Otolaryngology–Head and Neck Surgery (2008) 139, S47-S81

GUIDELINES

Clinical practice guideline: Benign paroxysmal positional vertigo

Neil Bhattacharyya, MD, Reginald F. Baugh, MD, Laura Orvidas, MD, David Barrs, MD, Leo J. Bronston, DC, MAppSc, Stephen Cass, MD, MPH, Ara A. Chalian, MD, Alan L. Desmond, AuD, Jerry M. Earll, MD, Terry D. Fife, MD, Drew C. Fuller, MD, MPH, James O. Judge, MD, Nancy R. Mann, MD, Richard M. Rosenfeld, MD, MPH, Linda T. Schuring, MSN, RN, Robert W. P. Steiner, MD, PhD, Susan L. Whitney, PhD, and **Jenissa Haidari, MPH,** Boston, MA; Temple, TX; Rochester, MN; Phoenix, AR; La Cross, WI; Denver, CO; Philadelphia, PA; Princeton, WV; Washington, DC; Baltimore, MD; Hartford, CT; Detroit, MI; Brooklyn, NY; New Smyrna Beach, FL; Louisville, KY; Pittsburgh, PA; and Alexandria, VA

OBJECTIVES: This guideline provides evidence-based recommendations on managing benign paroxysmal positional vertigo (BPPV), which is the most common vestibular disorder in adults, with a lifetime prevalence of 2.4 percent. The guideline targets patients aged 18 years or older with a potential diagnosis of BPPV, evaluated in any setting in which an adult with BPPV would be identified, monitored, or managed. This guideline is intended for all clinicians who are likely to diagnose and manage adults with BPPV.

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 tests such as radiographic imaging and vestibular testing, and to promote the use of effective repositioning maneuvers for treatment. In creating this guideline, the American Academy of Otolaryngology—Head and Neck Surgery Foundation selected a panel representing the fields of audiology, chiropractic medicine, emergency medicine, family medicine, geriatric medicine, internal medicine, neurology, nursing, otolaryngology–head and neck surgery, physical therapy, and physical medicine and rehabilitation.

RESULTS: The panel made *strong recommendations* that 1) clinicians should diagnose posterior semicircular canal BPPV when vertigo associated with nystagmus is provoked by the Dix-Hallpike maneuver.

The panel made *recommendations against* 1) radiographic imaging, vestibular testing, or both in patients diagnosed with BPPV, unless the diagnosis is uncertain or there are additional symptoms or signs unrelated to BPPV that warrant testing; and 2) routinely treating BPPV with vestibular suppressant medications such as antihistamines or benzodiazepines.

The panel made *recommendations* that 1) if the patient has a history compatible with BPPV and the Dix-Hallpike test is negative, clinicians should perform a supine roll test to assess for lateral semicircular canal BPPV; 2) clinicians should differentiate BPPV from other causes of imbalance, dizziness, and vertigo; 3) clinicians should question patients with BPPV for factors that modify management including impaired mobility or balance, CNS disorders, lack of home support, and increased risk for falling; 4) clinicians should treat patients with posterior canal BPPV with a particle repositioning maneuver (PRM); 5) clinicians should reassess patients within 1 month after an initial period of observation or treatment to confirm symptom resolution; 6) clinicians should evaluate patients with BPPV who are initial treatment failures for persistent BPPV or underlying peripheral vestibular or CNS disorders; and 7) clinicians should counsel patients regarding the impact of BPPV on their safety, the potential for disease recurrence, and the importance of follow-up.

The panel offered as *options* that 1) clinicians may offer vestibular rehabilitation, either self-administered or with a clinician, for the initial treatment of BPPV and 2) clinicians may offer observation as initial management for patients with BPPV and with assurance of follow-up.

The panel made *no recommendation* concerning audiometric testing in patients diagnosed with BPPV.

DISCLAIMER: This clinical practice guideline is not intended as a sole source of guidance in managing benign paroxysmal positional vertigo. 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 judgement or establish a protocol for all individuals with this condition, and may not provide the only appropriate approach to diagnosing and managing this problem.

Received August 20, 2008; accepted August 21, 2008.

0194-5998/$34.00 © 2008 American Academy of Otolaryngology–Head and Neck Surgery Foundation. All rights reserved.
doi:10.1016/j.otohns.2008.08.022

A primary complaint of dizziness accounts for 5.6 million clinic visits in the United States per year, and between 17 and 42 percent of patients with vertigo ultimately receive a diagnosis of benign paroxysmal positional vertigo (BPPV).[1-3] BPPV is a form of positional vertigo.

- *Positional vertigo* is defined as a spinning sensation produced by changes in head position relative to gravity.
- *Benign paroxysmal positional vertigo* is defined as a disorder of the inner ear characterized by repeated episodes of positional vertigo.

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 CNS disorder and that the overall prognosis for recovery was favorable.[4] However, undiagnosed and untreated BPPV may not have "benign" functional, health, and quality-of-life impacts. The term *paroxysmal* in this context describes the rapid and sudden onset of the vertigo associated with an episode of 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 because it is the most common terminology encountered in the literature and in clinical practice.

BPPV is most commonly clinically encountered as one of two variants: BPPV of the posterior semicircular canal (posterior canal BPPV) or BPPV of the lateral semicircular canal (also known as horizontal canal BPPV).[5-7] Posterior canal BPPV is more common than horizontal canal BPPV, constituting approximately 85 to 95 percent of BPPV cases.[7] Although debated, posterior canal BPPV is most commonly thought to be due to canalithiasis. Debris (thought to be fragmented endolymph particles) entering the posterior canal becomes "trapped" and causes inertial changes in the posterior canal, thereby resulting in abnormal nystagmus and vertigo with head motion in the plane of the canal.[7,8] Lateral (horizontal) canal BPPV accounts for between 5 and 15 percent of BPPV cases.[6,7] 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, multiple canal BPPV, and bilateral multiple canal BPPV.

HEALTH CARE BURDEN OF BPPV

Overall, the prevalence of BPPV has been reported to range from 10.7 to 64 per 100,000 population[9,10] with a lifetime prevalence of 2.4 percent.[11] BPPV is also the most common vestibular disorder across the lifespan,[7,12,13] although the age of onset is most commonly between the fifth and seventh decades of life.[4] 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 86 percent of patients suffer some interrupted daily activities and lost days at work because of BPPV.[11,14] Therefore, health care costs associated with the diagnosis of BPPV alone approach $2 billion per year. 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 percent of elderly patients undergoing comprehensive geriatric assessment for non–balance-related complaints have unrecognized BPPV.[15]

Older patients with BPPV experience a greater incidence of falls, depression, and impairments of their daily activities.[15] Furthermore, falls can cause secondary injury including fractures or brain injury and may lead to unplanned hospital and nursing home admission. 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. 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.[16] These variations relate to both diagnostic strategies for BPPV and rates of utilization of various treatment options available for BPPV within and across the various medical specialties and disciplines involved in its management.[17] Delays in the diagnosis and treatment of BPPV have both cost and quality-of-life implications for both patients and their caregivers.

Recent data suggest that patients with BPPV suffer from delays in diagnosis and treatment on the order of months, and that patients with underlying diagnosis of BPPV often received inappropriately prescribed medications such as vestibular suppressants and potentially unnecessary diagnostic testing.[17] 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.

PURPOSE OF BPPV GUIDELINE

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 applies to any setting in which BPPV would be identified, monitored, or managed. The target patient for the guideline is aged 18 years or older with a clinical diagnosis of BPPV. No specific recommendations are made concerning surgical therapy for BPPV.

The guideline will focus on BPPV, recognizing that BPPV may arise in conjunction with other neurological or otological conditions, and that the treatment of the symptom components specifically related to BPPV may still be managed according to the guideline. This guideline will not discuss BPPV affecting the anterior semicircular canal.[18] It also will not discuss benign paroxysmal vertigo of childhood, disabling positional vertigo due to vascular loop compression in the brain stem 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.

Existing guidelines and recommendation documents on BPPV are sparse and are broad reviews of the literature with limited multidisciplinary input. Recently published reviews and practice parameters have focused on treatment, and have not reported recommendations for diagnosis and follow-up of this condition.[19] Our goal was to create a multidisciplinary guideline with a specific set of focused recommendations based on an established and transparent process that considers levels of evidence, harm-benefit balance, and expert consensus to resolve gaps in evidence. These specific recommendations may then be used to develop performance measures and identify avenues for quality improvement.

The primary outcome considered in this guideline is the resolution of the symptoms associated with BPPV. Secondary outcomes considered include a more efficient return to regular activities and work, minimization of the use of inappropriate medications and unnecessary diagnostic tests, reduction in the recurrence of BPPV, and reduction in adverse events associated with undiagnosed or untreated BPPV. Other outcomes considered include minimization of costs in the diagnosis and treatment of BPPV, minimization of return physician visits, and maximization of the health-related quality of life of individuals afflicted with BPPV. The significant incidence of BPPV and the wide diversities of diagnostic and therapeutic interventions for BPPV (Table 1) make this an important condition for an up-to-date evidence-based practice guideline.

Table 1
Interventions considered in BPPV guideline development

Diagnosis	Clinical history
	Review of the medication list
	Physical examination
	Dix Hallpike (positional) testing
	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
	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
	Patient self-treatment with vestibular exercises (Brandt-Daroff exercises)
	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
	Prolonged bed rest
	General anesthesia

METHODS

General Methods and Literature Search

The guideline was developed by using an explicit and transparent a priori protocol for creating actionable statements based on supporting evidence and the harm-benefit balance.[20] The multidisciplinary guideline development panel was chosen to represent the fields of audiology, chiropractic medicine, emergency medicine, family medicine, geriatric medicine, internal medicine, neurology, nursing, otolaryngology–head and neck surgery, physical medicine and rehabilitation, and physical therapy. Several group members had significant prior experience in developing clinical practice guidelines, and consultant experts in guideline development were available throughout the guideline construction process.

General search strategy. Several literature searches were performed through December 2007 (initial search) and February 2008 (focused search) by American Academy of Otolaryngology—Head and Neck Surgery Foundation (AAO-HNS) staff. The initial MEDLINE search using "BPPV OR Benign Paroxysmal Position Vertigo" in any field, or "positional [tiab] vertigo [tiab]" or "benign [tiab] positional [tiab] vertigo [tiab]" or "paroxysmal [tiab] positional [tiab] vertigo [tiab]" or "benign [tiab] paroxysmal [tiab] positional [tiab] vertigo [tiab]" in the title or abstract, yielded 1004 potential articles:

1) *Clinical practice guidelines* were identified by limiting the MEDLINE search to one article using "guideline" as a publication type or title word. Search of the National Guideline Clearinghouse (www.guideline.gov) identified 21 guidelines with a topic of vertigo. After elimination of articles that did not have BPPV as the primary focus, no guidelines met quality criteria of being produced under the auspices of a medical association or organization. and having an explicit method for ranking evidence and linking evidence to recommendations. One article by the American College of Radiology addressed "appropriateness criteria" for imaging for BPPV.
2) *Systematic reviews (meta-analyses)* were identified by limiting the MEDLINE search to 26 articles using a validated filter strategy for systematic reviews.[21] Search of the Cochrane Library identified two relevant reviews that met quality criteria of having explicit criteria for conducting the literature search and selecting source articles for inclusion or exclusion.
3) *Randomized controlled trials (RCTs)* were identified by a search of the Cochrane Controlled Trials Register, which identified 28 trials with "BPPV" as a title word.
4) *Original research studies* were identified by limiting the MEDLINE search to articles with a vertigo (MeSH term) as a focus, published in English with human subjects, and not having a publication type of case report. The resultant data set of 741 articles yielded 323 related to diagnosis, 119 to treatment, 223 to etiology, and 125 to prognosis.

Results of all literature searches were distributed to guideline panel members at the first meeting. The materials included full-text hard copy and/or electronic versions of the articles or the listings with abstracts (if available) of the searches for randomized trials and original research. This material was supplemented with targeted searches to address specific needs identified in writing the guideline and specific statements of recommendation.

Targeted searches. From the set of 741 articles, key words from each "bold-faced statement" were used to refine the literature search. For example; from the statement "MEDICAL THERAPY: Clinicians should not routinely treat BPPV with vestibular suppressant medications such as antihistamines or benzodiazepines," the target search strategy would combine "BPPV OR Benign Paroxysmal Position Vertigo" search terms with pharmaco* OR drug therapy OR drug* OR medical OR side effect* OR vestibular suppressant OR suppressant, and so on.

Assessment of Implementability

During the 10 months devoted to guideline development ending in August 2008, the group met twice and participated in three conference calls with interval electronic review and feedback on each guideline draft to ensure accuracy of content and consistency with standardized criteria for reporting clinical practice guidelines. AAO-HNS staff, with guidance from the Yale Center for Medical Informatics, used the GuideLine Implementability Appraisal (GLIA) tool to appraise adherence of the guideline to methodological standards, to improve clarity of recommendations, and to predict potential obstacles to implementation.[22] Panel members received summary appraisals in June 2008 and modified an advanced draft of the guideline. The final draft practice guideline underwent extensive external peer review. Comments were compiled and reviewed by the group chairperson. The recommendations contained in the practice guideline are based on the best available published data through March 2008. Where data were lacking, a combination of clinical experience and expert consensus was used. 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[23] are listed in Tables 2 and 3.

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 might be expected with a recommendation. Options offer the most opportunity for practice variability.[24] Clinicians should always decide and subsequently act in a way that they believe will best serve their 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.[23]

Making recommendations about health practices involves value judgments on the desirability of various outcomes associated with management options. Values applied by the guideline panel sought to minimize harm, diminish

Table 2
Guideline definitions for evidence-based statements

Statement	Definition	Implication
Strong recommendation	A strong recommendation means the benefits of the recommended approach clearly exceed the harms (or that the harms clearly exceed the benefits in the case of a strong negative recommendation) and that the quality of the supporting evidence is excellent (grade A or B)*. In some clearly identified circumstances, strong recommendations may be made on the basis of 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 the benefits exceed the harms (or that the harms exceed the benefits in the case of a negative recommendation), but the quality of evidence is not as strong (grade B or C)*. In some clearly identified circumstances, recommendations may be made on the basis of 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 that either the quality of evidence that exists is suspect (grade D)* or that well-done studies (grade A, B, or C)* 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.
No recommendation	No recommendation means there is both a lack of pertinent evidence (grade D)* and an unclear balance between benefits and harms.	Clinicians should feel little constraint in their decision making and be alert to new published evidence that clarifies the balance of benefit versus harm; patient preference should have a substantial influencing role.

*See Table 3 for definition of evidence grades.

unnecessary testing and inappropriate therapy, and reduce the unnecessary use of vestibular suppressants. The panel also strongly valued expeditious treatment with effective therapeutic maneuvers to minimize symptomatology and quality-of-life impact of BPPV. A major goal of the committee 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-HNS; there was no support or direct involvement of industry at any phase of the development process. Potential conflicts of interest for all panel members in the past 5 years were compiled and distributed before the first conference call. After review and discussion of these disclosures,[25] 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. Finally, 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.[26]

Table 3
Evidence quality for grades of evidence

Grade	Evidence quality
A	Well-designed randomized controlled trials or diagnostic studies performed on a population similar to the guideline's target population
B	Randomized controlled trials or diagnostic studies with minor limitations; overwhelmingly consistent evidence from observational studies
C	Observational studies (case-control and cohort design)
D	Expert opinion, case reports, reasoning from first principles (bench research or animal studies)
X	Exceptional situations for which validating studies cannot be performed and there is a clear preponderance of benefit over harm

BPPV GUIDELINE EVIDENCE-BASED STATEMENTS

Each evidence-based statement is organized in a similar fashion: **evidence-based statement in boldface type**, *followed by an italicized statement on the strength of the recommendation.* Several paragraphs then discuss the evidence base supporting the statement, concluding with an "evidence profile" of aggregate evidence quality, benefit-harm assessment, and statement of costs. Where appropriate, specific exclusionary criteria for patients that may be exceptions to the intended scope or purpose of the evidence-based statement are listed. Finally, there is an explicit statement of the value judgments, the role of patient preferences, and a repeat statement of the strength of the recommendation. An overview of evidence-based statements in the guideline and their interrelationship is shown in Table 4.

The role of patient preference in clinical decision making deserves clarification. For some statements, the evidence base demonstrates clear benefit, which would minimize the role of patient preference. If the evidence is weak or benefits are unclear, however, not all *informed* patients might opt to follow the suggestion. In these cases, the practice of *shared decision making*, in which the management decision is made collaboratively between the clinician and the informed patient, becomes more useful. Factors related to patient preference include (but are not limited to) absolute benefits, adverse effects, costs of drugs or tests, frequency and duration of treatment, and desire for immediate versus delayed therapy. Comorbidity can also impact patient preferences by several mechanisms such as physical comorbidities precluding certain therapeutic maneuvers.

