TITLE

Clinical Practice Guideline: Benign Paroxysmal Positional Vertigo (Update)

AUTHORS

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Differences from Prior Guideline

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

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

- Enhanced external review process to include public comment and journal peer review
- New algorithm to clarify decision making and action statement relationships
- New recommendation regarding canalith repositioning post-procedural restrictions.
- Expansion of the recommendations regarding radiographic and vestibular testing.
- Removal of the "no recommendation" for audiometric testing.
- A diagnostic and treatment visual algorithm was added.

INTRODUCTION

A primary complaint of dizziness accounts for 5.6 million clinic visits in the United States per year and between 17 and 42% of patients with vertigo ultimately receive a diagnosis of benign paroxysmal positional vertigo (Shappert 1992; Katsarkas 1999; Hanley et al., 2001). Benign paroxysmal positional vertigo (BPPV) is a form of positional vertigo.

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

Traditionally, the terms "benign" and "paroxysmal" have been used to characterize this particular form of positional vertigo. In this context, the descriptor *benign* historically implies that BPPV was a form of positional vertigo not due to any serious central nervous system (CNS) disorder and that there was an overall favorable prognosis for recovery. (Baloh et al 1987). This favorable prognosis is based in part on the fact that BPPV can recover spontaneously in

approximately 20% of patients by one month of follow up and up to 50% at 3 months (Lynn 1995; Burton et al, 2012) However, the clinical and quality-of-life impacts of undiagnosed and untreated BPPV may be far from "benign", as patients with BPPV are at increased risk for falls and impairment in the performance of daily activities (Lopez-Escamez et al, 2005). Furthermore, patients with BPPV experience effects on individual health-related quality of life and utility measures demonstrate that treatment of BPPV results in improvement in quality of life. (Roberts, et al, 2009). The term *paroxysmal* in this context describes the rapid and sudden onset of the vertigo initiated at any time by a change of position thus resulting in BPPV. BPPV has also been termed: benign positional vertigo, paroxysmal positional vertigo, positional vertigo, benign paroxysmal nystagmus, and paroxysmal positional nystagmus. In this guideline, the panel chose to continue to retain the terminology of BPPV as it is the most common terminology encountered in the literature and in clinical practice (Lopez-Escamez et al, 2005).

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). (White et al 2005; Cakir et al 2006; Parnes et al 2003) Posterior canal BPPV is more common than horizontal canal BPPV, constituting approximately 85-95% of BPPV cases. (Parnes et al, 2003) Although debated, posterior canal BPPV is most commonly thought to be due to canalithiasis, wherein fragmented otolith particles (otoconia) entering the posterior canal become displaced and cause inertial changes to the cupula in the posterior canal and thereby resulting in abnormal nystagmus and vertigo when the head encounters motion in the plane of the affected semicircular canal.(Parnes et al, 2003; Parnes & McClure, 1992) Lateral (horizontal) canal BPPV accounts for between 5% and 15% of BPPV cases. (Cakir et al, 2006; Parnes et al, 2003) 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, multi-canal BPPV, and bilateral multi-canal BPPV.

Table 1. Definitions of common terms

Term	**Definition**
Vertigo	An illusory sensation of motion of either the self or the surroundings in the absence of true motion.
Nystagmus	A rapid, involuntary, oscillatory movement of the eyeball.
Vestibular system/apparatus	The sensory system within the inner ear that together with the vestibular nerve and its connections in the brain provides the fundamental input to the brain regarding balance and spatial orientation.
Positional vertigo	Vertigo produced by changes in the head position relative to gravity
Benign paroxysmal positional vertigo (BPPV)	A disorder of the inner ear characterized by repeated episodes of positional vertigo.
Posterior canal BPPV	A form of BPPV in which dislodged inner ear particles in the posterior semicircular canal abnormally influence the balance system producing the vertigo, most commonly diagnosed with the Dix-Hallpike test.
Lateral canal BPPV	A form of BPPV in which dislodged inner ear particles in the lateral semicircular canal abnormally influence the balance system producing the vertigo, most commonly diagnosed by the supine roll test.

