Otolaryngol Head Neck Surg.* 1994;120:497-504.
248. Sacco RR, Burmeister DB, Rupp VA, et al. Management of benign paroxysmal positional vertigo: a randomized controlled trial. *J Emerg Med.* 2014;46:575-581.
249. Salvinelli F, Trivelli M, Casale M, et al. Treatment of benign positional vertigo in the elderly: a randomized trial. *Laryngoscope.* 2004;114:827-831.
250. Itaya T, Yamamoto E, Kitano H, et al. Comparison of effectiveness of maneuvers and medication in the treatment of benign paroxysmal positional vertigo. *ORL J Otorhinolaryngol Relat Spec.* 1997;59:155-158.
251. McClure JA, Willett JM. Lorazepam and diazepam in the treatment of benign paroxysmal vertigo. *J Otolaryngol.* 1980;9:472-477.
252. Maslovara S, Soldo SB, Puksec M, Balaban B, Penavic IP. Benign paroxysmal positional vertigo (BPPV): influence of

- pharmacotherapy and rehabilitation therapy on patients' recovery rate and life quality. *NeuroRehabilitation*. 2012;31:435-441.
253. Sundararajan I, Rangachari V, Sumathi V, Kumar K. Epley's manoeuvre versus Epley's manoeuvre plus labyrinthine sedative as management of benign paroxysmal positional vertigo: prospective, randomised study. *J Laryngol Otol*. 2011;125:572-575.
 254. Jung HJ, Koo J-W, Kim CS, Kim JS, Song JJ. Anxiolytics reduce residual dizziness after successful canalith repositioning maneuvers in benign paroxysmal positional vertigo. *Acta Otolaryngol*. 2012;132:277-284.
 255. Guneri EA, Kustutan O. The effects of betahistine in addition to epley maneuver in posterior canal benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2012;146:104-108.
 256. Cohen HS, Kimball KT. Effectiveness of treatments for benign paroxysmal positional vertigo of the posterior canal. *Otol Neurotol*. 2005;26:1034-1040.
 257. Rupa V. Persistent vertigo following particle repositioning maneuvers: an analysis of causes. *Arch Otolaryngol Head Neck Surg*. 2004;130:436-439.
 258. Amor-Dorado JC, Barreira-Fernández MP, Aran-Gonzalez I, Casariego-Vales E, Llorca J, Gonzalez-Gay MA. Particle repositioning maneuver versus Brandt-Daroff exercise for treatment of unilateral idiopathic BPPV of the posterior semicircular canal: a randomized prospective clinical trial with short- and long-term outcome. *Otol Neurotol*. 2012;33:1401-1407.
 259. Badawy WM, Gad El-Mawla EK, Chedid AE, Mustafa AH. Effect of a hybrid maneuver in treating posterior canal benign paroxysmal positional vertigo. *J Am Acad Audiol*. 2015;26:138-144.
 260. Balıkcı HH, Ozbay I. Effects of postural restriction after modified Epley maneuver on recurrence of benign paroxysmal positional vertigo. *Auris Nasus Larynx*. 2014;41:428-431.
 261. Dal T, Özlüoğlu LN, Ergin NT. The canalith repositioning maneuver in patients with benign positional vertigo. *Eur Arch Otorhinolaryngol*. 2000;257:133-136.
 262. Woodworth BA, Gillespie MB, Lambert PR. The canalith repositioning procedure for benign positional vertigo: a meta-analysis. *Laryngoscope*. 2004;114:1143-1146.
 263. Teixeira LJ, Machado JN. Maneuvers for the treatment of benign positional paroxysmal vertigo: a systematic review. *Braz J Otorhinolaryngol*. 2006;72:130-139.
 264. Froehling DA, Bowen JM, Mohr DN, et al. The canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo: a randomized controlled trial. *Mayo Clin Proc*. 2000;75:695-700.
 265. Lynn S, Pool A, Rose D, Brey R, Suman V. Randomized trial of the canalith repositioning procedure. *Otolaryngol Head Neck Surg*. 1995;113:712-720.
 266. Munoz JE, Miklea JT, Howard M, et al. Canalith repositioning maneuver for benign paroxysmal positional vertigo: randomized controlled trial in family practice. *Can Fam Physician*. 2007;53:1049-1053, 1048.
 267. Sekine K, Imai T, Sato G, et al. Natural history of benign paroxysmal positional vertigo and efficacy of Epley and Lempert maneuvers. *Otolaryngol Head Neck Surg*. 2006;135:529-533.
 268. von Brevern M, Seelig T, Radtke A, et al. Short-term efficacy of Epley's manoeuvre: a double-blind randomised trial. *J Neurol Neurosurg Psychiatry*. 2006;77:980-982.
 269. Rupa V. Persistent vertigo following particle repositioning maneuvers: an analysis of causes. *Arch Otolaryngol Head Neck Surg*. 2004;130:436-439.