Statement 1a. Diagnosis of Posterior Canal BPPV

Clinicians should diagnose posterior semicircular canal BPPV when vertigo associated with nystagmus is provoked by the Dix-Hallpike maneuver, performed by bringing the patient from an upright to supine position with the head turned 45 degrees to one side and neck extended 20 degrees. *Strong recommendation based on diagnostic studies with minor limitations and a preponderance of benefit over harm.*

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

Table 4
Outline of evidence-based statements

Guideline segment (*Evidence-based statement number*)	Statement strength
I. Presumed benign paroxysmal positional vertigo (BBPV)	
a. Diagnosis of posterior canal BPPV (*Statement #1a*)	Strong recommendation
b. Diagnosis of lateral canal BPPV (*Statement #1b*)	Recommendation
c. Differential diagnosis (*Statement #2a*)	Recommendation
d. Modifying factors (*Statement #2b*)	Recommendation
II. Diagnostic testing	
a. Radiographic and vestibular testing (*Statement #3a*)	Recommendation against
b. Audiometric testing (*Statement #3b*)	No recommendation
III. Treatment	
a. Initial therapy of BPPV	
i. Repositioning maneuvers as initial therapy (*Statement #4a*)	Recommendation
ii. Vestibular rehabilitation as initial therapy (*Statement #4b*)	Option
iii. Observation as initial therapy (*Statement #4c*)	Option
b. Medical therapy (*Statement #5*)	Recommendation against
c. Reassessment of treatment response (*Statement #6a*)	Recommendation
d. Evaluation of treatment failure (*Statement #6b*)	Recommendation
e. Education (*Statement #7*)	Recommendation

Table 5
Diagnostic criteria for posterior canal BPPV

History	Patient reports repeated episodes of vertigo with changes in head position.
Physical examination	Each of the following criteria are fulfilled: • Vertigo associated with nystagmus is provoked by the Dix-Hallpike test. • There is a latency period between the completion of the Dix-Hallpike test and the onset of vertigo and nystagmus. • The provoked vertigo and nystagmus increase and then resolve within a time period of 60 seconds from onset of nystagmus.

examination, characteristic nystagmus is provoked by the Dix-Hallpike maneuver (Table 5).

History

Vertigo has been defined as an "illusory sensation of motion of either the self or the surroundings."[27] The symptoms of vertigo resulting from posterior canal BPPV are typically described by the patient as a rotational or spinning sensation when the patient 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 shoes).[11,28-30]

Patients with BPPV most commonly report discrete, episodic periods of vertigo lasting 1 minute or less and often report modifications or limitations of their general movements to avoid provoking the vertiginous episodes.[31] 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 lightheadedness, dizziness, nausea, or the feeling of being "off balance."[2,11,28,32-37] Approximately 50 percent of patients also report subjective imbalance between the classic episodes of BPPV.[11] In contrast, a history of vertigo *without* associated lightheadedness may increase the a priori likelihood of a diagnosis of posterior canal BPPV.[15] 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.[37]

Other authors have loosened the historical criteria required for BPPV diagnosis with coinage of the term "subjective BPPV" without a positive Dix-Hallpike test.[35,38] 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 episodic symptoms of positional vertigo noted in the patients' history 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 (Table 5, Fig 1).

The nystagmus produced by the Dix-Hallpike maneuvers in posterior canal BPPV typically displays two 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 onset of the nystagmus with this maneuver is largely unspecified in the literature, but the panel felt that a typical latency period would range from 5 to 20 seconds, although it may be as long as 1 minute in rare cases.[4] Second, the provoked subjective vertigo and the nystagmus increase, and then resolve within a time period of 60 seconds from the onset of nystagmus.

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 (Fig 1).[28,39] Temporally, the rate of nystagmus typically begins gently, increases in intensity, and then declines in intensity as it resolves. This has been termed crescendo-decrescendo nystagmus. The nystagmus is again commonly observed after the patient returns to the upright head position and upon arising, but the direction of the nystagmus may be reversed.

Another classical feature of the nystagmus associated with posterior canal BPPV is that the nystagmus typically fatigues (a reduction in severity of nystagmus) when the maneuver is repeated.[29,39] However, repeated performance of the Dix-Hallpike maneuver to demonstrate fatigability is not recommended, because it unnecessarily subjects patients to repeated symptoms of vertigo that may be discomforting, and repeat performance may interfere with the immediate bedside treatment of BPPV.[28] Therefore, the panel did not include fatigability of the nystagmus as a diagnostic criterion.

Performing the Dix-Hallpike Diagnostic Maneuver

The Dix-Hallpike maneuver is performed by the clinician moving the patient through a set of specified head-positioning maneuvers to elicit the expected characteristic nystagmus of posterior canal BPPV (Fig 1).[28,29] Before beginning the maneuver, the clinician should counsel the patient regarding the upcoming movements and warn that

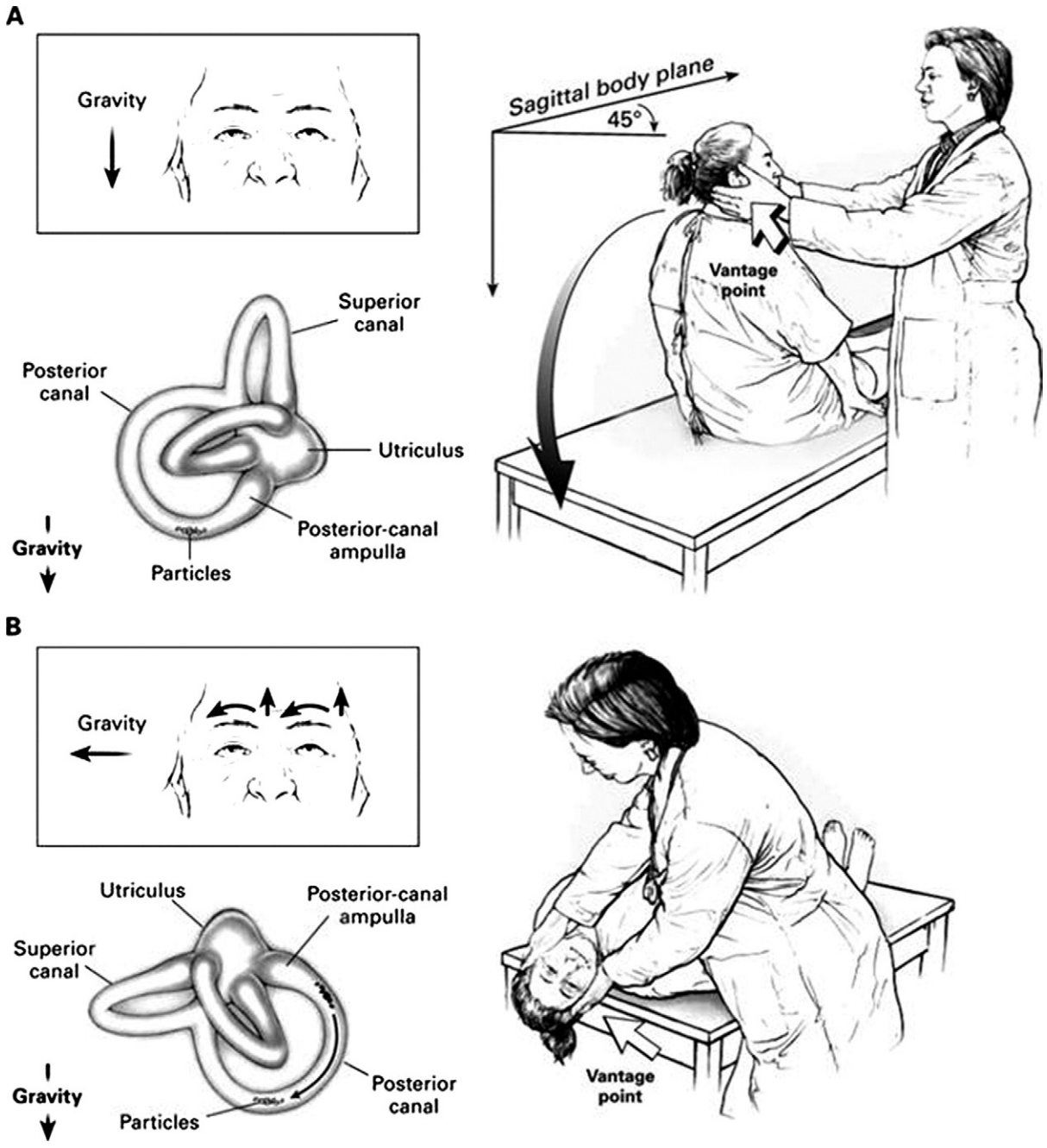

Figure 1 Diagrammatic representation of performance of the Dix-Hallpike maneuver for the diagnosis of posterior canal BPPV (adapted from reference 28). (**A**) The examiner stands at the patient's right side and rotates the patient's head 45 degrees to the right to align the right posterior semicircular canal with the sagittal plane of the body. (**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 slightly 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 benign paroxysmal positional vertigo. A presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown.

they may provoke a sudden onset of intense subjective vertigo, possibly with nausea, which will subside within 60 seconds. Because 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, in the supine position, the head can "hang" with support off the posterior edge of the examination table by about 20 degrees. The examiner should ensure that he can support the patient's head and guide the patient through the maneuver safely and securely, without the examiner losing support or balance himself.

1. The maneuver begins with the patient in the upright seated position with the examiner standing at the pa-

tient's side.[28] 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 degrees to the right and, with manual support, maintains the 45-degree head turn to the right during the next part of the maneuver.
3. Next, the examiner fairly quickly moves the patient (who is instructed to keep the eyes open) from the seated to the supine right-ear down position and then extends the patient's neck slightly (approximately 20 degrees below the horizontal plane) so that the patient's chin is pointed slightly upward, with the head hanging off the edge of the examining table and supported by the examiner. The examiner observes the patient's eyes for the latency, duration, and direction of the nystagmus.[40,41] 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 as to the presence of subjective vertigo.
4. After 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.
5. The Dix-Hallpike maneuver (steps 1-4) should then be repeated for the left side, with the left ear arriving at the dependent position.[38] Again, the examiner should inquire about subjective vertigo and identify objective nystagmus, when present. The examination of the left side completes the test.

The Dix-Hallpike maneuver is considered the gold standard test for the diagnosis of posterior canal BPPV.[19] It is the most common diagnostic criterion required for entry into clinical trials and for inclusion of such trials in meta-analyses.[42,43] 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 differ between specialty and nonspecialty clinicians. Lopez-Escamez et al[44] have reported a sensitivity of 82 percent and specificity of 71 percent for the Dix-Hallpike maneuvers in posterior canal BPPV, primarily among specialty clinicians. In the primary care setting, Hanley and O'Dowd[45] have reported a positive predictive value for a positive Dix-Hallpike test of 83 percent and a negative predictive value of 52 percent for the diagnosis of BPPV. Therefore, a negative Dix-Hallpike maneuver does not necessarily rule out a diagnosis of posterior canal BPPV. Because of the lower negative predictive values of the Dix-Hallpike maneuver, it has been suggested that this maneuver may need to be repeated at a separate visit to confirm the diagnosis and avoid a false-negative result.[38,46,47]

Factors that may affect the diagnostic accuracy of the Dix-Hallpike maneuver include the speed of movements during the test, time of day, and the angle of the plane of the occiput during the maneuver.[38] The Dix-Hallpike test must be done bilaterally to determine which ear is involved or if both ears are involved.[38] In a small percent 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.[2]

Although 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.[48] Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity.[30,48] Patients who are obese may be difficult for a single examiner to fully support throughout the maneuver, so additional assistance may be required. For patients with physical limitations, special tilting examination tables may allow the safe performance of the Dix-Hallpike maneuver.

Evidence Profile

- Aggregate evidence quality: Grade B, based on diagnostic studies with minor limitations
- Benefit: improved diagnostic accuracy and efficiency
- Harm: risk of provoking temporary symptoms of BPPV
- Cost: minimal
- Benefit-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 should not be relied upon for the diagnosis of posterior canal BPPV.)
- Role of patient preferences: minimal
- Patient exclusions: patients with physical limitations including cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity
- Policy level: strong recommendation

Statement 1b. Diagnosis of Lateral Canal BPPV

If the patient has a history compatible with BPPV and the Dix-Hallpike test is negative, the clinician should

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.*

Lateral canal BPPV (also called horizontal canal BPPV) is the second most common type of BPPV.[49-51] Because this type of BPPV has received considerably less attention in the literature, clinicians may be relatively unaware of its existence and the appropriate diagnostic maneuvers for lateral 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 meet diagnostic criteria for posterior canal BPPV should be investigated for lateral canal BPPV. In many instances, the presenting symptoms of lateral canal BPPV are indistinguishable from posterior canal BPP.[50]

Several studies have cited an incidence of approximately 10 to 15 percent in populations referred for evaluation and treatment of BPPV.[5,6,52-54] Furthermore, lateral canal BPPV may occur following performance of the PRMs (eg, Epley maneuver) for an initial diagnosis of posterior canal BPPV. This transition from posterior canal BPPV to lateral canal BPPV is thought to occur as free-floating particulate material migrates from the posterior canal to the lateral canal (so-called canal switch). Because this type of transition is relatively common, clinicians should be aware of lateral canal BPPV and its diagnosis.[5]

The supine roll test is the preferred maneuver to diagnose lateral canal BPPV.[6,51,55] Clinicians should inform the patient that this test is a provocative maneuver and may cause the patient to become subjectively intensely dizzy for a short period of time. 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 degrees to one side with the clinician observing the patient's eyes for nystagmus (Fig 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 quickly turned 90 degrees to the opposite side, and the eyes are once again observed for nystagmus. Two potential nystagmus findings may occur with this maneuver, reflecting two types of lateral canal BPPV.[5,55,56]

- *Geotropic type:* In most cases of lateral canal BPPV, rotation to the pathological side causes a very intense horizontal nystagmus beating toward the undermost (affected) ear, known as geotropic nystagmus (ie, nystagmus with a fast component toward the ground). When the patient is rolled to the other, healthy side, there is a less intense horizontal nystagmus, again beating toward the undermost ear (again geotropic; the direction of the nystagmus has now changed).
- *Apogeotropic type:* In less common cases, performance of 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.

In both types of lateral canal BPPV, the affected ear is presumed to be the ear to which the side of rotation pro-

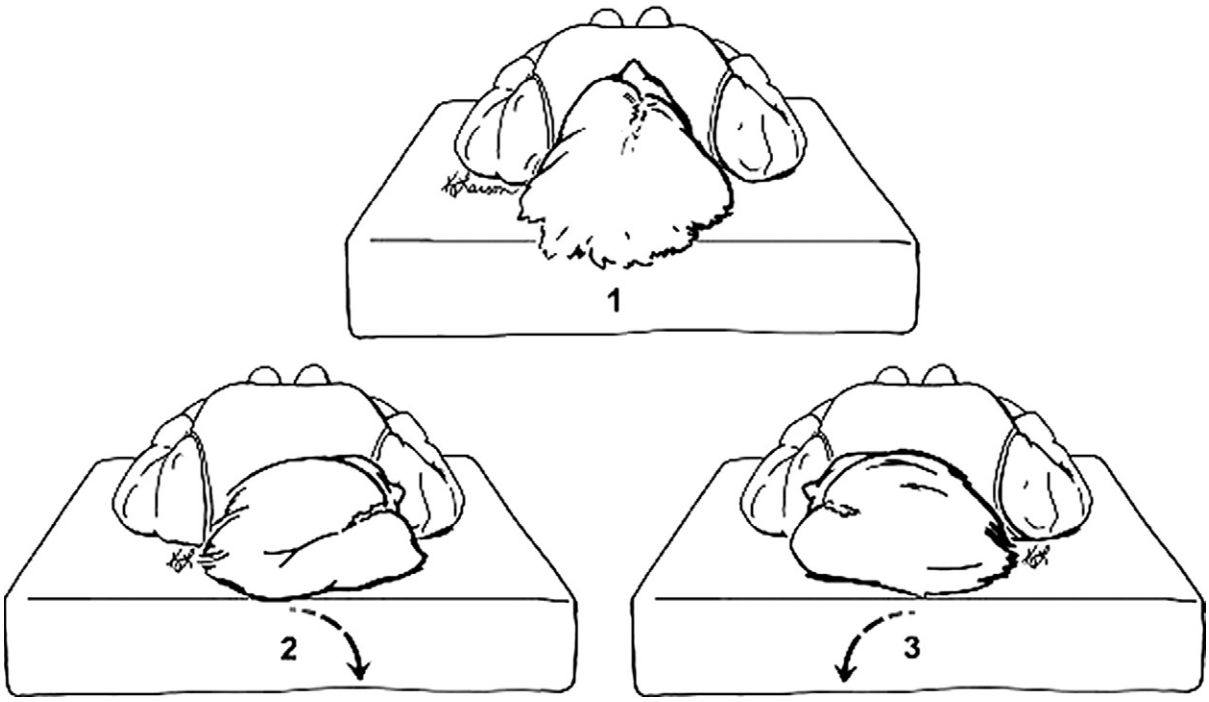

Figure 2 Diagrammatic views of the supine roll test. (**1**) The patient is in the starting neutral position. The patient's head is turned rapidly to the right side (**2**) to examine for characteristic nystagmus. Then the head is returned to the face-up position (**1**), allowing all nystagmus to subside, and then turned rapidly to the left side (**3**) to examine once again for nystagmus. (Adapted from reference 19.)

duces the most intense nystagmus.[53,55,57] Between the two types of lateral canal BPPV, the geotropic variant predominates.[50,55,58] Not uncommonly, because of CNS adaptation, the initially intense nystagmus may spontaneously change direction without rolling toward the opposite ear.[56]

The supine roll test has not received as much widespread use or diagnostic validation as the Dix-Hallpike maneuver. Review of the literature reveals that the sensitivity and specificity of the supine roll test in the diagnosis of lateral canal BPPV have not been determined. The lack of a more accurate, commonly accepted (gold standard) test for the diagnosis of lateral canal BPPV may be responsible, in part, for the absence of data for these statistical measures. A positive supine roll test, however, is the most commonly required and consistent diagnostic entry criterion for therapeutic trials of lateral canal BPPV.[50,53]

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 from the population under study. Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity.[30,48] The benefit of performing the supine roll test is that it allows clinicians to confirm a diagnosis of lateral canal BPPV quickly and efficiently.[5,19] 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 canal BPPV might be diagnostically missed if only traditional Dix-Hallpike testing was done. Further benefit might be derived from the supine roll test by decreasing the need to perform potentially unnecessary or unhelpful diagnostic testing.