Canalithiasis	A theory for the pathogenesis of BPPV that proposes that there are free-floating particles (otoconia) that have moved from the utricle and collect near the cupula of the affected canal, causing forces in the canal leading to abnormal stimulation of the vestibular apparatus.
Cupulolithiasis	A theory for the pathogenesis of BPPV that proposes that otoconial debris attached to the cupula of the affected semicircular canal cause abnormal stimulation of the vestibular apparatus.
Canalith repositioning procedures (CRP)	A group of procedures in which the patient moves through specific body positions designed to relocate dislodged particles within the inner ear for the purpose of relieving symptoms of BPPV. The specific CRP chosen relates to the type of BPPV diagnosed. These have also been termed canalith repositioning maneuvers or canalith repositioning techniques.

GUIDELINE PURPOSE

The primary purposes of this guideline are to improve quality of care and outcomes for BPPV by improving the accurate and efficient diagnosis of BPPV, reducing the inappropriate use of vestibular suppressant medications, decreasing the inappropriate use of ancillary testing such as radiographic imaging and increasing the use of appropriate therapeutic repositioning maneuvers. The guideline is intended for all clinicians who are likely to diagnose and manage patients with BPPV, and 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 suspected or potential diagnosis of BPPV. The pediatric population was not included in the target population in part due to substantially smaller body of evidence on pediatric 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 neurologic or otologic conditions, and that the treatment of the symptom components specifically related to BPPV may still be managed according to the guideline. This guideline will not discuss BPPV affecting the anterior semicircular canal, as this diagnosis is quite rare and its pathophysiology is poorly understood (Kim et al, 2014; Jackson et al, 2007) It will also not discuss benign paroxysmal vertigo of childhood, disabling positional vertigo due to vascular loop compression in the brainstem, or vertigo that arises from changes in head position *not* related to gravity (i.e. vertigo of cervical origin or vertigo of vascular origin). These conditions are physiologically distinct from BPPV.

In 2008, the American Academy of Otolaryngology-Head and Neck Surgery published a multidisciplinary clinical practice guideline: benign positional vertigo (Bhattacharyya et al, 2008). As eight years have elapsed since the publication of that guideline, a multidisciplinary guideline update group was convened to perform an assessment and planned update of that guideline utilizing the most current evidence base. Our goal was to revise the prior guideline with an a priori determined, transparent process, reconsidering a more current evidence base while also taking into account advances in knowledge with respect to BPPV.

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

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

Table 2. Interventions considered in BPPV guideline development.

Diagnosis	clinical history review of the medication list physical examination Dix Hallpike (positional) testing Supine roll test and Bow and lean test side-lying maneuver post head shaking nystagmus audiometry magnetic resonance imaging computed tomography blood tests: complete blood count, serum chemistry, etc. frenzel lenses and infrared goggle testing electronystagmography videonystagmography vestibular evoked myogenic potentials balance and gait testing vestibular function testing computerized posturography orthostatic balance testing vestibular caloric testing

Treatment	watchful waiting/observation education/information/counseling medical therapy (vestibular suppressant medications, benzodiazepines) cervical immobilization with cervical collar prolonged upright position patient self-treatment with home-based maneuvers or rehabilitation Brandt-Daroff exercises Epley maneuver and modifications of the Epley maneuver Semont maneuver Gufoni maneuver physical therapy/vestibular physical therapy spinal manipulative therapy mastoid vibration posterior semicircular canal occlusion (excluded from guideline) singular neurectomy (excluded from guideline) vestibular neurectomy (excluded from guideline)
Prevention	head trauma or whiplash injury as potential causative factors use of helmets to prevent head trauma and/or cervical collars fall prevention

HEALTHCARE BURDEN

Overall, the prevalence of BPPV has been reported to range from 10.7 to 140 per 100,000 population (Mizukoshi et al, 1984; Froehling et al, 1991, van der Zaag-Loonen et al. 2015) however studies of select patients have estimated a prevalence of 900 per 10,000 (Oghalai et al, 2000, Kollen et al, 2012, Kerrigan et al, 2013). Others have reported a lifetime prevalence of 2.4%, a one-year prevalence of 1.6% and a one-year incidence of 0.6%. (von Brevern et al, 2007) Women are more frequently affected than men with a female:male ratio of 2.2 to 1.5:1 (Neuhauser and Lempert, 2009). BPPV is also the most common vestibular disorder across the