 270. Furman JM, Cass SP. A practical work-up for vertigo. *Contemp Intern Med*. 1995;7:24-27, 31-32, 35-38.
 271. von Brevern M, Seelig T, Radtke A, et al. Short-term efficacy of Epley's manoeuvre: a double-blind randomised trial. *J Neurol Neurosurg Psychiatry*. 2006;77:980-982.
 272. Helminski JO, Zee DS, Janssen I, Hain TC. Effectiveness of particle repositioning maneuvers in the treatment of benign paroxysmal positional vertigo: a systematic review. *Phys Ther*. 2010;90:663-678.
 273. Van Duijn JG. Rapid systematic review of the epley maneuver for treating posterior canal benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2014;15:925-932.
 274. Brocchetti F, Garaventa G, Ameli F, et al. Effect of repetition of Semont's manoeuvre on benign paroxysmal positional vertigo of posterior semicircular canal. *Acta Otorhinolaryngol Ital*. 2003;23:428-435.
 275. Beynon GJ, Baguley DM, da Cruz MJ. Recurrence of symptoms following treatment of posterior semicircular canal benign positional paroxysmal vertigo with a particle repositioning manoeuvre. *J Otolaryngol*. 2000;29:2-6.
 276. Monobe H, Sugawara K, Murofushi T. The outcome of the canalith repositioning procedure for benign paroxysmal positional vertigo: are there any characteristic features of treatment failure cases? *Acta Otolaryngol Suppl*. 2001;545:38-40.
 277. Bergenius J, Perols O. Vestibular neuritis: a follow-up study. *Acta Otolaryngol*. 1999;119:895-899.
 278. Dornhoffer JL, Colvin GB. Benign paroxysmal positional vertigo and canalith repositioning: clinical correlations. *Am J Otol*. 2000;21:230-233.
 279. Kayan A, Hood JD. Neuro-otological manifestations of migraine. *Brain*. 1984;107:1123-1142.
 280. Chang W-C, Hsu L-C, Yang Y-R, et al. Balance ability in patients with benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2006;135:534-540.
 281. Black FO, Nashner LM. Postural disturbance in patients with benign paroxysmal positional nystagmus. *Ann Otol Rhinol Laryngol*. 1984;93:595-599.
 282. Bertholon P, Bronstein AM, Davies RA, et al. Positional down beating nystagmus in 50 patients: cerebellar disorders and possible anterior semicircular canalithiasis. *J Neurol Neurosurg Psychiatry*. 2002;72:366-372.
 283. Dal T, Özlüoğlu LN, Ergin NT. The canalith repositioning maneuver in patients with benign positional vertigo. *Eur Arch Otorhinolaryngol*. 2000;257:133-136.
 284. Smouha EE, Roussos C. Atypical forms of paroxysmal positional nystagmus. *Ear Nose Throat J*. 1995;74:649-656.
 285. Buttner U, Helmchen C, Brandt T. Diagnostic criteria for central versus peripheral positioning nystagmus and vertigo: a review. *Acta Otolaryngol*. 1999;119:1-5.

286. Sakaida M, Takeuchi K, Ishinaga H, et al. Long-term outcome of benign paroxysmal positional vertigo. *Neurology*. 2003;60:1532-1534.
287. Brandt T, Dieterich M. Vestibular falls. *J Vestib Res*. 1993;3:3-14.
288. Imbaud Genieys S. Vertigo, dizziness and falls in the elderly. *Ann Otolaryngol Chir Cervicofac*. 2007;124:189-196.
289. Howick J, Chalmers I, Glasziou; OCEBM Levels of Evidence Working Group. The Oxford 2011 levels of evidence. <http://www.cebm.net/index.aspx?o=5653>. Accessed October 22, 2015.
290. Lee JB, Han DH, Choi SJ, et al. Efficacy of the "bow and lean test" for the management of horizontal canal benign paroxysmal positional vertigo. *Laryngoscope*. 2010;120(11):2339-2346.
291. Choung YH, Shin YR, Kahng H, Park K, Choi SJ. "Bow and lean test" to determine the affected ear of horizontal canal benign paroxysmal positional vertigo. *Laryngoscope*. 2006;116(10):1776-1781.
292. Liang S-B, Li L, He H-Y. The efficacy of Epley procedure for treatment of benign paroxysmal positional vertigo of the posterior semicircular canal. *Journal of Youjiang Medical University for Nationalities*. 2010;2:7.
293. Mazoor T, Niazi SB. Efficacy of semont manoeuvre verses epley manoeuvre in benign paroxysmal positional vertigo. *PAMFJ*. 2011;61:2.
294. Xie K, Du S-W, Gao J-J, Shou G-l, Jian H-Y, Li Y-Z. Clinical efficacy of Epley procedure for treatment of benign paroxysmal positional vertigo of posterior semicircular canal. *Chinese Journal of General Practice*. 2012;2:20.