Evidence Profile

- Aggregate evidence quality: Grade C, based on observational studies with limitations and selected populations
- Benefit: avoidance of a false-negative result in the diagnosis of BPPV attributable to a missed lateral canal variant; allowance of confirmation of a diagnosis of lateral canal BPPV, thereby avoiding unnecessary diagnostic tests.
- Harm: risk of provoking temporary symptoms of BPPV
- Cost: minimal
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: the importance of evaluating additional variants of BPPV rather than limiting the evaluation to posterior canal BPPV
- Role of patient preferences: minimal
- Exclusions: patients with physical limitations including cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries
- Policy level: recommendation

2a. Differential Diagnosis of BPPV

Clinicians should differentiate BPPV from other causes of imbalance, dizziness, and vertigo. *Recommendation based on observational studies and a preponderance of benefit over harm.*

Despite being the most common cause of peripheral vertigo,[59] BPPV is still often underdiagnosed or misdiagnosed.[60] Other causes of vertigo that may be confused with BPPV can be divided into otological, neurological, and other entities. In a nonspecialty setting evaluation of patients presenting with vertigo, BPPV has been found to account for 42 percent of cases followed by vestibular neuritis (41%), Ménière's disease (10%), vascular causes (3%), and other causes (3%).[45] In subspecialty settings, Ménière's disease may predominate (43% of cases), followed by BPPV (23%) and vestibular neuritis (26%).[61] The most common diagnoses that require distinction from BPPV are listed in Table 6. These conditions require distinction from BPPV because their natural history, treatment, and potential for serious medical sequelae differ significantly.

Otological Disorders

Other otological disorders causing vertigo may be differentiated from BPPV by their clinical characteristics including their temporal pattern and the presence or absence of hearing loss. Whereas BPPV is characterized by acute, discrete episodes of brief positional vertigo without associated hearing loss, other otological causes of vertigo manifest differ-

Table 6
Basic differential diagnosis of BPPV

Otological disorders	Neurological disorders	Other entities
Ménière's disease	Migraine-associated dizziness	Anxiety or panic disorder
Vestibular neuritis	Vertebrobasilar insufficiency	Cervicogenic vertigo
Labyrinthitis	Demyelinating diseases	Medication side effects
Superior canal dehiscence syndrome	CNS lesions	Postural hypotension
Posttraumatic vertigo		

ent temporal patterns and may additionally demonstrate associated hearing loss.[61]

In distinction to BPPV, Ménière's disease is characterized by discrete episodic attacks, with each attack exhibiting a characteristic triad of sustained vertigo, fluctuating hearing loss, and tinnitus.[4,62] As opposed to BPPV, the duration of vertigo in an episode of Ménière's disease typically lasts longer (usually on the order of hours) and is typically more disabling owing to both severity and duration. 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.[63] Protracted nausea and vomiting are also more common during an attack of Ménière's disease.

Acute peripheral vestibular dysfunction syndromes, 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 result.[64] 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.[61,65,66] 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, which are also typically sustained along with the vertigo.

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.[67] Similar to perilymphatic fistula, it differs from BPPV in that vertigo is induced by pressure changes and not position changes. SCD may also present with an associated conductive hearing loss and is diagnosed through CT of the temporal bones.[68]

Posttraumatic vertigo can present with a variety of clinical manifestations including vertigo, disequilibrium, tinnitus, and headache.[69] Although BPPV is most often idiopathic, in specific cases, traumatic brain injury is associated with BPPV.[70] BPPV has been described as occurring in conjunction with or as a sequelae to other vestibular disorders as well, such as Ménière's disease and vestibular neuritis.[71] Therefore, clinicians must consider the possibility of more than one vestibular disorder being present in any patient who does not clearly have the specific symptoms of a single vestibular entity.

Neurological 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 distinction may be difficult, several clinical features may suggest a central cause of vertigo rather than BPPV.[72,73] Nystagmus findings that more strongly suggest a neurological cause for vertigo, rather than a peripheral cause such as BPPV, include down-beating nystagmus on the Dix-Hallpike maneuver, direction-changing nystagmus occurring without changes in head position (ie, periodic alternating nystagmus), or baseline nystagmus manifesting without provocative maneuvers. Among the central causes of vertigo that should be distinguished from BPPV are migraine-associated vertigo, vertebrobasilar insufficiency, and intracranial tumors.

Migraine-associated vertigo has been described as a common cause of vertigo in the adult population[74] and may account for as many as 14 percent of cases of vertigo.[61] Diagnostic criteria include 1) episodic vestibular symptoms; 2) migraine according to International Headache Society criteria; 3) at least two of the following migraine symptoms during at least two vertiginous episodes: migrainous headache, photophobia, phonophobia, or visual or other aura; and 4) other causes ruled out by appropriate investigations.[75] Migraine-associated vertigo is heterogeneous in that both central disorders and peripheral disorders have been described, although more often it is believed to be central in nature.[76,77] It is distinguishable from BPPV by virtue of the necessary migraine/headache components, which are not associated with classic BPPV.

Several reports have suggested that isolated attacks of vertigo can be the initial and only symptom of vertebrobasilar insufficiency.[78-80] Isolated transient vertigo may precede a stroke in the vertebrobasilar artery by weeks or months. The attacks of vertigo in vertebrobasilar insufficiency usually last less then 30 minutes and have no associated hearing loss. The type of nystagmus (typically gaze-evoked in central lesions), the severity of postural instability, and the presence of additional neurological signs are the main distinguishing features between vertebrobasilar insufficiency and BPPV.[81] In addition, the nystagmus arising in vertebrobasilar insufficiency does not fatigue and is not easily suppressed by gaze fixation, helping to separate this diagnosis from BPPV.

Intracranial tumors and other brain stem lesions may rarely present with a history and symptomatology similar to those of BPPV.[82] In these cases, associated symptoms such as tinnitus, aural fullness, new-onset hearing loss, and/or other neurological symptoms should help differentiate these diagnoses from BPPV. Atypical nystagmus during Dix-Hallpike testing (eg, sustained down-beating nystagmus) argues against BPPV and suggests a more serious cause. Finally, failure to respond to conservative management such as the PRM or vestibular rehabilitation should raise concern that the underlying diagnosis may not be BPPV.[82]

Other Disorders

Several other non-otological and non-neurological disorders may present similarly to BPPV. Patients with panic disor-

der, anxiety disorder, or agoraphobia may complain of symptoms of lightheadedness and dizziness. Although these symptoms are usually attributed to hyperventilation, other studies have shown high prevalences of vestibular dysfunction in these patients.[83,84] These conditions may also mimic BPPV. Several medications, such as Mysoline, carbamazepine, phenytoin, antihypertensive medications, 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.[85] Cervical vertigo may produce symptoms similar to those of BPPV owing to proprioceptive abnormalities arising from cervical spine dysfunction.[86] 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). Postural hypotension also may produce episodic dizziness or vertigo. The dizziness or vertigo in postural hypotension, however, is provoked by moving from the supine 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 on the basis of 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.

Evidence Profile

- Aggregate evidence quality: Grade C, based on observational studies with limitations
- Benefit: prevention of false-positive diagnosis of BPPV when another condition actually exists
- Harm: none
- Cost: minimal
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: none
- Role of patient preferences: minimal
- Policy level: recommendation

Statement 2b. Modifying Factors

Clinicians should question patients with BPPV for factors that modify management including impaired mobility or balance, CNS disorders, a lack of home support, and increased risk for falling. *Recommendation based on observational and cross-sectional studies and a preponderance of benefit over harm.*

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 outcome of BPPV. Assessment of the patient with BPPV for factors that modify management is essential for improved treatment outcomes and ensuring patient safety with an underlying diagnosis of BPPV. 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.[87]

Given that BPPV occurs most commonly in the second half of the lifespan and its prevalence increases with age, patients suffering from BPPV often have medical comorbidities that may alter the management of BPPV.[16] In cross-sectional surveys, patients with BPPV demonstrate higher rates of diabetes, history of head trauma, and anxiety.[88] Other studies have also found higher relative rates of migraine (34% in BPPV patients vs 10% in non-dizziness control group), history of stroke (10% in BPPV patients vs 1% in controls), diabetes (14% vs 5%), and hypertension (52% vs 22%).[11] 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 syndromes in general is the risk for falls and resultant injury.[89] In multiple studies concerning etiology of falls, dizziness and vertigo were deemed the primary etiology for 13 percent of falls, compared with existing balance and gait problems (17%) and person-environment interactions (31%).[90] In a study by Oghalai,[15] 9 percent 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. Thus, evaluation of patients with a diagnosis of BPPV should also include an assessment of risk for falls.[16] In particular, elderly patients will be more statistically at risk for falls with BPPV. Clinicians may use various fall assessment tools to determine the patient's fall risk and appropriate precautionary recommendations.[87]

As noted above, comorbid conditions that occur commonly with BPPV such as a history of stroke or diabetes should also be identified during evaluation of patients with BPPV. Patients with a history of stroke or a history of diabetes, particularly with peripheral neuropathy, may already have preexisting gait, balance, or proprioceptive deficit.[91-93] The additional symptoms of BPPV may increase their risk for fall and injury. Patients with visual disturbances often lack the ability to correct for or compensate for a balance deficit with visual cues, and may also be at increased risk for falls. Associations between osteopenia and osteoporosis and BPPV have been reported.[94] Patients with both osteoporosis and BPPV 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 one of the potential fall-precipitating diagnoses.[95]

BPPV may occur in the setting of other CNS disorders. Patients should be questioned as to the presence of preex-

isting CNS disorders that may modify the management of BPPV. BPPV may occur relatively commonly after trauma or traumatic brain injury.[2,96] Posttraumatic BPPV is most likely to involve the posterior semicircular canal, and studies indicate that posttraumatic BPPV is significantly more likely to require repeated physical treatments (up to 67% of cases) for resolution compared with nontraumatic forms (14% of cases).[97] In rare instances, posttraumatic BPPV may be bilateral.[2] 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.[96] Although dizziness in the setting of multiple sclerosis may have a wide 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 PRM.[98,99] This study suggests that patients with BPPV and an underlying CNS disorder may be successfully diagnosed and treated with conventional methods for BPPV.

Finally, in a small percentage of cases, refractory or persistent BPPV may create difficulties from a psychological and/or social-functional perspective for affected individuals.[100,101] Outcomes studies have shown that patients with BPPV exhibit a significant negative quality-of-life impact from the diagnosis compared with the normative population in multiple subscales of the Short Form-36.[101,102] Patients who have preexisting comorbid conditions may require additional home supervision in the setting of BPPV.[30] This supervision may include counseling about the risk of falling at home or a home safety assessment. In rare cases, patients disabled by BPPV-related vertigo, especially if chronic or refractory, may need home assistance or temporary nursing home placement for their safety.

Evidence Profile

- Aggregate evidence quality: Grade C, based on observational and cross-sectional studies
- Benefit: allowance for global management of patients with BPPV with appropriately structured comprehensive treatment plan; identification of patients at risk for falls and prevention of fall-related injury
- Harm: none
- Cost: none
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: the management of BPPV will benefit from assessment of these modifying factors
- Role of patient preferences: minimal
- Policy level: recommendation

Statement 3a. Radiographic and Vestibular Testing

Clinicians should not obtain radiographic imaging, vestibular testing, or either in a patient diagnosed with BPPV, unless the diagnosis is uncertain or there are additional symptoms or signs unrelated to BPPV that warrant testing. *Recommendation against based on diagnostic studies with limitations and a preponderance of benefit over harm.*

The diagnosis of BPPV is based on the clinical history and physical examination. Routine radiographic imaging or vestibular testing is unnecessary in patients who already meet clinical criteria for the diagnosis of BPPV (Table 5). Further radiographic or vestibular testing may have a role in the 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 otological disorder.

Radiographic Imaging

Radiographic imaging, most commonly CNS imaging using magnetic resonance or CT techniques, is commonly obtained in the evaluation of a primary symptom complaint of vertigo. However, imaging is not useful in the routine diagnosis of BPPV because there are no radiological findings characteristic of or diagnostic for BPPV.[103,104] The lack of characteristic findings 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.[8] On a broader scale, previous retrospective reviews of elderly patients with dizziness failed to detect any significant differences in cranial MRI findings when comparing dizzy versus non-dizzy patients.[105,106]

Radiographic imaging of the CNS should be reserved for patients who present with a clinical history compatible with BPPV but who also demonstrate additional neurological symptoms atypical for BPPV. Radiographic imaging may also be considered for patients with suspected BPPV but inconclusive positional testing, or in patients with other neurological signs on physical examination that are not typically associated with BPPV. Such symptoms include abnormal cranial nerve findings, visual disturbances, and severe headache, among others. It should be noted that intracranial lesions causing vertigo are rare.[3] Potential lesions causing vertigo identifiable on CNS imaging include cerebrovascular disease, demyelinating disease, or an intracranial mass; they are most often located in the brain stem cerebellum, thalamus, or cortex.[3] In small case series, positional vertigo and nystagmus have been associated with neurovascular compression of cranial nerve VIII, vestibular schwannoma, Arnold Chiari malformation, and a variety of cerebellar disorders.[107-109]

In distinction to standard BPPV, such conditions are quite rare and typically present with additional neurological symptoms in conjunction with the vertigo. Routine neuroimaging has not been recommended to discern these conditions from the more common causes of vertigo.[110] The costs of routine imaging in cases of BPPV are not justified given that diagnostic neuroimaging does not improve the diagnostic accuracy in the vast majority of BPPV cases.

Therefore, neuroimaging should not be routinely used to confirm the diagnosis of BPPV.

Vestibular Function Testing

When patients meet clinical criteria for the diagnosis of BPPV (Table 5), no additional diagnostic benefit is obtained from vestibular function testing. Vestibular function testing is indicated when the diagnosis of a vertiginous or dizziness syndrome is unclear or possibly when the patient remains symptomatic following treatment. It may also be beneficial when multiple concurrent peripheral vestibular disorders are suspected.[4,65,111]

Vestibular function testing involves a battery of specialized tests that primarily record nystagmus in response to labyrinthine stimulation and/or voluntary eye movements. Most vestibular function testing relies on the neurological relationship between the regulation of eye movement and the balance organs: the vestibular-ocular reflex. These tests are useful in the evaluation of vestibular disorders that may not be evident from the history and clinical examination, and may provide information for quantification, prognostication, and treatment planning.[112]

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.[113] Some or all of these test elements may be included in a vestibular test battery.

In cases of BPPV in which the nystagmus findings are suggestive but not clear, it may be beneficial to use videooculographic recordings of nystagmus associated with posterior canal BPPV, because the eye can be enlarged on a screen for detail, and the image 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 percent in patients with diagnoses suspicious for BPPV.[114]

BPPV is relatively frequently associated with additional vestibular pathology. Symptoms associated with chronic vestibular function 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 percent of BPPV patients.[4,115,116] This percentage, however, is higher than what might be expected in the nonspecialty population. Vestibular disorders that have been associated with BPPV include Ménière's disease, viral vestibular neuritis, or labyrinthitis.[71,117] 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, PRMs appear to be equally effective in resolving the positional nystagmus associated with BPPV, but complete symptom resolution is significantly less likely in those patients with additional vestibular pathology. In one study, 86 percent of patients with BPPV but without associated vestibular pathology reported complete resolution of symptoms after PRMs versus only 37 percent reporting complete resolution when additional vestibular pathology was present.[118] Thus, patients with suspected associated vestibular pathology in addition to BPPV may be a subset who would benefit from the additional information obtained from vestibular function testing. Similarly, up to 25 percent of patients with separate recurrences of BPPV are more likely to have associated vestibular pathology[119]; therefore, patients with recurrent BPPV may be candidates for vestibular function testing.

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 in these cases, vestibular testing adds significant cost to the diagnosis and management of BPPV, and the information obtained does not alter the subsequent management of BPPV in the vast majority of the cases. Therefore, vestibular function testing should not be routinely obtained when the diagnosis of BPPV has already been confirmed by clinical diagnostic criteria. Vestibular function testing, however, may be warranted in patients with 1) atypical nystagmus, 2) suspected additional vestibular pathology, 3) a failed (or repeatedly failed) response to CRP, or 4) frequent recurrences of BPPV.[120,121]

Evidence Profile

- Aggregate evidence quality: Grade C, based on diagnostic studies with limitations in referred patient populations and observational studies for vestibular testing; Grade C, based on observational studies for radiographic imaging
- Benefit: facilitation of prompt treatment by avoiding unnecessary testing associated with low yield and potential false-positive diagnoses; avoidance of radiation exposure and adverse reactions to testing
- Harm: potential missed diagnosis of comorbid conditions; discomfort such as nausea and vomiting produced by vestibular testing
- Cost: cost savings associated with decreased testing
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: importance of reducing unnecessary testing and delays in diagnosis
- Role of patient preferences: minimal

- Exclusions: patients who have separate indications for radiographic or vestibular testing aside from confirmation of a diagnosis of BPPV
- Policy level: recommendation against

Statement 3b. Audiometric Testing

No recommendation is made concerning audiometric testing in patients diagnosed with BPPV. *No recommendation based on insufficient evidence for the diagnostic or prognostic value of audiometry in the evaluation of BPPV.*

Audiometry is the most commonly obtained objective test of hearing. Recent Medicare data indicate that approximately 9 percent of audiograms obtained annually are ordered in association with diagnostic categories related to vertigo (International Classification of Diseases, Version 9 codes: 386 and/or 780.4).[122] Specialty clinicians with access to audiometry frequently obtain audiometry as part of the evaluation of vertigo in contradistinction to nonspecialty clinicians. However, limited diagnostic cohort studies and cost-effectiveness studies supporting this practice are available.

Audiometry is not required to diagnose BPPV; however, audiometry may offer some diagnostic benefit for patients in whom the clinical diagnosis of BPPV is unclear. Both hearing loss and BPPV are more prevalent in older patients. Therefore, BPPV and some degree of hearing loss (likely long-standing, as in presbyacousis) are likely to coexist in patients with BPPV.[123] From a pathophysiological standpoint, a preexisting, stable hearing loss should be unrelated to and not influence the diagnosis of BPPV. In such cases, routine audiometry is unlikely to reinforce or influence the diagnosis of BPPV.