lifespan, (Parnes et al, 2003; Nedzelski et al, 1986; Neuhauser, 2007) although the age of onset is most commonly between the fifth and seventh decades of life. (Baloh et al, 1987) 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 greater than 65% of patients with this condition will undergo potentially unnecessary diagnostic testing or therapeutic interventions (Wang, et al, 2014). Therefore, healthcare costs associated with the diagnosis of BPPV alone approach $2 billion per year. Furthermore despite the fact that the natural history of BPPV includes a spontaneous resolution rate ranging from 27 to 50%, this often takes a significant amount of time and almost 86% of patients with BPPV will suffer some interrupted daily activities and lost days at work due to BPPV.(von Brevern et al, 2007; Li et el, 2000) In addition, 68% of patients with BPPV will reduce their workload while 4% will change their job and 6% will quit their job as a result of the condition (Benecke et al 2014). Furthermore, BPPV is more common in older individuals with a correspondingly more pronounced health and quality-of-life impact. It has been estimated that 9% of elderly patients undergoing comprehensive geriatric assessment for non-balance related complaints have unrecognized BPPV. (Oghalai et el, 2000). More recent studies of symptomatic individuals have found BPPV to be present in 40% of geriatric patients seen for dizziness (Ekvall et al 2005; Katsarkas 2008) Others have found a cumulative lifetime incidence of BPPV of approximately 10% with a prevalence of 3.4% of those over age 60. (von Brevern et al, 2007).

Older patients with BPPV experience a greater incidence of falls, depression and impairments of their daily activities. (Oghalai et el, 2000) 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. Among an estimated 7.0 million elderly individuals reporting dizziness in the prior 12 months, 2.0 million (30.1%) reported vertigo and there were 230,000 office visits among the elderly with a diagnosis of BPPV. (Lin & Bhattacharyya, 2012; Lin & Bhattacharyya, 2011) With the increasing age of the United States 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, quality of life and economic impacts, considerable practice variations exist in the management of BPPV across disciplines. (Lawson et al, 2005) These variations relate to both diagnostic strategies for BPPV, timeliness of referral and rates of utilization of various treatment options available for BPPV within and across the various medical specialties and disciplines involved in its management. For example, the utilization of medications for the treatment of BPPV vary substantially among primary care providers and across specialties (Fife et al, 2005) Delays in the diagnosis and treatment of BPPV have both cost and quality-of-life implications for both patients and their caregivers.

Fife and colleagues found that patients with BPPV suffer from delays in diagnosis and treatment on the order of months. (Fife et al, 2005) Other authors have found that only 10-20% of patients with BPPV seen by a physician will receive appropriate repositioning maneuvers (von Brevern 2004, von Brevern 2007). Furthermore, a large number of patients with BPPV will undergo unnecessary diagnostic testing and treatments prior to referral to a specialist. A recent study reported that 70% of patients with BPPV will undergo MRI scanning, 45% will have a CT scan and 41% will have an EKG while 53% will be treated with medications. (Grill et al, 2014)

Therefore, significant improvements in the diagnosis and treatment of patients with BPPV may lead to significant healthcare quality improvements as well as medical and societal cost savings. Such improvements may be achievable with the composition and implementation of a well-constructed clinical practice guideline for BPPV.

METHODS

General methods and literature search

In developing this update of the evidence-based clinical practice guideline, the methods outlined in the AAO-HNSF Guideline Development Manual, 3rd edition were followed explicitly. (Rosenfeld, et al, 2013)

An executive summary of the original BPPV guideline (Bhattacharyya 2008) was sent to a panel of expert reviewers from the fields of general otolaryngology, otology, neurotology, neurology, family practice, nursing, physical therapy, emergency medicine, radiology, audiology, and complimentary medicine who assessed the key action statements to decide if they should be kept in their current form, revised, or removed, and to identify new research that might affect the guideline recommendations. The reviewers concluded that the original guideline action statements remained valid but should be updated with minor modifications. Suggestions were also made for new key action statements.

An information specialist conducted two systematic literature searches using a validated filter strategy to identify clinical practice guidelines, systematic reviews, and randomized controlled trials (RCTs) published since the prior guideline (2008). Search terms used were "Benign Paroxysmal Positional Vertigo"[Mesh] OR "Benign Paroxysmal Positional Vertigo"[tab] OR

"Benign Positional Vertigo"[tiab] OR BPPV[tiab] OR (BPV[tiab] AND vertigo). In certain instances, targeted searches for lower level evidence were performed to address gaps from the systematic searches identified in writing the guideline. The original search was updated from January 2008 to September 2015 to include Medline, National Guidelines Clearinghouse, Canadian Medical Association (CMA) Database, NHS Evidence ENT and Audiology, National Institutes for Health and Care Excellence UK, Australian National Health and Medical Research Council, Guideline Internal Network, Cochrane Database of Systematic Reviews, Excerpta Medica database (EMBASE), Cumulative Index to Nursing and Allied Health (CINAHL), Web of Science, and the Allied and Complimentary Medicine Database (AMED).

1. The initial search for clinical practice guidelines identified two guidelines. Quality criteria for including guidelines were (a) an explicit scope and purpose, (b) multidisciplinary stakeholder involvement, (c) systematic literature review, (d) explicit system for ranking evidence, and (e) explicit system for linking evidence to recommendations. The final dataset retained two guidelines that met inclusion criteria.
2. The initial search for systematic reviews identified 44 systematic reviews or meta-analyses that were distributed to the panel members. Quality criteria for including reviews were (a) relevance to the guideline topic, (b) clear objective and methodology, (c) explicit search strategy, and (d) valid data extraction methods. The final data set retained was 20 systematic reviews or meta-analyses that met inclusion criteria.
3. The initial search for RCTs identified 38 RCTs that were distributed to panel members for review. Quality criteria for including RCTs were (a) relevance to the guideline topic, (b) publication in a peer-reviewed journal, and (c) clear methodology with randomized allocation to treatment groups. The total final data set retained 27 RCTs that met

inclusion criteria.

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

The evidence profile for each statement in the earlier guideline was then converted into an expanded action statement profile for consistency with our current development standards. (Rosenfeld 2013) Information was added to the action statement profiles regarding the quality improvement opportunity to which the action statement pertained, the guideline panel's level of confidence in the published evidence, differences of opinion among panel members, intentional vagueness, and any exclusion to which the action statement does not apply. New key action statements were developed using an explicit and transparent a priori protocol for creating actionable statements based on supporting evidence and the associated balance of benefit and harm. Electronic decision support (BRIDGE-Wiz, Yale Center for Medical Informatics, CT) software was used to facilitate creating actionable recommendations and evidence profiles (Shiffman 2012).

The updated guideline then underwent Guideline Implementability Appraisal (GLIA) to appraise adherence to methodologic standards, to improve clarity of recommendations, and to predict potential obstacles to implementation (Shiffman, et al 2005). The GUG received

summary appraisals and modified an advanced draft of the guideline based on the appraisal. The final draft of the updated clinical practice guideline was revised based on comments received during multidisciplinary peer review, open public comment, and journal editorial peer review. A scheduled review process will occur at five years from publication, or sooner if new compelling evidence warrants earlier consideration.

Classification of evidence-based statements

Classification of evidence-based statements

Guidelines are intended to reduce inappropriate variations in clinical care, to produce optimal health outcomes for patients, and to minimize harm. The evidence-based approach to guideline development requires that the evidence supporting a policy be identified, appraised, and summarized and that an explicit link between evidence and statements be defined. Evidence-based statements reflect both the *quality of evidence* and the *balance of benefit and harm* that is anticipated when the statement is followed. The definitions for evidence-based statements are listed in Tables 3 and 4.

Table 3. Strength of action terms in guideline statements and implied levels of obligation

Strength	**Definition**	**Implied obligation**

Strong Recommendation	A strong recommendation means the benefits of the recommended approach clearly exceed the harms (or, in the case of a strong negative recommendation, that the harms clearly exceed the benefits) and that the quality of the supporting evidence is high (Grade A or B)*. In some clearly identified circumstances, strong recommendations may be made based on 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, in the case of a negative recommendation, that the harms exceed the benefits), but the quality of evidence is not as high (Grade B or C)*. In some clearly identified circumstances, recommendations may be made based on 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 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.