In the majority of cohort studies of BPPV, audiometric studies, when obtained, have been largely normal. In some of these studies, however, the inclusion criteria for a diagnosis of BPPV included no history of antecedent hearing loss.[124] In two algorithmic studies, audiometry was found to be cost-effective and diagnostically effective in the broad evaluation of patients with vertigo.[61,111] In a study of 192 patients referred to an academic center for the evaluation of vertigo, Stewart et al[125] found that the audiogram was the most cost-effective test among various studies including electronystagmography, posturography, MRI, and blood tests. Notably, however, the cost-effectiveness (diagnostic benefit) of the history and physical examination (ie, Dix-Hallpike maneuver or supine role test) was not directly studied. This diagnostic focus notably differs from the current guideline, which emphasizes the value of the clinical history and physical examination.

In a study of 564 cases, Kentala et al[66] found in a diagnostic algorithm analysis that the presence of a normal audiogram was corroborating for a diagnosis of BPPV, distinguishing BPPV from other associated conditions such as Ménière's disease, vestibular schwannoma, and so on. However, the panel felt that distinction from such associated conditions could be made accurately and more cost-effectively on the basis of the history, rather than relying on audiometry. Upon review of the literature, no meaningful observational or diagnostic cohort studies either supporting or arguing against the use of audiometry in the diagnosis of the BPPV population was identified.

Traditional BPPV should not manifest with symptoms of a new-onset hearing loss. A newly reported hearing loss arising in conjunction with vertigo suggests a diagnosis other than BPPV and such patients merit audiometry. Clinicians should distinguish patients with vertigo and new-onset hearing loss from those patients with preexisting otological disease who subsequently develop BPPV. As noted, studies have reported rates of associated otological or vestibular pathology in 30 to 50 percent of cases in referred populations with BPPV.[4,115,116] In cases with preexisting otological disease and a diagnostic concern for BPPV, audiometry may help establish the independent stability of the otological disease, thereby helping to confirm a diagnosis of BPPV.

Audiometry is a noninvasive test with widespread availability and no reported harms from testing. The potential benefits of obtaining audiometry in the evaluation of BPPV include the ability to establish baseline stability or, alternatively, to help rule out other otological conditions such as Ménière's disease or labyrinthitis.[66] The primary disadvantage of routinely obtaining audiometry in patients undergoing evaluation for BPPV is clearly the cost to the health care system. In the vast majority of cases of BPPV with stable hearing by history, the audiogram is most likely to be normal or demonstrate an age-appropriate sensorineural hearing loss and, therefore, likely will not influence the diagnosis of BPPV. Overall, insufficient evidence exists to either confirm or disaffirm the value of routine audiometry in the initial assessment of BPPV.

Evidence Profile

- Aggregate evidence quality: Grade D, based on expert opinion specifically in the BPPV population and an absence of diagnostic studies on audiometry in BPPV
- Benefit: possible identification of an unsuspected hearing loss or an underlying otological condition
- Harm: delay in treatment if audiometry is not readily available
- Cost: possible realization of cost savings if fewer audiograms are performed
- Benefit-harm assessment: relative balance of benefit and harm
- Value judgments: Ease of identification of a small subset of patients in whom audiometry might be valuable on the basis of the clinical history
- Role of patient preferences: minimal
- Policy level: no recommendation

Statement 4a. Repositioning Maneuvers as Initial Therapy

Clinicians should treat patients with posterior canal BPPV with a particle repositioning maneuver. *Recom-*

mendation based on randomized controlled trials with small sample sizes and heterogeneity conducted in specialty practice settings and a preponderance of benefit over harm.

Although it has been historically commonplace to reassure patients diagnosed with BPPV that their condition is benign and is likely to spontaneously remit in the subsequent months, recent relatively high-quality evidence supports active, expeditious treatment with a particle repositioning maneuver (PRM). Treatment with PRMs consistently eliminates the vertigo due to BPPV, improves quality of life, and reduces the risks of falling.

Posterior Canal BPPV Treatments

Two types of PRMs have been found effective for posterior canal BPPV: 1) the canalith repositioning procedure (CRP, also referred to as the Epley maneuver) and 2) the liberatory maneuver (also called the Semont maneuver). Other PRMs have been proposed for the treatment of posterior canal BPPV, but high-quality, reproducible data that demonstrate their clinical efficacies are lacking.

Treatment with canalith repositioning procedure. CRP was first described by Epley in 1992.[126] Through a series of head position changes, the CRP moves the canaliths from the posterior semicircular canal to the vestibule, thereby relieving the stimulus from the semicircular canal that had been producing the vertigo in BPPV.

CRP is most commonly performed in the outpatient setting by a clinician after confirmation of the diagnosis of posterior canal BPPV.[19] Patients should be informed that nausea, occasional vomiting, and/or a sense of falling may arise during the CRP.[127] Patients who previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuver may be considered for antiemetic prophylaxis during the CRP. Figure 3 depicts the CRP for posterior canal BPPV.

Several RCTs have been published evaluating the efficacy of the CRP in the treatment of posterior canal BPPV. A number of these are high-quality RCTs, three of which have been included in a relatively recent Cochrane collaborative review of the Epley maneuver for BPPV.[42,59,128,129] The Cochrane review identified a statistically significant effect in favor of the CRP compared with controls. An odds ratio of 4.2 (95% confidence interval, 2.0-9.1) was found in favor of treatment for subjective symptom resolution in posterior canal BPPV; an odds ratio of 5.1 (95% confidence interval, 2.3-11.4) was found in favor of treatment for conversion of a positive to negative Dix-Hallpike test.

Subsequently, additional RCTs have been published regarding the CRP, reflecting similar results. Table 7 summarizes recent RCTs evaluating CRP for posterior canal BPPV. Of note, consistent with the expected spontaneous resolution of posterior canal BPPV over time, treatment effects between CRP and control patients tended to diminish over time. In the short term, typically at 1 week, the CRP is very effective at providing symptom resolution for posterior canal BPPV with small numbers needed to treat (NNT).

All but one of the RCTs for CRP has taken place in the specialized clinic setting, most commonly with a referred population, which may limit the generalizability of these results. In the only RCT conducted in the primary care setting, investigators were unable to demonstrate a significant benefit for the CRP based on symptomatic outcome.[130] At 1 week follow-up, 31.6 percent (12/38) of CRP patients demonstrated symptom resolution versus 24.4 percent (10/41) of sham patients ($P = 0.48$). Objectively, however, 34.2 percent of CRP-treated patients converted to a negative Dix-Hallpike at 1 week, versus 14.6 percent in the sham group ($P = 0.04$). Although statistically significant, this objective conversion rate is still lower than those reported among RCTs in the specialty setting (typically ranging from 66%-89%).[42] Because both the symptomatic response rates and 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. Reasons for discrepancy between primary care and specialty settings may include differences in performance of the CRP (ie, a single maneuver vs repeated maneuvers at the same visit), intrinsic patient variability with comorbid balance disorders, differences in symptom reporting, or combinations thereof.

The positive treatment results of the CRP have also been demonstrated in lesser quality nonrandomized trials and case series.[131-137] In addition to the Cochrane review, four meta-analyses have been reported.[41,138-140] Each analysis concluded that the CRP is significantly more effective than placebo in posterior canal BPPV. Among these trials, however, significant heterogeneity has also been demonstrated.[140]

Many trials also report a secondary outcome of conversion from a positive to negative Dix-Hallpike maneuver after CRP. The odds ratios for this more objective measure of resolution for posterior canal BPPV range from 3.2 to 22 across studies, similar to reported rates of symptom resolution.[42] In most nonrandomized case series assessing treatment response, symptom resolution is the only commonly reported outcome measure for the CRP.

Considerable variability exists in terms of the number of times the CRP is applied for the initial treatment of BPPV, even across RCTs.[59,128,129] Some investigators perform only one 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.[128] Even further variability exists among published case series for CRP.[141-143] On the basis of a review of the literature, it was not possible to determine the optimal number of cycles for the CRP or a protocol for repeated procedures. The repeated application of the CRP is likely to be determined by the severity of the symptoms, if they persist; clinician availability; and the clinician's historical success with the CRP.

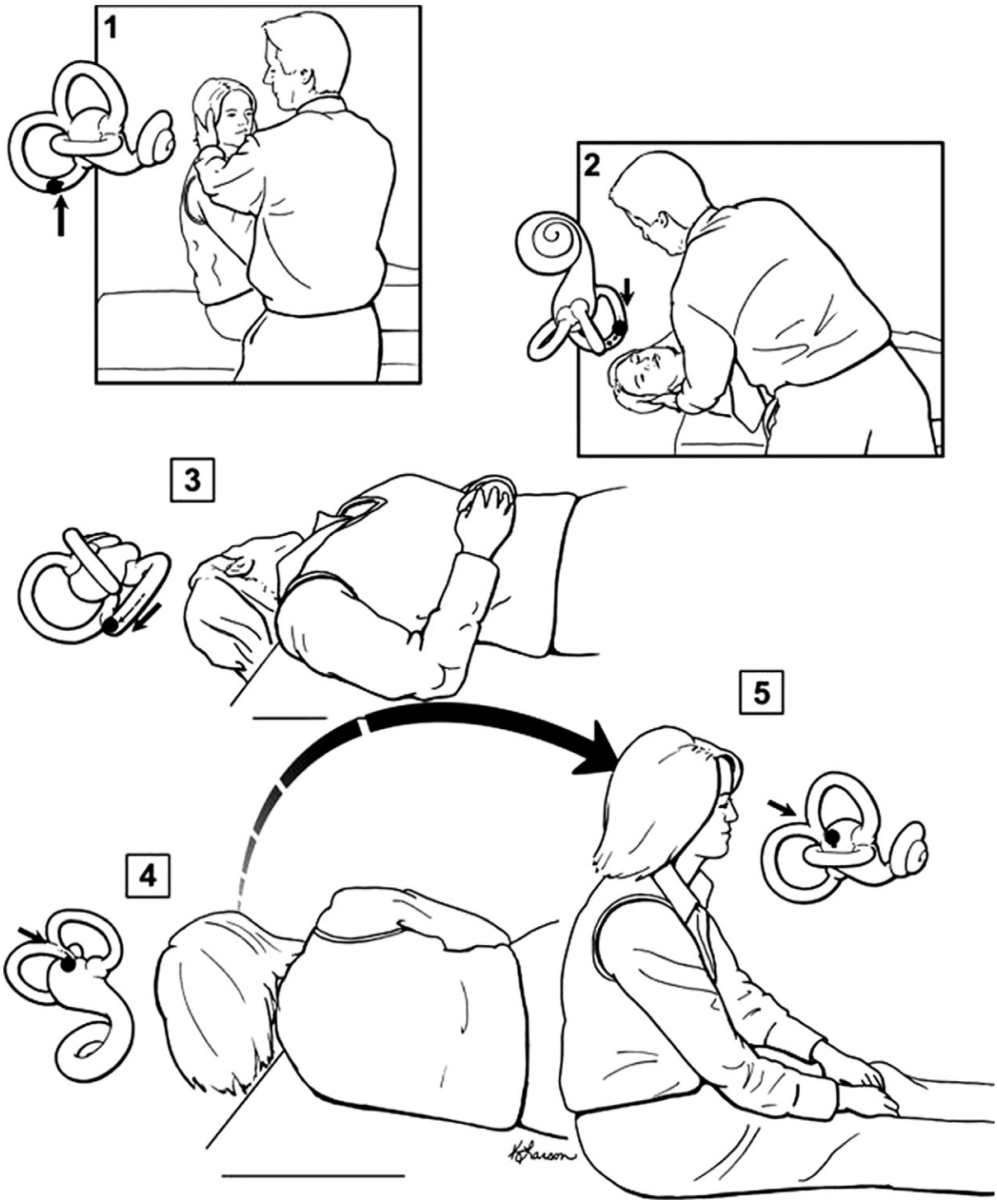

Figure 3 Performance of the therapeutic canalith repositioning procedure for right-sided posterior canal BPPV. (Adapted from reference 19.) (**1**) The patient is placed in the upright position with the head turned 45 degrees toward the affected ear (the ear that was positive on the Dix-Hallpike testing). (**2**) The patient is rapidly laid back to the supine head-hanging position, which is then maintained for 20 to 30 seconds. (**3**) Next, the head is turned 90 degrees toward the other (unaffected) side and held for about 20 seconds. (**4**) Following this rotation, the head is turned a further 90 degrees (usually necessitating the patient's body to also move from the supine position to the lateral decubitus position) such that the patient' head is nearly in the facedown position. This position is also held for 20 to 30 seconds. (**5**) The patient is then brought into the upright sitting position, completing the maneuver.

With respect to complications of treatment, CRP is associated with mild and generally self-limiting adverse effects in about 12 percent of those treated.[19] 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

Table 7
Randomized controlled trials evaluating the effectiveness of CRP for posterior canal BPPV

Reference	Improved in treatment group n/N (%)	Improved in control group n/N (%)	Endpoint	Time to assessment	*P* value	Odds ratio (95% CI)	NNT
Lynn 1995[128]	11/18 (61%)	3/20 (15%)	Vertigo resolution	2 weeks	0.033	6.3 (1.29-30.5)	2.2
Froehling 2000[59]	12/24 (50%)	5/26 (19%)	Vertigo resolution	1-2 weeks	0.020	4.2 (1.2-14.8)	3.3
Simhadri 2003[177]	19/20 (95%)	3/20 (15%)	Vertigo resolution	1 week	0.001	107.7 (10.2-1135.5)	1.3
	19/20 (95%)	3/20 (%)		4 weeks	0.001	107.7 (10.2-1135.5)	1.3
Yimtae 2003[129]	12/29 (41%)	1/27 (4%)	Vertigo resolution	1 week	0.005	18.4 (2.2-154.4)	2.7
	16/25 (64%)	7/20 (35%)		4 weeks	0.336	3.3 (1.0-11.3)	3.4
Cohen 2005[43]	*/24 (CRP)	*/25 (CRP)	Vertigo frequency scale (0-10)	4 weeks†	0.021		
	*/25 (LM)	*/25 (LM)		4 weeks†	0.010		
von Brevern 2006[159]	28/35 (80%)	4/31 (13%)	Vertigo resolution	24 hours	0.001	27.0 (7.1-109.9)	1.5

CI, confidence interval; *CRP,* canalith repositioning procedure; *LM,* Semont's liberatory maneuver; *NNT,* number needed to treat.
*Responses were analyzed with multilevel methods and expressed as fitted linear regression graphs, so no discrete numerical expression of the response rates could be determined.
†Time to evaluation was varied, so data presented are based on fitted linear regression curves at 4 weeks.

canal BPPV during the course of treatment (so-called canal switch). Such a canal switch occurs in about 6 to 7 percent of those treated with CRP,[129,144] underscoring the importance of recognizing the lateral canal variant of BPPV. 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.[145]

Treatment with the liberatory (Semont's) maneuver. Clinical trials concerning the treatment effectiveness of the liberatory maneuver (Fig 4) are limited. One study,[43] which

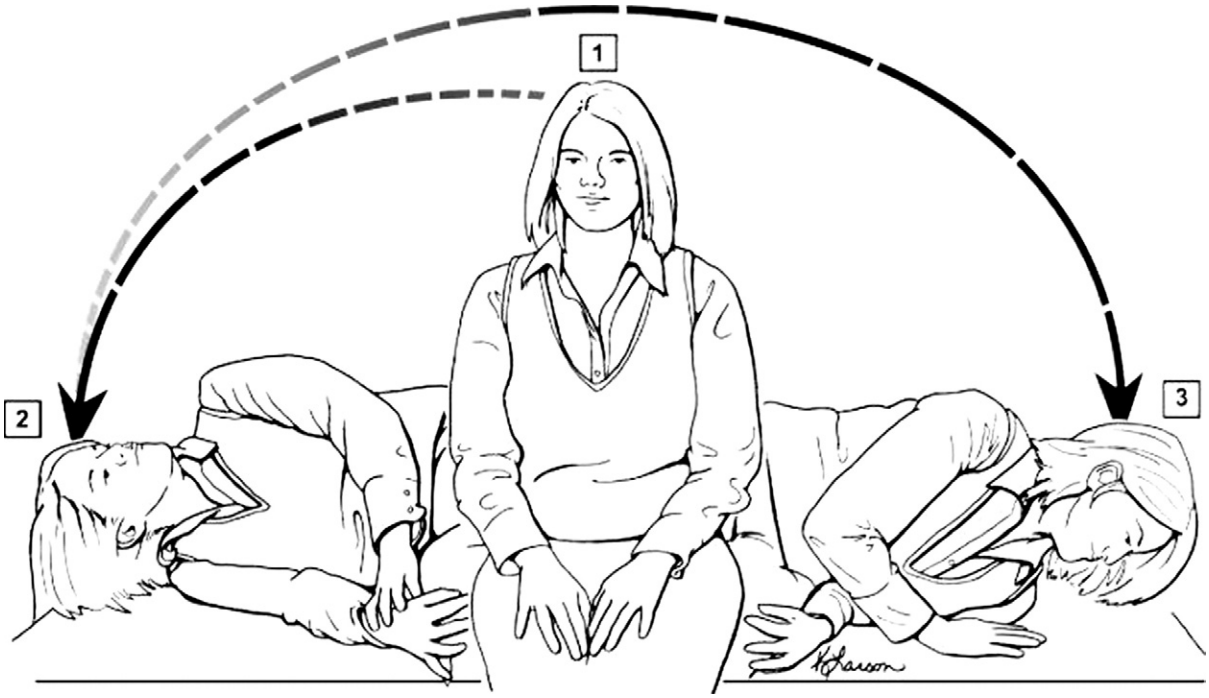

Figure 4 The Semont maneuver for right-sided BPPV. (**1**) Patient is seated in the upright position; then the patient's head is turned 45 degrees toward the left side, and the patient is then rapidly moved to the side-lying position as depicted in position (2). This position is held for approximately 30 seconds, and then the patient is rapidly moved to the opposite side-lying position without pausing in the sitting position and without changing the head position relative to the shoulder, resulting in position (3). This position is maintained for 30 seconds and then the patient gradually resumes the upright sitting position. (Adapted from reference 19.)

included a treatment arm with the Semont maneuver, demonstrated that this maneuver improved vertigo intensity more than the sham treatment ($P < 0.009$). A study by Salvinelli et al[146] randomized 156 patients to the Semont maneuver, flunarizine (a calcium channel blocker), or no treatment. At 6-month follow-up, symptom resolution occurred in 94.2 percent of patients treated with the Semont maneuver, 57.7 percent of patients treated with flunarizine, and 34.6 percent of untreated patients. Soto Varela et al[147] randomized patients to treatment with CRP, Semont maneuver, or Brandt-Daroff exercises. Symptom resolution among those treated with either CRP or Semont maneuver at 1 week was the same (74% vs 71%) but only 24 percent for Brandt-Daroff exercises. At 3-month follow-up, however, patients treated with CRP demonstrated superior outcomes compared with those treated with Semont maneuver ($P = 0.027$).