*See Table 4 for definitions of evidence grades

Table 4. Aggregate grades of evidence by question type*

Grade	CEBM level	Treatment	Harm	Diagnosis	Prognosis
A	1	Systematic review‡ of randomized trials	Systematic review‡ of randomized trials, nested case-control studies, or observational studies with dramatic effect‡	Systematic review‡ of cross-sectional studies with consistently applied reference standard and blinding	Systematic revie of inception coh studies†
B	2	Randomized trials, or observational studies with dramatic effects or highly consistent evidence	Randomized trials, or observational studies with dramatic effects or highly consistent evidence	Cross-sectional studies with consistently applied reference standard and blinding	Inception cohort studies†
C	3-4	Non-randomized or historically controlled studies, including case-control and observational studies	Non-randomized controlled cohort or follow-up study (post-marketing surveillance) with sufficient numbers to rule out a common harm; case-series, case-control,	Non-consecutive studies, case-control studies, or studies with poor, non-independent, or inconsistently applied reference standards	Cohort study, control arm of a randomized trial case series, or ca control studies; poor quality prognostic coho study

			or historically controlled studies		
D	5	Case reports, mechanism-based reasoning, or reasoning from first principles			
X	n/a	Exceptional situations where validating studies cannot be performed and there is a clear preponderance of benefit over harm			

CEBM, Oxford Centre for Evidence-Based Medicine

*Adapted from Howick and coworkers. (2011)

†A group of individuals identified for subsequent study at an early, uniform point in the course of the specified health condition, or before the condition develops

‡A systematic review may be downgraded to level B because of study limitations, heterogeneity, or imprecision

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 (Eddy, 1992). Clinicians should always act and decide in a way that they believe will best serve their individual patients' interests and needs, regardless of guideline recommendations. Guidelines represent the best judgment of a team of experienced clinicians and methodologists addressing the scientific evidence for a particular topic (AAP SCQIM, 2004).

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

Financial disclosure and conflicts of interest

The cost of developing this guideline, including travel expenses of all panel members, was covered in full by the AAO-HNSF. Potential conflicts of interest for all panel members in the past 5 years were compiled and distributed before the first conference call and were updated at each subsequent call and in-person meeting. After review and discussion of these disclosures (Choudry, et al, 2002), 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. Lastly, 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 (Detsky, 2006).

Guideline Key Action Statements

Each evidence-based statement is organized in a similar fashion: a key action statement is in bold, followed by the strength of the recommendation in italics. Each key action statement is followed by an 'action statement profile' that explicitly states the quality improvement opportunity, aggregate evidence quality, level of confidence in evidence (high, medium, low), benefit, harms, risks, costs and a benefits-harm assessment. Additionally, there are statements of any value judgments, the role of patient preferences, clarification of any intentional vagueness by the panel, exceptions to the statement, any differences of opinion, and a repeat statement of the strength of the recommendation. Several paragraphs subsequently discuss the evidence base supporting the statement. An overview of each evidence-based statement in this guideline can be

found in Table 5.

The role of patient preferences in making decisions deserves further clarification. The GUG classified the role of patient preference based upon consensus among the group as "none, small, moderate or large". For some statements, where the evidence base demonstrates clear benefit, although the role of patient preference for a range of treatments may not be relevant (such as with intraoperative decision-making), clinicians should provide patients with clear and comprehensible information on the benefits in order to facilitate patient understanding and shared-decision making, which in turn leads to better patient adherence and outcomes. In cases where evidence is weak or benefits unclear, the practice of shared decision-making, again where the management decision is made by a collaborative effort between the clinician and an informed patient, is extremely useful. Factors related to patient preference include (but are not limited to) absolute benefits, adverse effects, cost of drugs or procedures, and frequency and duration of treatment, as well as certain less tangible factors such as religious and/or cultural beliefs or personal levels of desire for intervention.

Table 5. Summary of guideline key action statements

Statement	**Action**	**Strength**

1a. Diagnosis of posterior canal BPPV	Clinicians should diagnose posterior semicircular canal BPPV when vertigo associated with torsional, up-beating 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 with the affected ear down. The maneuver should be repeated with the opposite ear down if the initial maneuver is negative	Strong recommendation
1b. Diagnosis of lateral (horizontal) canal BPPV	If the patient has a history compatible with BPPV and the Dix-Hallpike test exhibits horizontal or no nystagmus, the clinician should perform, or refer to a clinician who can perform, a supine roll test to assess for lateral semicircular canal BPPV.	Recommendation
2a. Differential diagnosis	Clinicians should differentiate, or refer to a clinician who can differentiate, BPPV from other causes of imbalance, dizziness and vertigo.	Recommendation
2b. Modifying factors	Clinicians should assess patients with BPPV for factors that modify management including impaired mobility or balance, central nervous system disorders, a lack of home support, and/or increased risk for falling.	Recommendation
3a. Radiographic testing	RADIOGRAPHIC testing: Clinicians should not obtain radiographic imaging in a patient who meets diagnostic criteria for BPPV in the absence of additional signs and/or symptoms inconsistent with BPPV that warrant imaging.	Recommendation (against)

3b. Vestibular testing	Clinicians should not order vestibular testing in a patient who meets diagnostic criteria for BPPV in the absence of additional vestibular signs and/or symptoms inconsistent with BPPV that warrant testing.	Recommendation (against)
4a. Repositioning procedures as initial therapy	Clinicians should treat, or refer to a clinician who can treat, patients with posterior canal BPPV with a canalith repositioning procedure.	Strong recommendation
4b. Post procedural restrictions	Clinicians should not recommend post- procedural postural restrictions after canalith repositioning procedure for posterior canal BPPV.	Strong recommendation (against)
4c. Observation as initial therapy	Clinicians may offer observation with follow up as initial management for patients with BPPV.	Option
5. Vestibular rehabilitation therapy	The clinician may offer vestibular rehabilitation, either self-administered or with a clinician, in the treatment of BPPV.	Option
6. Medical therapy	Clinicians should not routinely treat BPPV with vestibular suppressant medications such as antihistamines and/or benzodiazepines.	Recommendation (against)
7a. Outcome Assessment	Clinicians should reassess patients within 1 month after an initial period of observation or treatment to document resolution or persistence of symptoms.	Recommendation
7b. Evaluation of treatment failure	Clinicians should evaluate or refer to a clinician who can evaluate, patients with persistent symptoms for unresolved BPPV and/or underlying peripheral vestibular or central nervous system disorders.	Recommendation

8. Education	Clinicians should educate patients regarding the impact of BPPV on their safety, the potential for disease recurrence and the importance of follow-up.	Recommendation

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1a. DIAGNOSIS OF POSTERIOR SEMICIRCULAR CANAL BPPV: Clinicians should diagnose posterior semicircular canal BPPV when vertigo associated with torsional, up-beating 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 with the affected ear down. The maneuver should be repeated with the opposite ear down if the initial maneuver is negative. *Strong recommendation based on diagnostic studies with minor limitations and a preponderance of benefit over harm.*

Action Statement Profile

- Quality improvement opportunity: Promoting accurate and efficient diagnosis of BPPV (National Quality Strategy domains: promoting effective prevention/treatments, affordable quality care)
- Aggregate evidence quality: Grade B, based on diagnostic studies with minor limitations
- Level of confidence in the evidence: High
- Benefits: Improved diagnostic accuracy and efficiency
- Risks, harms, costs: Risk of provoking temporary symptoms of BPPV
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Conclusion that paroxysmal positional nystagmus induced by the Dix-

Hallpike maneuver confirms the diagnosis of BPPV and is the gold standard test for diagnosis. The panel emphasized that a history of positional vertigo alone is not adequate to make the diagnosis of posterior canal BPPV

- Role of patient preferences: Small
- Intentional vagueness: None
- Exceptions: Patients with physical limitations including cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, known cerebrovascular disease and the morbidly obese
- Policy level: Strong recommendation
- Differences of opinion: None

Supporting Text

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

Posterior semicircular canal BPPV is diagnosed when (1) patients report a history of vertigo provoked by changes in head position relative to gravity and (2) when, on physical examination, characteristic nystagmus is provoked by the Dix-Hallpike maneuver (Table 6).