In conclusion, the Semont maneuver is more effective than no treatment or Brandt-Daroff exercises in relieving symptoms of posterior canal BPPV, according to studies with small sample sizes and limitations. No adverse events have been reported in trials with the liberatory maneuver. Because of limited studies with direct comparisons between the liberatory maneuver and the CRP, no conclusions about differential effectiveness can be drawn.

Lateral (Horizontal) Canal BPPV Treatments

Lateral canal BPPV is usually unresponsive to CRPs used for posterior canal BPPV but may respond to other maneuvers intended to move canaliths from the lateral canal into the vestibule.[144,148,149] The roll maneuver (Lempert maneuver or barbecue roll maneuver) or its variations are the most commonly employed maneuvers for the treatment of lateral canal BPPV.[5,143] This maneuver involves rolling the patient 360 degrees in a series of steps to effect particle repositioning. It may be performed in the outpatient setting after a diagnosis of lateral canal BPPV has been made with the supine roll test.

Rather limited data exist with respect to the effectiveness of the roll maneuver in lateral canal BPPV treatment. Based primarily on cohort studies and case series, the effectiveness of the roll maneuver in treating lateral canal BPPV appears to be approximately 75 percent, although reported response rates vary widely from 50 percent to almost 100 percent.[5,19,55,56,58,143,148-152] Because lateral canal BPPV may spontaneously remit more quickly than other forms of BPPV, a control group is especially important in assessing treatment efficacy.[51,142]

Forced prolonged positioning is another treatment maneuver reported to be as effective in treating lateral canal BPPV. It may be performed either alone or concurrently with other maneuvers with a reported effectiveness of 75-90 percent based on case series.[58,150,152,153] Other lesser-known maneuvers such as the Gufoni maneuver and the Vannucchi-Asprella liberatory maneuver[151,154,155] have also been reported as effective in uncontrolled studies.

In conclusion, variations of the roll maneuver appear moderately effective and are the most widely used treatments for lateral canal BPPV. Other methods of treatment have also been advocated, but currently no RCTs provide reliable measures of effectiveness. At this time, there is insufficient evidence to recommend a preferred treatment maneuver for lateral canal BPPV treatment.

Self-Administration and Posttreatment Restrictions

Three studies have assessed patient self-treatment for BPPV. One study found slightly greater improvement in those patients given instructions for self-administered CRP at home after initial CRP in the office.[156] Self-administered CRP appeared to be more effective (64% improvement) than self-treatment with Brandt-Daroff exercises (23% improvement).[157] Another study reported 95 percent resolution of positional nystagmus 1 week after self-treatment with CRP compared with 58 percent in patients who self-treated using a modified Semont maneuver ($P < 0.001$).[158] No comparison studies have been published from which to make recommendations regarding self-treatment vs clinician-administered treatment of BPPV. In motivated individuals, self-treatment of BPPV may be an option.

Comparison of studies, in particular the treatment arms for RCTs, reveals similar response rates whether or not posttreatment positional or activity restrictions (ie, cervical collar or positional avoidance) are observed.[43,59,128,129,159] Two studies looking at posttreatment restrictions after CRP found no evident improvement in those given restrictions.[160,161] Another study found slight benefit in patients with post-activity restrictions, as measured by the number of maneuvers required to produce a negative Dix-Hallpike maneuver.[162] Overall, there is insufficient evidence to recommend post-maneuver restrictions in patients treated with CRP.

Evidence Profile

- Aggregate evidence quality: Grade B, based on RCTs with small sample sizes and significant heterogeneity (Most studies were conducted in specialty practice settings with limited data from other treatment settings, potentially limiting generalizability of results.
- Benefit: prompt resolution of symptoms with a relatively low NNT ranging from 1 to 3
- Harm: transient provocation of symptoms of BPPV by the maneuver; risk for falls due to imbalance after the procedure; no serious adverse events reported in RCTs
- Cost: cost of the procedure
- Benefit-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
- Role of patient preferences: limited

- Exclusions: patients with physical limitations including cervical stenosis, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, retinal detachment, and spinal cord injuries may not be candidates for this maneuver or may need specialized examination tables for performance of the maneuver
- Policy level: recommendation

Statement 4b. Vestibular Rehabilitation as Initial Therapy

The clinician may offer vestibular rehabilitation, either self-administered or with a clinician, for the initial treatment of BPPV. *Option based on controlled observational studies and a balance of benefit and harm.*

Overview of Vestibular Therapy

Vestibular rehabilitation is a form of physical therapy designed to promote habituation, adaptation, and compensation for deficits related to a wide variety of balance disorders. It may also be referred to as vestibular habituation, vestibular exercises, or vestibular therapy. There is no single specific protocol for vestibular rehabilitation, but rather a program of therapy is developed on the basis of the underlying diagnosis. Programs can include canalith repositioning exercises, adaptation exercises for gaze stabilization, habituation exercises, substitution training for visual or somatosensory input, postural control exercises, fall prevention training, relaxation training, conditioning exercises, functional skills retraining, and patient and family education.[163-165]

With respect to BPPV, vestibular rehabilitation programs most commonly focus on habituation exercises either in formal outpatient therapy programs or with home exercise programs. Vestibular rehabilitation programs may also include PRMs, but repositioning maneuvers will be covered separately in the guideline. Herein, we refer to vestibular rehabilitation as a series of exercises or training maneuvers performed by the patient for the treatment of BPPV with or without direct clinician supervision.

Vestibular rehabilitation habituation exercises were first described by Cawthorne and Cooksey in the 1940s.[166] These exercises consist of a series of eye, head, and body movements in a hierarchy of increasing difficulty, which provokes vestibular symptoms. The exercises begin with simple head movements, 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.

In 1980, Brandt and Daroff[167,168] described home repositioning exercises that involve a sequence of rapid lateral head/trunk tilts repeated serially to promote loosening and ultimately dispersion of debris toward the utricular cavity. In these exercises, the patient starts in a sitting position and moves quickly to the right-side lying position, with the head rotated 45 degrees and facing upward. This position is maintained for 30 seconds after the vertigo stops. The patient then moves rapidly to a left-side lying position, with the head rotated 45 degrees and facing upward. In early work with patients with BPPV, patients repeated these maneuvers moving from the sitting to side-lying position three times a day for 2 weeks while hospitalized and had excellent resolution of BPPV symptoms.[169]

Vestibular Rehabilitation as a Treatment of BPPV

Relatively few RCTs and case series have been published regarding the effectiveness of vestibular rehabilitation as the initial therapy for BPPV. In a prospective analysis of 25 consecutive patients with BPPV, Banfield et al[170] reported that patients demonstrate an excellent short-term response rate of 96 percent subjectively to vestibular rehabilitation treatment with an average of three clinic visits per patient, but the authors noted a significant recurrence rate of BPPV with long-term follow-up (mean follow-up 3.8 years). The authors cited one advantage of vestibular rehabilitation: the capability of patients to be self-reliant in their ability to return to habituation exercises should symptoms recur. In a controlled trial of 60 patients with BPPV comparing a PRM, vestibular rehabilitation exercises and no treatment, vestibular rehabilitation provided better resolution of vertigo compared with no treatment.[171] The PRM arm demonstrated resolution of symptoms with fewer treatments than those required for vestibular rehabilitation, although the relative improvements at 3-month follow-up were comparable.

Several studies have compared vestibular rehabilitation exercises to particle rehabilitation maneuvers in the treatment of posterior canal BPPV. In an RCT of 124 patients randomized to CRP, modified liberatory maneuver, sham maneuver, Brandt-Daroff exercises, and vestibular habituation exercises by Cohen, repositioning maneuvers were more effective than Brandt-Daroff exercises or habituation exercises.[43] Both types of vestibular rehabilitation treatments, however, were individually more effective than a sham intervention.[43,172] Soto Varela et al[147] comparatively analyzed a total of 106 BPPV patients randomly assigned to receive Brandt-Daroff habituation exercises, the Semont maneuver, or the Epley maneuver. At the 1-week follow-up, similar cure rates were obtained with the Semont and Epley maneuvers (74% and 71%, respectively), both cure rates being significantly higher than that obtained with Brandt-Daroff exercises (24%). At 3-month follow-up, the cure rate for the Brandt-Daroff exercises increased significantly to 62 percent, although the rate was still lower than that of PRMs. Other studies have demonstrated similar results for vestibular rehabilitation in BPPV.[28,173]

Vestibular rehabilitation is thought to improve long-term outcomes for BPPV. Although data are mixed, a few studies have indicated that use of vestibular rehabilitation may decrease recurrence rates for BPPV.[136,174] This protective

effect against recurrence of vestibular rehabilitation may be more pronounced in the elderly.[136]

Several prospective studies have demonstrated the safety and effectiveness of vestibular rehabilitation for unilateral peripheral vestibular disorders; the results are summarized in a recent Cochrane collaboration report.[172] Among 21 included randomized trials, there were no reports of adverse effects due to vestibular rehabilitation therapy. Current published evidence is inadequate to indicate superiority for one form of vestibular rehabilitation vs another. There is also not enough evidence to favor formal outpatient vestibular therapy performed with a clinician over independent home therapy.[175]

In summary, with respect to posterior canal BPPV, vestibular rehabilitation demonstrates superior treatment outcomes compared with placebo. In short-term evaluation, vestibular rehabilitation is less effective at producing complete symptom resolution than PRMs. With longer-term follow-up, however, its effectiveness approaches that of PRMs. Insufficient data exist concerning the response of lateral canal BPPV to vestibular therapy; this area needs further research.

Cost considerations may become important if repeated visits for clinician-supervised therapy are required as opposed to initial patient instruction followed by home-based therapy. Patients with certain comorbidities may not be appropriate candidates for vestibular rehabilitation or may need specialized, individually tailored vestibular rehabilitation protocols. Examples of such comorbidities include cervical stenosis, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries. On the other hand, patients with preexisting otological or neurological disorders may derive more benefit from vestibular rehabilitation as a treatment for BPPV.

Evidence Profile

- Aggregate evidence quality: Grade C, based on controlled observational studies and limited RCTs
- Benefit: potentially faster resolution of symptoms compared with observation alone
- Harm: no serious adverse events noted in published trials; transient provocation of BPPV symptoms during rehabilitation exercises; potential for delayed symptom resolution compared with PRMs as a sole intervention
- Cost: need for repeated visits if done with clinician supervision; cost of therapy
- Benefit-harm assessment: relative balance of benefits and harm
- Value judgments: vestibular rehabilitation considered possibly 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.)
- Role of patient preferences: substantial role for shared decision making
- Exclusions: patients with physical limitations such as cervical stenosis, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries
- Policy level: option

Statement 4c. Observation as Initial Therapy

Clinicians may offer observation as initial management for patients with BPPV and with assurance of follow-up. *Option based on data from cohort and observational studies with heterogeneity and a relative balance of benefits and harms.*

Observation may be defined as a "watchful waiting" or the withholding of specific therapeutic interventions for a given disease. Observation is often considered when the disease course is self-limited and/or felt to be benign with limited sequelae occurring from the withholding of therapy. In BPPV, observation implies that therapeutic interventions such as vestibular rehabilitation and/or PRMs will be withheld, anticipating a natural and spontaneous improvement of the symptoms of BPPV. Under a course of observation, patients may still be instructed to avoid provocative positions and activities where the risk of injury (ie, falls) may be increased until symptoms resolve spontaneously or until they are reassessed for symptom resolution.

To consider observation as an option in the management of BPPV, the clinician must determine the natural history of the BPPV. It has been presumed that the natural history of BPPV is one of eventual resolution in most patients. It should be noted, however, that an often quoted study by Blakley,[176] which reported high rates of spontaneous resolution of BPPV, relied on subjective symptom reporting, rather than objective testing with a Dix-Hallpike maneuver, as the outcome measure for resolution. It is believed that a significant fraction of patients reporting subjective improvement actually have reduction in symptoms secondary to avoiding provocative (vertigo-producing) positions rather than actual cure.[139] More recent RCTs have utilized objective testing with the Dix-Hallpike maneuver as an additional outcome measure to assess for objective resolution of BPPV. Notably, to observe proper blinding, most RCTs also use a sham positional maneuver in the control group, which theoretically may affect the natural history of BPPV.

In several studies, the spontaneous rate of symptomatic resolution of BPPV ranges from 15 to 86 percent. The reported rate of spontaneous improvement based on objective positional testing (ie, conversion to a negative Dix-Hallpike maneuver) ranges from 35 percent to 50 percent.[139] As demonstrated in Table 8, the natural history of posterior canal BPPV varies widely across studies at a 1-month and a 3-month follow-up interval. Further variability in the spontaneous resolution rate arises from differences in duration of symptoms prior to actual diagnoses of BPPV

Table 8
Symptom resolution rates for observation alone for BPPV*

Reference	Resolved n/m	% Resolved	Sham or pure observation	Time to assessment
von Brevern 2007[11]	22/26	84.6	Sham	4 weeks
Sekine 2006[142]	48/60	80.0	Observation	1 month
Imai 2005[49]	45/70	64.0	Observation	1 month
Simhadri 2003[177]	3/15	20.0	Observation	4 weeks
Yimtae 2003[129]	7/20	35.0	Observation	1 month
Sherman 2001[131]	11/22	50.0	Sham	3 months
Asawavichianginda 2000[135]	18/22	81.8	Observation	3 months
Steenerson 1996[171]	17/40	42.5	Observation	3 months
Lynn 1995[128]	3/15	20.0	Sham	1 month
Blakley 1994[176]	19/22	86.4	Observation	1 month

*Endpoint: resolution of vertigo symptoms at the time of assessment.

as well as differences in duration of follow-up.[42,59,128,142] Longitudinal follow-up studies of untreated BPPV patients are lacking, but one study of completely untreated patients determined a mean time interval from onset of symptoms to spontaneous resolution of BPPV of 39 ± 47 days.[49] As would be expected, spontaneous symptom resolution rates increase with increasing duration of follow-up among observed patients.

Although observation of posterior canal BPPV is an option for management, clinicians should also be aware that other treatments such as the PRM have been shown to offer patients faster resolution of BPPV symptoms. A meta-analysis of nine separate trials examining the efficacy of the PRM for BPPV treatment demonstrated consistent improvement in the treatment group, with up to 4.1 times greater rates of symptom resolution (95% confidence interval, 3.1-5.2) in the PRM groups vs the control groups at initial assessments within 1 month. Studies with follow-up at beyond 1 month still demonstrated an improvement rate of nearly three times that of controls.[139] Other longer-term follow-up data also suggest that patients treated with a PRM had lower rates of relapse of BPPV at 6 months and 1 year posttreatment.[177]

Observation as an option for the management of posterior canal BPPV offers the potential benefits of avoiding repositioning maneuvers or vestibular rehabilitation, which in turn may provoke symptoms and discomfort. There may also be a cost savings from decreased rates of referral for vestibular rehabilitation or PRMs. From a potential harms perspective, patients who elect for the observation option should be informed about a typically longer duration of symptoms compared with a treatment maneuver and potentially higher recurrence rates. Appropriate precautions for the risks associated with BPPV symptoms should be taken during the watchful waiting period.

The natural history of lateral canal BPPV is less well defined than that of posterior canal BPPV. Several authors have commented that lateral canal BPPV may be prone to more rapid spontaneous resolution than posterior canal BPPV.[51,142] In one study, the mean time between the onset of vertigo in lateral canal BPPV to spontaneous resolution was 16 ± 19 days.[49] Although repositioning maneuvers have shown success in lateral canal BPPV, overall high quality comparative data regarding treatment vs observation such as RCTs are limited in this subtype of BPPV.[58,142,148] Thus, observation of lateral canal BPPV remains an option for management. Future RCTs need to be dedicated to the interventional management of lateral canal BPPV.

Evidence Profile

- Aggregate evidence quality: Grade B, based on control groups from RCTs and observational studies with heterogeneity in follow-up and outcomes measures
- Benefit: symptom resolution in 15 to 85 percent of patients at 1 month without intervention
- Harm: prolonged symptoms compared with other interventions that may expose patients to increased risks for falls or lost days of work
- Cost: indirect costs of delayed resolution compared with other measures
- Benefit-harm assessment: relative balance of benefits and harms
- Value judgments: bias of the panel for treatment intervention rather than observation, particularly with respect to the value of a quicker time to resolution (The panel felt that older patients and patients with preexisting balance disorders or high risks for falls may not be suitable for observation.)
- Role of patient preferences: substantial for shared decision making
- Exclusions: none
- Policy level: option

Statement 5. Medical Therapy

Clinicians should not routinely treat BPPV with vestibular suppressant medications such as antihistamines or benzodiazepines. *Recommendation against based on observational studies and a preponderance of benefit over harm.*

The symptoms of vertigo due to many different underlying etiologies are commonly treated with medications. Clinicians may prescribe pharmacological management to either 1) reduce the spinning sensations of vertigo specifically and/or 2) to reduce the accompanying motion sickness symptoms. These motion sickness symptoms include a constellation of autonomic or vegetative symptoms such as nausea, vomiting, and diarrhea, which can accompany the vertigo. Such pharmacological therapies for vertigo may be broadly termed *vestibular suppressant medications*.[178,179]

Several categories of vestibular suppressant medications are in common use. Of these, the most commonly used are benzodiazepines and antihistamines. 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 they also interfere with central compensation in peripheral vestibular conditions. Antihistamines, on the other hand, 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-5-hydroxytryptamine-3 antagonist. Finally, anticholinergic medications such as scopolamine block acetylcholine, which is a widespread CNS transmitter, and help with motion sickness by reducing neural mismatching.[178,179]

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 as a substitute for repositioning maneuvers.[98,178,180-182] Some studies show a resolution of BPPV over time with medications, but these studies follow patients for the period of time in which spontaneous resolution would occur.[139,183-185] In one double-blind controlled trial by McClure and Willet[185] comparing diazepam, lorazepam, and placebo, all groups showed a gradual decline in symptoms with no additional relief in the drug treatment arms. In a small study, Itaya et al[184] compared PRMs to a medication-alone treatment arm and found that PRMs had substantially higher treatment responses (78.6%-93.3% improvement) compared with medication alone (30.8% improvement) at 2 weeks follow-up. These data reinforced previous data from Fujino et al[182] that also indicated superiority of vestibular training for BPPV over medication use alone. A lack of benefit from vestibular suppressants and their inferiority to PRMs indicate that clinicians should not substitute pharmacological treatment of symptoms associated with BPPV in lieu of other more effective treatment modalities.

Conversely, vestibular suppressant medications have the potential for significant harm. All of these medications may produce drowsiness, cognitive deficits, and interference with driving vehicles or operating machinery.[186-190] Medications used for vestibular suppression, especially psychotropic medications such as benzodiazepines, are a significant independent risk factor for falls.[191] The risk of falls increases in patients taking multiple medications and with the use of medications such as antidepressants.[16,192] The potential for polypharmacy when adding vestibular suppressants further exposes the elderly to additional risk.[193] Educational programs to modify practitioner's use of such medications can result in a reduction of falls.[194]

There are other potential harmful side effects of vestibular suppressants. Benzodiazepines and antihistamines interfere with central compensation for a vestibular injury.[3,195,196] 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 functioning,[186] and on gastrointestinal motility, urinary retention, vision, and dry mouth in the elderly.[197]

In summary, vestibular suppressant medications are not recommended for treatment of BPPV, other than for the short-term management of vegetative 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 PRM. 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 PRM 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.

Evidence Profile

- Aggregate evidence quality: Grade C, based on observational and cross-sectional studies
- Benefit: unknown or unclear benefit in patients with BPPV
- Harm: adverse effects from or medication interactions with these medications; decreased diagnostic sensitivity during Dix-Hallpike maneuvers from vestibular suppression
- Cost: none
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: avoidance of harm from ineffective treatments
- Role of patient preferences: minimal
- Exclusions: severely symptomatic patients refusing other treatment options and patients requiring prophylaxis for CRP
- Policy level: recommendation against

Statement 6a. Reassessment of Treatment Response

Clinicians should reassess patients within 1 month after an initial period of observation or treatment to confirm symptom resolution. *Recommendation based on observational outcomes studies and expert opinion and a preponderance of benefit over harm.*

Patients with BPPV, regardless of initial treatment option rendered, will have variable responses to therapy.[43] The response to therapy may depend on several factors including the accuracy of the diagnosis of BPPV, the duration of symptoms prior to the diagnosis of BPPV, compliance with prescribed therapy, and other factors.[42,120] Patients with BPPV should be reassessed within a set time interval after the diagnosis of BPPV for several reasons.

Failure to respond to initial therapy may indicate an initially erroneous diagnosis of BPPV, and one of the major goals of reassessment is to ensure the accuracy of diagnosis of BPPV. As noted, other more serious CNS disorders may mimic BPPV, and these conditions would not be expected to respond to traditional therapies prescribed for BPPV. In cohort studies, the rate of false-positive diagnosis for BPPV subsequently found to be CNS lesions *after failed treatment* (therefore, a highly selected population) with PRM ranges from 1.1 to 3 percent.[120,198] 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 initial therapy such as a PRM would corroborate an accurate diagnosis of BPPV.

Patients who are initially treated with vestibular rehabilitation may fail to resolve symptoms owing to multiple factors including poor compliance. In addition, patients who do not respond to initial therapy are likely to remain at risk for falls, decreased quality of life, and other consequences of unresolved BPPV. For these reasons, patients whose symptoms of BPPV fail to resolve should also be identified and classified as initial treatment failures.

To define a treatment failure in BPPV, the clinician needs to determine both a failed outcome criterion and an appropriate time interval for assessment of treatment failure. Successful treatment outcomes for interventions for BPPV are traditionally measured in clinical trials by subjective symptom resolution and/or by conversion to a negative Dix-Hallpike test. Almost all treatment trials for BPPV report an outcome measure in the form of the patient's reported symptoms, typically reported among three categorical outcomes: complete resolution of symptoms, improvement, or no improvement/worsening.[42] When included in meta-analyses, treatment responses are typically incorporated as "all or none" for the complete resolution of vertigo.[42,139,140]

Because effective treatment options are available for BPPV that typically render patients symptom free (if treatment is successful), it is logical to use complete symptom resolution as the outcome of choice at the time of reassessment by the clinician. A symptom-based reassessment also allows clinicians to use clinical judgment as to the most appropriate modality for follow-up for individual patients, including telephone communication, electronic communication, or office based reexamination. This symptom-based assessment of treatment resolution should be detailed enough to distinguish patients with truly decreased symptoms related to treatment or patients with minimized symptoms attributable to positional avoidance (who, in fact, may not be treatment successes) from those with true symptom resolution.[139]

Although conversion to a negative Dix-Hallpike test may have the advantage of being a more objective reassessment than patients' reported symptoms, it also carries the disadvantage of requiring a repeat clinical visit on the part of the patient with associated direct and indirect costs. The Dix-Hallpike test status is commonly reported in therapeutic trials of BPPV. Persistent symptoms of BPPV and other underlying conditions, however, have been reported in the face of negative Dix-Hallpike testing after therapy, potentially making this a less sensitive reassessment tool.[128,199] Conversely, patients may report an absence of symptoms after therapeutic intervention yet still have a positive Dix-Hallpike test.[43,59,131] "Subclinical BPPV" has been offered as an explanation for this.[43] Because of the potential discordance between negative Dix-Hallpike conversion and patients' reported symptoms after treatment for BPPV, Dix-Hallpike conversion is not recommended as the primary reassessment criterion in routine clinical practice but may still be used as a secondary outcome measure.

There is no widely accepted time interval at which to assess patients for treatment failure. Therapeutic trials in BPPV variably report follow-up assessments for treatment outcomes at 40 hours, 2 weeks, 1 month, and up to 6 months, although the most commonly chosen interval for follow-up assessment of treatment response is within or at 1 month.[42,139,140] Because the natural history of BPPV exhibits a relatively consistent spontaneous rate of resolution with observation alone, a longer time interval between diagnosis and reassessment would allow patients with true BPPV to resolve symptoms spontaneously, likely irrespective of treatment.[142]

Conversely, the choice of an excessively long time interval between diagnosis and reassessment would also allow cases of an erroneous BPPV diagnosis to potentially progress, leading to potential patient harm. In addition, because recurrence of BPPV may occur as early as 3 months after initial treatment, further delaying the time interval for reassessment may erroneously incorporate a recurrent BPPV syndrome (ie, the initial BPPV responded to treatment with a suitable symptom-free interval thereafter, followed by recurrent BPPV) rather than a persistent BPPV syndrome.[38,174]

Given that commonly reported rates of spontaneous complete symptom resolution at the 1-month interval for BPPV range from 20 to 80 percent at 1 month, reassessment

at 1 month will also better allow for patients to be reconsidered for further interventional treatment to treat unresolved BPPV.[59,128-130,142,159] Thus, choosing a reassessment time interval of 1 month after diagnosis allows a relative balance between overly early reassessment (which would force the unnecessary reassessment of patients who would likely resolve with additional time) and unduly delayed reassessment (which would potentially allow harm from an unknown missed diagnosis or relegate patients to an excess time interval of symptomatic suffering from BPPV).

One potential problem with a strict time interval for reassessment is that patients may not have been exposed to their initial treatment (vestibular rehabilitation or PRM as opposed to observation, which may begin immediately after diagnosis) within 1 month of diagnosis depending on referral patterns, patient preferences, or waiting lists for specialty evaluation and treatment. This situation is especially true when the diagnosing clinician may not be the same as the treating clinician. Even if a delay occurs between BPPV diagnosis and completion of the initial treatment, clinicians should still reassess patients at 1 month but may choose to reassign a second time interval for reassessment after completion of the initial treatment option.

Evidence Profile

- Aggregate evidence quality: Grade C, based on studies with known significant failure rates for an observation option and lower failure rates for PRM
- Benefit: increased accuracy of diagnosis of BPPV; identification of patients with persistent symptoms who were initially treated with observation and may benefit from vestibular rehabilitation or PRM to hasten symptom resolution
- Harm: none
- Cost: cost of reassessment
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: assurance of accuracy of diagnosis and capture of patients who could benefit from treatment or re-treatment to improve symptom resolution
- Role of patient preferences: minimal
- Policy level: recommendation

Statement 6b. Evaluation of Treatment Failure

Clinicians should evaluate patients with BPPV who are initial treatment failures for persistent BPPV 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.*

Patients with persistent symptoms of vertigo, dizziness, or unsteadiness at the time of reassessment of the initial treatment response 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.[28,120,200]

Persistent BPPV

Patients with BPPV who initially are treated with observation may fail to resolve spontaneously and have persistent BPPV at the time reassessment. Also, on the basis of failure rates of vestibular rehabilitation or a single-session PRM ranging from 15 to 50 percent, a significant number of patients initially managed with vestibular rehabilitation or PRM will have persistent BPPV at reassessment, which also indicates a treatment failure.[28,42,43,140,159] Reevaluation of a treatment failure should include obtaining a history of vertigo, 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 PRMs can then be performed as a preferred treatment. The rate of successful treatment of BPPV reaches 90 to 98 percent when additional repositioning maneuvers are subsequently performed.[201,202] Therefore, the PRMs are the treatment of choice for initial BPPV treatment failures deemed to be due to persistent BPPV.

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 PRM appropriate for lateral canal BPPV may be repeated as well. There are limited data regarding the management of treatment failures after PRM for lateral canal BPPV, because this condition seems to respond more consistently to PRM and it also has a higher spontaneous resolution rate.[56,142,148,151] Some studies indicate cure rates of 86 to 100 percent with up to four PRM treatments in lateral canal BPPV.[58,152] Further subanalysis suggests that the apogeoptropic variant of lateral canal BPPV may be more refractory to therapy.[5,58]

A small percentage of patients initially diagnosed and treated for lateral canal BPPV or horizontal canal BPPV may experience a canal switch. In these cases, initial horizontal canal BPPV may transform into posterior canal BPPV in up to 6 percent of cases.[55,56] Similarly, a small fraction of patients (also approximating 6%) initially presenting with posterior canal BPPV may transition after treatment to lateral canal BPPV.[129,144] 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.[18] Finally, although rare, two semicircular canals may be simultaneously involved. The second canal's involvement may become evident at the time of reassessment if one of the involved canals was appropriately treated.[120] Thus, reassessment of persistent positional vertigo in BPPV should

include examination for involvement of other semicircular canals than originally diagnosed.

Coexisting Vestibular System Dysfunction

A BPPV treatment failure subsequently may be 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 at the time of reevaluation; such findings suggest the presence of vestibular system dysfunction associated with or in addition to the initially treated BPPV. There may be several possible factors at play when vestibular system dysfunction accompanies BPPV.

In a study by Monobe et al,[203] treatment failure of the PRM was most commonly seen in patients with BPPV secondary to head trauma or vestibular neuritis. Because 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.[204] 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 of BPPV, and increased risk for falls, particularly in older persons.[97,115,117,205-207]

In addition, BPPV not associated with any other otological or neurological disease can still be associated with an underlying impaired vestibular function, and these individuals are more likely to have incomplete resolution of symptoms even if their Dix-Hallpike testing normalizes with PRM.[118] Finally, transient vestibular dysfunction can also occur following repositioning maneuvers. Evidence suggests that balance function continues to be affected between 1 to 3 months after repositioning maneuvers, and that some of these patients may need additional balance therapy (ie, counseling, vestibular rehabilitation) to prevent falls and decrease their fear of falling after the vertigo from BPPV has resolved.[36,208-210] 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 testing may include audiometric testing to screen for Ménière's disease and nerve VIII 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. Vestibular rehabilitation has been shown to be an effective treatment for vestibular symptoms due to the potentially persistent vestibular dysfunction associated with BPPV; this treatment may reduce the risk for falls.[136]

CNS Disorders Masquerading as BPPV

Although vertigo of central origin is frequently associated with neurological symptoms such as gait, speech, and autonomic dysfunction, it is important to recognize that, rarely, CNS disorders can masquerade as BPPV.[211] Many of these have been previously discussed in the section on differential diagnosis, but the relative likelihood of their diagnosis increases in the face of initial treatment failure. In one study, a CNS disorder that explained BPPV treatment failure was found in 3 percent of patients.[198]

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.[212] Patients with symptoms consistent with those of BPPV who do not show improvement or resolution after undergoing the PRM, especially after two or three attempted maneuvers, or those who describe associated auditory or neurological symptoms should be evaluated with a thorough neurological examination, additional CNS testing, and/or MRI of the brain and posterior fossa to identify possible intracranial pathological conditions.[82,213]

Evidence Profile

- Aggregate evidence quality: Grade C, based on case series of treatment failure and limited retrospective diagnostic studies
- Benefit: expedition of effective treatment of patients with persistent BPPV and associated comorbidities; decrease in the potential for missed serious medical conditions that require a different treatment algorithm
- Harm: none
- Cost: costs of reevaluation and the additional testing
- Benefit-harm assessment: preponderance of benefit vs harm
- Value judgments: comprehensive treatment of not only BPPV but associated conditions that affect balance and function; expeditious treatment of cases of persistent BPPV with a PRM (as more definitive therapy) following the failure of observation or vestibular rehabilitation
- Role of patient preferences: minimal
- Policy level: recommendation

Statement 7. Education

Clinicians should counsel 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.*

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 percent at

6-month follow-up.[33,145] At 1 year after treatment, the rate of recurrence has been reported at a slightly higher rate of 10 to 18 percent.[143,214] The recurrence rate continues to increase and may be as high as 37 to 50 percent at 5 years by Kaplan-Meier estimation.[38,214] Overall the recurrence rate of BPPV may be estimated at 15 percent per year.[38] Patients with BPPV after trauma are likely to demonstrate an even higher recurrence rate of their BPPV.[97]

Thus, clinicians should be aware of the recurrence risk of BPPV and should counsel patients accordingly. Counseling will likely have several benefits, which include earlier recognition by patients of recurrent BPPV, allowing earlier return for PRM or vestibular rehabilitation. Also, counseling regarding recurrence will offset the potential anxiety 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 regarding the potential that BPPV may place them at greater risk for falls.[215] This risk may apply particularly to 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 of underlying BPPV.[16] The risk of falls and fear of falls are significant considerations in the management of the elderly who suffer from chronic dizziness.[216] In a study of 120 elderly patients with chronic vestibular disorders, 36.7 percent carried the diagnosis of BPPV. Fifty-three percent of subjects had fallen at least once in the past year, and 29.2 percent had recurrent falls.[216] Other authors have confirmed a relatively high rate of BPPV and associated falling tendencies in the elderly.[15,217]

Practically speaking, clinicians should counsel patients and their families regarding the risk of falls associated with BPPV. This information is particularly important for 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.[90] 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 PRM or vestibular rehabilitation. Counseling should therefore occur at the time of initial diagnosis. The costs of such counseling are anticipated to be minimal and will enhance patient and public safety while avoiding potential posttraumatic sequelae.

Finally, patients should be counseled regarding the importance of follow-up after diagnosis of BPPV. Patients initially treated with observation should be counseled that, if BPPV fails to resolve spontaneously, effective therapies such as the PRM may then be undertaken. Also, patients should be educated about atypical symptoms (subjective hearing loss, gait disturbance, non-positional vertigo, nausea, vomiting, etc.) whose occurrence or persistence after resolution of the primary symptoms of BPPV warrant further clinical evaluation.[120] As noted, such symptoms, particularly when unmasked by the resolution of BPPV may indicate an underlying vestibular or CNS disorder. Clinicians may also educate patients with refractory BPPV or repeatedly recurrent BPPV that in select cases a surgical remedy ("canal plugging procedure" or singular neurectomy) may be considered.[7,218]

Evidence Profile

- Aggregate evidence quality: Grade C, based on observational and cross-sectional studies of recurrence and fall risk
- Benefit: increased awareness of fall risk, potentially decreasing injuries related to falls; increased patient awareness of BPPV recurrence, allowing prompt intervention
- Harm: none
- Cost: none
- Benefit-harm assessment: preponderance of benefit over harm
- Value judgments: inadequate data to elaborate recommendations for patients with BPPV with regard to driving vehicles
- Role of patient preferences: none
- Policy level: recommendation

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 highlighting key recommendations from the guideline will be published to facilitate information dissemination. Portions of the guideline will be presented at various clinical meetings including a planned presentation in the workshop series of the American College of Physicians annual meeting. Existing brochures and publications by the AAO-HNS Foundation will be updated to reflect the guideline recommendations. Members of the panel will be representing the guideline at their specialty societies, and executive summaries to be copublished in the primary care and physical therapy literature are anticipated.

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, readers will be provided with Web-based video links that illustrate performance of these maneuvers, as well as video representations of the expected diagnostic nystagmus findings, especially in the case of lateral canal BPPV. These media aids may also be assisted by a laminated teaching card that describes the maneuvers. 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 because they perceive that such testing is required or a safer course of action in the routine management of vertigo. Informational pamphlets for patients that explain their diagnosis and provide realistic expectations with regard to the natural history of BPPV may ease this difficulty. Specialty clinicians will likely exhibit a natural tendency for ordering additional diagnostic testing owing to a variety of factors. Clinician and patient education regarding outcome expectations and counseling on proper follow-up may offset these issues. Physician and patient education, either Web-based or published results of large trials on diagnostic outcomes for BPPV, will also help offset these tendencies.

With respect to treatment with PRMs, several barriers may 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 time the diagnosis is made. Because of a paucity of data in the primary care setting (only one RCT that failed to demonstrate effectiveness of the CRP), convincing primary care physicians to use the CRP as an initial treatment modality may be difficult. 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 societal 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 as to these risks.[87]

RESEARCH NEEDS

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

1) Conduct prospective epidemiological studies of the incidence, prevalence, and burden of untreated BPPV among older adults.
2) Conduct prospective diagnostic cohort studies to determine the sensitivity, specificity, and predictive values for the Dix-Hallpike maneuvers in the diagnosis of posterior canal BPPV. Such studies should also determine the latency duration and duration of subjective vertigo and objective nystagmus with the maneuver. Diagnostic cohort studies should be extended to nonspecialist environments including the primary care and emergency department settings.
3) Conduct prospective diagnostic cohort studies to determine the sensitivity, specificity, and predictive values for the supine roll test for lateral canal BPPV. Diagnostic cohort study should be extended to nonspecialty environments including the primary care and emergency department settings.
4) Conduct diagnostic and cost-effectiveness studies to identify which subsets of patients, according to specific history or physical examination findings, should be submitted for additional vestibular testing and/or radiographic imaging in the setting of presumed BPPV.
5) Conduct diagnostic and cost-effectiveness studies evaluating the utility and costs of audiometry in the diagnostic evaluation of BPPV.
6) 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.
7) Define the natural history of untreated posterior canal BPPV and lateral canal BPPV to determine proper endpoints for clinical trials and follow-up assessments.
8) Determine the optimal number of CRPs and the time interval between performances of CRP for patients with posterior canal BPPV.
9) Conduct RCTs of CRP for posterior canal BPPV with emphasis on 1) larger sample sizes, 2) (faster) time to resolution of symptoms with CRP rather than resolution of symptoms at a set endpoint in time, 3) trials in the primary care and/or emergency department settings, and 4) outcomes such as quality of life, return to work, reduced fall risk.
10) Conduct RCTs of PRMs for lateral canal BPPV to determine the effectiveness of proposed treatments. Time to resolution rather than resolution at a fixed endpoint should also be emphasized.
11) Conduct RCTs comparing PRMs to vestibular rehabilitation including comparisons among different vestibular rehabilitation options.
12) Conduct cost-effectiveness studies for the potential advantages of earlier intervention based on earlier diagnosis and earlier symptom resolution with expedient PRMs for BPPV. Both direct health care and global economic costs require assessment.

13) Conduct extended cohort studies with longer follow-up to determine if measures such as self-performance of CRP or longitudinal vestibular rehabilitation decrease recurrence rates for BPPV or complications from BPPV such as falls.
14) Conduct studies on the functional impact of BPPV as they relate to home safety, work safety and absences, and driving risks.
15) Conduct epidemiological studies on the rates of falls with BPPV as an underlying cause/diagnosis.
16) Develop and validate a disease-specific quality-of-life measure for BPPV to assess treatment outcomes.

DISCLAIMER

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 they are not absolute. Guidelines are not mandates and do not and should not purport to be a legal standard of care. The responsible physician, in light of all the 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 American Academy of Otolaryngology—Head and Neck Surgery (AAO-HNS), Inc. 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.

ACKNOWLEDGEMENT

We gratefully acknowledge the support provided by Milesh Patel, MS, and Kristine Schulz, MPH, from the AAO-HNS Foundation.

AUTHOR INFORMATION

Brigham & Women's Hospital (Dr Bhattacharyya), Boston, MA; Scott & White Memorial Hospital (Dr Baugh), Temple, TX; Mayo Clinic (Dr Orvidas), Rochester, MN; Mayo Clinic (Dr Barrs), Phoenix, AR; Bronston Chiropractic Clinics (Dr Bronston), La Cross, WI; University of Colorado School of Medicine (Dr Cass), Denver, CO; University of Pennsylvania Health System (Dr Chalian), Philadelphia, PA; Blue Ridge Hearing and Balance Clinic (Dr Desmond), Princeton, WV; The Washington Home (Dr Earll), Washington, DC; Johns Hopkins Bayview Medical Center (Dr Fuller), Baltimore, MD; Barrow Neurological Institute (Dr Fife), Phoenix, AZ; Evercare Connecticut (Dr Judge), Hartford, CT; Detroit Medical Center (Dr Mann), Detroit, MI; SUNY Downstate Medical College and Long Island College Hospital (Dr Rosenfeld), Brooklyn, NY; Society of Otorhinolaryngology and Head and Neck Nurses, Inc. (Ms. Schuring), New Smyrna Beach, FL; University of Louisville School of Public Health and Information Sciences (Dr Steiner), Louisville, KY; University of Pittsburgh (Dr Whitney), Pittsburgh, PA; American Academy of Otolaryngology—Head and Neck Surgery Foundation (Ms. Haidari), Alexandria, VA.

Corresponding author: Neil Bhattacharyya, MD, Brigham & Women's Hospital, 75 Francis Street, Boston, MA 02115.

E-mail address: neiloy@massmed.org.

AUTHOR CONTRIBUTIONS

Neil Bhattacharyya: writer, chair; **Reginald Baugh:** writer, assistant chair; **Laura Orvidas:** writer, assistant chair; **David Barrs, Leo Bronston, Stephen Cass, Ara Chalian, Alan Desmond, Jerry Earll, Terry Fife, Drew Fuller, James Judge, Nancy Mann:** writers; **Richard Rosenfeld:** writer, consultant; **Linda Schuring, Robert Steiner, Susan Whitney, Jenissa Haidari:** writers.

FINANCIAL DISCLOSURE

Neil Bhattacharyya: none; **Reginald F. Baugh:** consultant: Gerson Lehrman Group Councils, stockholder: American Laser Center; **Laura Orvidas:** research funding: Aventis; **David Barrs:** none; **Leo J. Bronston:** none; **Stephen Cass:** none; **Ara A. Chalian:** none; **Alan L. Desmond:** none; **Jerry M. Earll:** none; **Terry D. Fife:** none; **Drew C. Fuller:** none; **James O. Judge:** none; **Nancy R. Mann:** none; **Richard M. Rosenfeld:** none; **Linda T. Schuring:** none; **Robert W. P. Steiner:** none; **Susan L. Whitney:** none; **Jenissa Haidari:** none.

REFERENCES

1. Schappert SM. National Ambulatory Medical Care Survey: 1989 summary. Vital Health Stat 1992;13:1–80.
2. Katsarkas A. Benign paroxysmal positional vertigo (BPPV): idiopathic versus post-traumatic. Acta Otolaryngol 1999;119:745–9.
3. Hanley K, O'Dowd T, Considine N. A systematic review of vertigo in primary care. Br J Gen Pract 2001;51:666–71.
4. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. Neurology 1987;37:371–8.
5. White JA, Coale KD, Catalano PJ, et al. Diagnosis and management of lateral semicircular canal benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 2005;133:278–84.
6. 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;134:451–4.
7. Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). CMAJ 2003;169:681–93.
8. Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. Laryngoscope 1992;102:988–92.
9. 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;406:178–81.
10. 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.
11. von Brevern M, Radtke A, Lezius F, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. J Neurol Neurosurg Psychiatry 2007;78:710–5.
12. Nedzelski JM, Barber HO, McIlmoyl L. Diagnoses in a dizziness unit. J Otolaryngol 1986;15:101–4.
13. Neuhauser HK. Epidemiology of vertigo. Curr Opin Neurol 2007;20:40–6.
14. Li JC, Li CJ, Epley J, et al. Cost-effective management of benign positional vertigo using canalith repositioning. Otolaryngol Head Neck Surg 2000;122:334–9.
15. Oghalai JS, Manolidis S, Barth JL, et al. Unrecognized benign paroxysmal positional vertigo in elderly patients. Otolaryngol Head Neck Surg 2000;122:630–4.

16. Lawson J, Johnson I, Bamiou DE, et al. Benign paroxysmal positional vertigo: clinical characteristics of dizzy patients referred to a Falls and Syncope Unit. QJM 2005;98:357–64.
17. 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;44:50–7.
18. Jackson LE, Morgan B, Fletcher JC, Jr., et al. Anterior canal benign paroxysmal positional vertigo: an underappreciated entity. Otol Neurotol 2007;28:218–22.
19. 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;70:2067–74.
20. Rosenfeld RM, Shiffman RN. Clinical practice guidelines: a manual for developing evidence-based guidelines to facilitate performance measurement and quality improvement. Otolaryngol Head Neck Surg 2006;135:S1–S28.
21. Montori VM, Wilczynski NL, Morgan D, et al. Optimal search strategies for retrieving systematic reviews from Medline: analytical survey. BMJ 2005;330:68.
22. Shiffman RN, Shekelle P, Overhage JM, et al. Standardized reporting of clinical practice guidelines: a proposal from the Conference on Guideline Standardization. Ann Intern Med 2003;139:493–8.
23. AAP SCQIM (American Academy of Pediatrics Steering Committee on Quality Improvement and Management). Policy Statement. Classifying recommendations for clinical practice guidelines. Pediatrics 2004;114:874–7.
24. Eddy DM. A manual for assessing health practices and designing practice policies: the explicit approach. Philadelphia: American College of Physicians; 1992.
25. Choudhry NK, Stelfox HT, Detsky AS. Relationships between authors of clinical practice guidelines and the pharmaceutical industry. JAMA 2002;287:612–7.
26. Detsky AS. Sources of bias for authors of clinical practice guidelines. CMAJ 2006;175:1033, 35.
27. Blakley BW, Goebel J. The meaning of the word "vertigo." Otolaryngol Head Neck Surg 2001;125:147–50.
28. Furman JM, Cass SP. Benign paroxysmal positional vertigo. N Engl J Med 1999;341:1590–6.
29. 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.
30. Whitney SL, Marchetti GF, Morris LO. Usefulness of the dizziness handicap inventory in the screening for benign paroxysmal positional vertigo. Otol Neurotol 2005;26:1027–33.
31. 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;69:295–8.
32. Herdman SJ. Advances in the treatment of vestibular disorders. Phys Ther 1997;77:602–18.
33. Macias JD, Lambert KM, Massingale S, et al. Variables affecting treatment in benign paroxysmal positional vertigo. Laryngoscope 2000;110:1921–4.
34. Cohen HS. Side-lying as an alternative to the Dix-Hallpike test of the posterior canal. Otol Neurotol 2004;25:130–4.
35. 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.
36. 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; 21:356–63.
37. Norre ME. Reliability of examination data in the diagnosis of benign paroxysmal positional vertigo. Am J Otol 1995;16:806–10.
38. Nunez RA, Cass SP, Furman JM. Short- and long-term outcomes of canalith repositioning for benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 2000;122:647–52.
39. Honrubia V, Baloh RW, Harris MR, et al. Paroxysmal positional vertigo syndrome. Am J Otol 1999;20:465–70.
40. Norre ME, Beckers A. Benign paroxysmal positional vertigo in the elderly. Treatment by habituation exercises. J Am Geriatr Soc 1988; 36:425–9.
41. White J, Savvides P, Cherian N, et al. Canalith repositioning for benign paroxysmal positional vertigo. Otol Neurotol 2005;26:704–10.
42. Hilton M, Pinder D. The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. Cochrane Database Syst Rev 2004:CD003162.
43. Cohen HS, Kimball KT. Effectiveness of treatments for benign paroxysmal positional vertigo of the posterior canal. Otol Neurotol 2005;26:1034–40.
44. 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.
45. Hanley K, O' Dowd T. Symptoms of vertigo in general practice: a prospective study of diagnosis. Br J Gen Pract 2002;52:809–12.
46. Viirre E, Purcell I, Baloh RW. The Dix-Hallpike test and the canalith repositioning maneuver. Laryngoscope 2005;115:184–7.
47. Norre ME. Diagnostic problems in patients with benign paroxysmal positional vertigo. Laryngoscope 1994;104:1385–8.
48. Whitney SL, Morris LO. Multisensory impairment in older adults: evaluation and intervention. In: Geriatric Otolaryngology. Calhoun KH, Eibling DE, ed. New York: Taylor and Francis; 2006. p. 115.
49. Imai T, Ito M, Takeda N, et al. Natural course of the remission of vertigo in patients with benign paroxysmal positional vertigo. Neurology 2005;64:920–1.
50. Steenerson RL, Cronin GW, Marbach PM. Effectiveness of treatment techniques in 923 cases of benign paroxysmal positional vertigo. Laryngoscope 2005;115:226–31.
51. 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;21:539–43.
52. Hornibrook J. Horizontal canal benign positional vertigo. Ann Otol Rhinol Laryngol 2004;113:721–5.
53. Han BI, Oh HJ, Kim JS. Nystagmus while recumbent in horizontal canal benign paroxysmal positional vertigo. Neurology 2006;66:706–10.
54. Caruso G, Nuti D. Epidemiological data from 2270 PPV patients. Audiol Med 2005;3:7–11.
55. Nuti D, Agus G, Barbieri MT, et al. The management of horizontal-canal paroxysmal positional vertigo. Acta Otolaryngol 1998;118: 455–60.
56. Tirelli G, Russolo M. 360-Degree canalith repositioning procedure for the horizontal canal. Otolaryngol Head Neck Surg 2004;131: 740–6.
57. Baloh RW, Jacobson K, Honrubia V. Horizontal semicircular canal variant of benign positional vertigo. Neurology 1993;43:2542–9.
58. Casani AP, Vannucci G, Fattori B, et al. The treatment of horizontal canal positional vertigo: our experience in 66 cases. Laryngoscope 2002;112:172–8.
59. 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.
60. von Brevern M, Lezius F, Tiel-Wilck K, et al. Benign paroxysmal positional vertigo: current status of medical management. Otolaryngol Head Neck Surg 2004;130:381–2.
61. Kentala E, Rauch SD. A practical assessment algorithm for diagnosis of dizziness. Otolaryngol Head Neck Surg 2003;128:54–9.
62. Wladislavosky-Waserman P, Facer GW, Mokri B, et al. Ménière's disease: a 30-year epidemiologic and clinical study in Rochester, MN, 1951-1980. Laryngoscope 1984;94:1098–102.
63. 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;28:173–6.
64. Baloh RW. Clinical practice. Vestibular neuritis. N Engl J Med 2003;348:1027–32.
65. Kentala E. Characteristics of six otologic diseases involving vertigo. Am J Otol 1996;17:883–92.
66. Kentala E, Laurikkala J, Pyykko I, et al. Discovering diagnostic rules from a neurotologic database with genetic algorithms. Ann Otol Rhinol Laryngol 1999;108:948–54.
67. Minor LB, Cremer PD, Carey JP, et al. Symptoms and signs in superior canal dehiscence syndrome. Ann New York Acad Sci 2001; 942:259–73.
68. 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: 323–32.
69. Marzo SJ, Leonetti JP, Raffin MJ, et al. Diagnosis and management of post-traumatic vertigo. Laryngoscope 2004;114:1720–3.
70. Davies RA, Luxon LM. Dizziness following head injury: a neuro-otological study. J Neurol 1995;242:222–30.
71. Karlberg M, Hall K, Quickert N, et al. What inner ear diseases cause benign paroxysmal positional vertigo? Acta Otolaryngol 2000;120: 380–5.
72. Labuguen RH. Initial evaluation of vertigo. Am Fam Physician 2006; 73:244–51.
73. Baloh RW. Differentiating between peripheral and central causes of vertigo. Otolaryngol Head Neck Surg 1998;119:55–9.
74. Reploeg MD, Goebel JA. Migraine-associated dizziness: patient characteristics and management options. Otol Neurotol 2002;23:364–71.
75. Headache Classification Subcommittee of the International Headache S. The International Classification of Headache Disorders: 2nd edition. Cephalalgia 2004;24(suppl 1):9–160.
76. Neuhauser H, Leopold M, von Brevern M, et al. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology 2001;56: 436–41.
77. von Brevern M, Zeise D, Neuhauser H, et al. Acute migrainous vertigo: clinical and oculographic findings. Brain 2005;128:365–74.
78. Fife TD, Baloh RW, Duckwiler GR. Isolated dizziness in vertebrobasilar insufficiency: clinical features, angiography, and follow-up. J Stroke Cerebrovasc Dis 1994;4:4–12.
79. Grad A, Baloh RW. Vertigo of vascular origin. Clinical and electronystagmographic features in 84 cases. Arch Neurol 1989;46:281–4.
80. Gomez CR, Cruz-Flores S, Malkoff MD, et al. Isolated vertigo as a manifestation of vertebrobasilar ischemia. Neurology 1996;47:94–7.
81. Hotson JR, Baloh RW. Acute vestibular syndrome. N Engl J Med 1998;339:680–5.
82. Dunniway HM, Welling DB. Intracranial tumors mimicking benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 1998; 118:429–36.
83. Jacob RG, Furman JM, Durrant JD, et al. Panic, agoraphobia, and vestibular dysfunction. Am J Psychiatry 1996;153:503–12.
84. Furman JM, Redfern MS, Jacob RG. Vestibulo-ocular function in anxiety disorders. J Vestib Res 2006;16:209–15.
85. 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.
86. 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;118:778–82.
87. 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;135:686–93.
88. Cohen HS, Kimball KT, Stewart MG. Benign paroxysmal positional vertigo and comorbid conditions. ORL J Otorhinolaryngol Relat Spec 2004;66:11–5.
89. 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;72:388–92.
90. Rubenstein LZ. Falls in older people: epidemiology, risk factors and strategies for prevention. Age Ageing 2006;35 Suppl 2:ii37–ii41.
91. Casellini CM, Vinik AI. Clinical manifestations and current treatment options for diabetic neuropathies. Endocr Pract 2007;13:550–66.
92. Richardson JK. Factors associated with falls in older patients with diffuse polyneuropathy. J Am Geriatr Soc 2002;50:1767–73.
93. Tilling LM, Darawil K, Britton M. Falls as a complication of diabetes mellitus in older people. J Diabetes Complications 2006;20:158–62.
94. Vibert D, Kompis M, Hausler R. Benign paroxysmal positional vertigo in older women may be related to osteoporosis and osteopenia. Ann Otol Rhinol Laryngol 2003;112:885–9.
95. 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.
96. 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;19:693–7.
97. Gordon CR, Levite R, Joffe V, et al. Is posttraumatic benign paroxysmal positional vertigo different from the idiopathic form? Arch Neurol 2004;61:1590–3.
98. Frohman EM, Kramer PD, Dewey RB, et al. Benign paroxysmal positioning vertigo in multiple sclerosis: diagnosis, pathophysiology and therapeutic techniques. Mult Scler 2003;9:250–5.
99. Frohman EM, Zhang H, Dewey RB, et al. Vertigo in MS: utility of positional and particle repositioning maneuvers. Neurology 2000;55: 1566–9.
100. Gamiz MJ, Lopez-Escamez JA. Health-related quality of life in patients over sixty years old with benign paroxysmal positional vertigo. Gerontology 2004;50:82–6.
101. 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;262:507–11.
102. 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: 637–41.
103. 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. p. 8.
104. Turski P, Seidenwurm D, Davis P, et al. American College of Radiology: Expert Panel on Neuroimaging: vertigo and hearing loss. Reston (VA): American College of Radiology; 2006. p. 8.
105. 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;313:788–92.
106. 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;19:144–50.
107. Brandt T, Dieterich M. VIIIth nerve vascular compression syndrome: vestibular paroxysmia. Baillieres Clin Neurol 1994;3:565–75.
108. Jacobson G, Butcher JA, Newman CW, et al. When paroxysmal positional vertigo isn't benign. J Am Acad Audiol 1995;6:346–9.
109. Kumar A, Patni AH, Charbel F. The Chiari I malformation and the neurotologist. Otol Neurotol 2002;23:727–35.
110. Gizzi M, Riley E, Molinari S. The diagnostic value of imaging the patient with dizziness. A Bayesian approach. Arch Neurol 1996;53: 1299–304.
111. Kentala E, Viikki K, Pyykko I, et al. Production of diagnostic rules from a neurotologic database with decision trees. Ann Otol Rhinol Laryngol 2000;109:170–6.
112. Gordon CR, Shupak A, Spitzer O, et al. Nonspecific vertigo with normal otoneurological examination. The role of vestibular laboratory tests. J Laryngol Otol 1996;110:1133–7.
113. 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;55:1431–41.
114. Bath AP, Walsh RM, Ranalli P, et al. Experience from a multidisciplinary "dizzy" clinic. Am J Otol 2000;21:92–7.

115. 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;44:191–6.
116. Korres SG, Balatsouras DG. Diagnostic, pathophysiologic, and therapeutic aspects of benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 2004;131:438–44.
117. Hughes CA, Proctor L. Benign paroxysmal positional vertigo. Laryngoscope 1997;107:607–13.
118. 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.
119. Del Rio M, Arriaga MA. Benign positional vertigo: prognostic factors. Otolaryngol Head Neck Surg 2004;130:426–9.
120. Rupa V. Persistent vertigo following particle repositioning maneuvers: an analysis of causes. Arch Otolaryngol Head Neck Surg 2004; 130:436–9.
121. Gordon CR, Gadoth N. Benign paroxysmal positional vertigo: who can diagnose it, how should it be treated and where? Harefuah 2005;144:567–71, 97.
122. American Medical Association's Relative Value Scale Upgrade Committee (RUC) 2008 database, version 1. (Based on 2005-06 Medicare Part B data; Centers for Medicare and Medicaid Services). Chicago: American Medical Association; 2008.
123. Havlik RJ. Aging in the eighties, impaired senses for sound and light in persons aged 65 years and over, preliminary data from the supplement on aging to the national health interview survey, United States, January-June 1984. Advanced Data. Vital Health Stat 1986; 125:2.
124. Kentala E, Pyykko I. Vertigo in patients with benign paroxysmal positional vertigo. Acta Otolaryngol Suppl 2000;543:20–2.
125. Stewart MG, Chen AY, Wyatt JR, et al. Cost-effectiveness of the diagnostic evaluation of vertigo. Laryngoscope 1999;109:600–5.
126. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 1992;107:399–404.
127. Uneri A. Falling sensation in patients who undergo the Epley maneuver: a retrospective study. Ear Nose Throat J 2005;84:82, 84–5.
128. Lynn S, Pool A, Rose D, et al. Randomized trial of the canalith repositioning procedure. Otolaryngol Head Neck Surg 1995;113: 712–20.
129. Yimtae K, Srirompotong S, Sae-Seaw P. A randomized trial of the canalith repositioning procedure. Laryngoscope 2003;113:828–32.
130. 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;53:1049–53 48.
131. Sherman D, Massoud EA. Treatment outcomes of benign paroxysmal positional vertigo. Journal of Otolaryngology 2001;30:295–9.
132. Li JC. Mastoid oscillation: a critical factor for success in canalith repositioning procedure. Otolaryngol Head Neck Surg 1995;112: 670–5.
133. 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: 729–33.
134. Wolf M, Hertanu T, Novikov I, et al. Epley's manoeuvre for benign paroxysmal positional vertigo: a prospective study. Clin Otolaryngol Allied Sci 1999;24:43–6.
135. Asawavichianginda S, Isipradit P, Snidvongs K, et al. Canalith repositioning for benign paroxysmal positional vertigo: a randomized, controlled trial. Ear Nose Throat J 2000;79:732–4, 36–7.
136. Angeli SI, Hawley R, Gomez O. Systematic approach to benign paroxysmal positional vertigo in the elderly. Otolaryngol Head Neck Surg 2003;128:719–25.
137. 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;11:918–24.
138. 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;50:366–70.
139. Woodworth BA, Gillespie MB, Lambert PR. The canalith repositioning procedure for benign positional vertigo: a meta-analysis. Laryngoscope 2004;114:1143–6.
140. Teixeira LJ, Machado JN. Maneuvers for the treatment of benign positional paroxysmal vertigo: a systematic review. Rev Bras Otorrinolaringol (Engl Ed) 2006;72:130–9.
141. Ruckenstein MJ. Therapeutic efficacy of the Epley canalith repositioning maneuver. Laryngoscope 2001;111:940–5.
142. 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;135:529–33.
143. 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;115:1667–71.
144. Herdman SJ, Tusa RJ. Complications of the canalith repositioning procedure. Arch Otolaryngol Head Neck Surg 1996;122:281–6.
145. Sridhar S, Panda N. Particle repositioning manoeuvre in benign paroxysmal positional vertigo: is it really safe? J Otolaryngol 2005; 34:41–5.
146. 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.
147. 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;122:179–83.
148. Fife TD. Recognition and management of horizontal canal benign positional vertigo. Am J Otol 1998;19:345–51.
149. Lempert T, Tiel-Wilck K. A positional maneuver for treatment of horizontal-canal benign positional vertigo. Laryngoscope 1996;106: 476–8.
150. Appiani GC, Gagliardi G, Magliulo G. Physical treatment of horizontal canal benign positional vertigo. Eur Arch Otorhinolaryngol 1997;254:326–8.
151. Asprella Libonati G. Diagnostic and treatment strategy of lateral semicircular canal canalolithiasis. Acta Otorhinolaryngol Ital 2005; 25:277–83.
152. Chiou WY, Lee HL, Tsai SC, et al. A single therapy for all subtypes of horizontal canal positional vertigo. Laryngoscope 2005;115:1432–5.
153. Vannucchi P, Giannoni B, Pagnini P. Treatment of horizontal semicircular canal benign paroxysmal positional vertigo. J Vestib Res 1997;7:1–6.
154. Gufoni M, Mastrosimone L, Di Nasso F. [Repositioning maneuver in benign paroxysmal vertigo of horizontal semicircular canal]. Acta Otorhinolaryngol Ital 1998;18:363–7.
155. Appiani GC, Catania G, Gagliardi M, et al. Repositioning maneuver for the treatment of the apogeotropic variant of horizontal canal benign paroxysmal positional vertigo. Otol Neurotol 2005;26:257–60.
156. Sargent EW, Bankaitis AE, Hollenbeak CS, et al. Mastoid oscillation in canalith repositioning for paroxysmal positional vertigo. Otol Neurotol 2001;22.
157. Radtke A, Neuhauser H, von Brevern M, et al. A modified Epley's procedure for self-treatment of benign paroxysmal positional vertigo. Neurology 1999;53:1358–60.
158. Radtke A, von Brevern M, Tiel-Wilck K, et al. Self-treatment of benign paroxysmal positional vertigo: Semont maneuver vs Epley procedure. Neurology 2004;63:150–2.
159. 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;77:980–2.
160. Massoud EA, Ireland DJ. Post-treatment instructions in the nonsurgical management of benign paroxysmal positional vertigo. J Otolaryngol 1996;25:121–5.

161. Roberts RA, Gans RE, DeBoodt JL, et al. Treatment of benign paroxysmal positional vertigo: necessity of postmaneuver patient restrictions. J Am Acad Audiol 2005;16:357–66.
162. Cakir BO, Ercan I, Cakir ZA, et al. Efficacy of postural restriction in treating benign paroxysmal positional vertigo. Arch Otolaryngol Head Neck Surg 2006;132:501–5.
163. 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.
164. Telian SA, Shepard NT. Update on vestibular rehabilitation therapy. Otolaryngol Clin North Am 1996;29:359–71.
165. Whitney SL, Rossi MM. Efficacy of vestibular rehabilitation. Otolaryngol Clin North Am 2000;33:659–72.
166. Cawthorne T. The physiologic basis for head exercises. J Chart Soc Physiother 1944:106–7.
167. Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. Arch Otolaryngol 1980;106:484–5.
168. Brandt T, Steddin S, Daroff RB. Therapy for benign paroxysmal positioning vertigo, revisited. Neurology 1994;44:796–800.
169. Troost BT, Patton JM. Exercise therapy for positional vertigo. Neurology 1992;42:1441–4.
170. Banfield GK, Wood C, Knight J. Does vestibular habituation still have a place in the treatment of benign paroxysmal positional vertigo? J Laryngol Otol 2000;114:501–5.
171. Steenerson RL, Cronin GW. Comparison of the canalith repositioning procedure and vestibular habituation training in forty patients with benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 1996;114:61–4.
172. Hillier SL, Hollohan V. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. Cochrane Database Syst Rev 2007: CD005397.
173. Toledo H, Cortes ML, Pane C, et al. Semont maneuver and vestibular rehabilitation exercises in the treatment of benign paroxysmal postural vertigo. A comparative study. Neurologia 2000;15:152–7.
174. Helminski JO, Janssen I, Kotaspouikis D, et al. Strategies to prevent recurrence of benign paroxysmal positional vertigo. Arch Otolaryngol Head Neck Surg 2005;131:344–8.
175. Kammerlind AS, Ledin TE, Odkvist LM, et al. Effects of home training and additional physical therapy on recovery after acute unilateral vestibular loss—a randomized study. Clin Rehabil 2005;19: 54–62.
176. Blakley BW. A randomized, controlled assessment of the canalith repositioning maneuver. Otolaryngol Head Neck Surg 1994;110: 391–6.
177. Simhadri S, Panda N, Raghunathan M. Efficacy of particle repositioning maneuver in BPPV: a prospective study. Am J Otolaryngol 2003;24:355–60.
178. Hain TC, Uddin M. Pharmacological treatment of vertigo. CNS Drugs 2003;17:85–100.
179. Hain TC, Yacovino D. Pharmacologic treatment of persons with dizziness. Neurol Clin 2005;23:831–53, vii.
180. Carlow TJ. Medical treatment of nystagmus and ocular motor disorders. Int Ophthalmol Clin 1986;26:251–64.
181. Cesarani A, Alpini D, Monti B, et al. The treatment of acute vertigo. Neurol Sci 2004;25 Suppl 1:S26–S30.
182. 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.
183. Salvinelli F, Trivelli M, Casale M, et al. Treatment of benign positional vertigo in the elderly: a randomized trial. Laryngoscope 2004; 114:827–31.
184. 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: 155–8.
185. McClure JA, Willett JM. Lorazepam and diazepam in the treatment of benign paroxysmal vertigo. J Otolaryngol 1980;9:472–7.
186. 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;332:455–9.
187. Hebert C, Delaney JA, Hemmelgarn B, et al. Benzodiazepines and elderly drivers: a comparison of pharmacoepidemiological study designs. Pharmacoepidemiol Drug Saf 2007;16:845–9.
188. Barbone F, McMahon AD, Davey PG, et al. Association of road-traffic accidents with benzodiazepine use. Lancet 1998;352:1331–6.
189. Engeland A, Skurtveit S, Morland J. Risk of road traffic accidents associated with the prescription of drugs: a registry-based cohort study. Ann Epidemiol 2007;17:597–602.
190. 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.
191. Hartikainen S, Lonnroos E, Louhivuori K. Medication as a risk factor for falls: critical systematic review. J Gerontol A Biol Sci Med Sci 2007;62:1172–81.
192. Hien le TT, Cumming RG, Cameron ID, et al. Atypical antipsychotic medications and risk of falls in residents of aged care facilities. J Am Geriatr Soc 2005;53:1290–5.
193. Landi F, Russo A, Liperoti R, et al. Anticholinergic drugs and physical function among frail elderly population. Clin Pharmacol Ther 2007;81:235–41.
194. 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.
195. Baloh RW. Vertigo. Lancet 1998;352:1841–6.
196. Baloh RW. Dizziness: neurological emergencies. Neurol Clin 1998; 16:305–21.
197. Rudolph JL, Salow MJ, Angelini MC, et al. The anticholinergic risk scale and anticholinergic adverse effects in older persons. Arch Intern Med 2008;168:508–13.
198. Dal T, Ozluoglu LN, Ergin NT. The canalith repositioning maneuver in patients with benign positional vertigo. Eur Arch Otorhinolaryngol 2000;257:133–6.
199. Magliulo G, Bertin S, Ruggieri M, et al. Benign paroxysmal positional vertigo and post-treatment quality of life. Eur Arch Otorhinolaryngol 2005;262:627–30.
200. Furman JM, Cass SP. A practical work-up for vertigo. Contemp Intern Med 1995;7:24–7, 31–2, 35–8.
201. 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: 428–35.
202. 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.
203. Monobe H, Sugasawa 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.
204. Bergenius J, Perols O. Vestibular neuritis: a follow-up study. Acta Otolaryngol 1999;119:895–9.
205. Dornhoffer JL, Colvin GB. Benign paroxysmal positional vertigo and canalith repositioning: clinical correlations. Am J Otol 2000;21:230–3.
206. Uneri A. Migraine and benign paroxysmal positional vertigo: an outcome study of 476 patients. Ear Nose Throat J 2004;83:814–5.
207. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain 1984;107(pt 4):1123–42.
208. Chang WC, Hsu LC, Yang YR, et al. Balance ability in patients with benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 2006;135:534–40.
209. Giacomini PG, Alessandrini M, Magrini A. Long-term postural abnormalities in benign paroxysmal positional vertigo. ORL J Otorhinolaryngol Relat Spec 2002;64:237–41.

210. Black FO, Nashner LM. Postural disturbance in patients with benign paroxysmal positional nystagmus. Ann Otol Rhinol Laryngol 1984; 93:595–9.
211. 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;72:366–72.
212. Smouha EE, Roussos C. Atypical forms of paroxysmal positional nystagmus. Ear Nose Throat J 1995;74:649–56.
213. Buttner U, Helmchen C, Brandt T. Diagnostic criteria for central versus peripheral positioning nystagmus and vertigo: a review. Acta Otolaryngol 1999;119:1–5.
214. Sakaida M, Takeuchi K, Ishinaga H, et al. Long-term outcome of benign paroxysmal positional vertigo. Neurology 2003;60:1532–4.
215. Brandt T, Dieterich M. Vestibular falls. J Vestib Res 1993;3:3–14.
216. Gazzola JM, Gananca FF, Aratani MC, et al. Clinical evaluation of elderly people with chronic vestibular disorder. Rev Bras Otorrinolaringol (Engl Ed) 2006;72:515–22.
217. Imbaud Genieys S. Vertigo, dizziness and falls in the elderly. Annales d Oto-Laryngologie et de Chirurgie Cervico-Faciale 2007;124: 189–96.
218. Shaia WT, Zappia JJ, Bojrab DI, et al. Success of posterior semicircular canal occlusion and application of the dizziness handicap inventory. Otolaryngol Head Neck Surg 2006;134:424–30.