Although most cases of BPPV are due to freely mobile calcium carbonate material within the lumen of the affected semicircular canal (so-called canalolithiasis), a form of posterior canal BPPV due to calcium carbonate material actually attached to the cupula (cupulolithiasis) may occur which results in nystagmus that may persist for > 1 min. (von Brevern 2015).

Table 6: Diagnostic criteria for posterior canal BPPV

History	Patient reports repeated episodes of vertigo with changes in head position relative to gravity
Physical Examination	Each of the following criteria are fulfilled: • Vertigo associated with torsional (rotatory), up-beating (towards the forehead) nystagmus is provoked by the Dix-Hallpike test • There is a latency period between the completion of the Dix-Hallpike maneuver and the onset of vertigo and nystagmus • The provoked vertigo and nystagmus increase and then resolve within 60 seconds from the onset of the nystagmus

HISTORY

Vertigo has been defined as an "illusory sensation of motion of either the self or the surroundings". (Blakely & Goebel, 2001) 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 every day activities and commonly occur when rolling over in bed or when the patient is tilting the head to look upward (e.g. to place an object on a shelf higher than the head) or bending forward (e.g. to tie his or her shoes). (von Brevern et al, 2007; Furman & Cass, 1999; Dix & Hallpike, 1952;

Whitney et al, 2005)

Patients with BPPV most commonly report discrete, episodic periods of vertigo lasting one minute or less and often report modifications or limitations of their general movements to avoid provoking the vertiginous episodes.(Ruckenstein & Shepard, 2007) 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”.(Katsarkas, 1999; von Brevern et al, 2007; Furman & Cass, 1999; Herdman, 1997; Macias et al, 2000; Cohen, 2004; Haynes et al, 2002; Blatt et al, 2000;p Norre, 1995) Approximately 50% of patients also report subjective imbalance between the classic episodes of BPPV.(von Brevern et al, 2007) In contrast, a history of vertigo *without* associated lightheadedness may increase the a priori likelihood of a diagnosis of posterior canal BPPV.(Oghalai et al, 2000) 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.(Norre, 1995)

Other authors have loosened the historical criteria required for a BPPV diagnosis and have coined the term "subjective BPPV" without a positive Dix-Hallpike test. (Haynes et al, 2002; Nunez et al, 2000) However, in clinical practice there is a practical need to balance inclusiveness of diagnosis with accuracy of diagnosis. Given that the majority of treatment trials and systematic reviews of BPPV require both a history of episodic positional vertigo symptoms and a positive Dix-Hallpike test, history alone is insufficient to render an accurate diagnosis of BPPV.

PHYSICAL EXAMINATION

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

The nystagmus produced by the Dix-Hallpike maneuver in posterior canal BPPV typically displays 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 nystagmus onset with this maneuver is largely unspecified in the literature, but the panel felt that a typical latency period would range from 5-20 seconds. In rare cases, the latency period may be as long as one minute (Baloh et al, 1987). Second, the provoked subjective vertigo and the nystagmus increase and then resolve within 60 seconds from the nystagmus onset.

The fast component of the nystagmus provoked by the Dix-Hallpike maneuver demonstrates a characteristic mixed torsional and vertical movement (often described as upbeating-torsional) with the upper pole of the eye beating toward the dependent ear and the vertical component beating toward the forehead (when the eyes positioned looking straight forward in the mid-orbit when the provoking position is assumed) (**Figure 1**).(Furman & Cass, 1999; Honrubia et al, 1999) 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. After the patient returns to the upright head position, the nystagmus is again commonly observed, and the direction of the nystagmus may be reversed.

Another classic feature associated with posterior canal BPPV is that the nystagmus

typically fatigues (a reduced nystagmus response) when the maneuver is repeated. (Dix & Hallpike, 1952; Honrubia et al, 1999) However, repeating the Dix-Hallpike maneuver to demonstrate fatigability is not recommended because it unnecessarily subjects patients to repeated vertigo symptoms, which is discomforting. Furthermore, repeating Dix-Hallpike maneuvers may interfere with the immediate bedside treatment of BPPV. (Furman & Cass, 1999) Therefore, the panel did not include nystagmus fatigability as a diagnostic criterion.

In addition to posterior canal BPPV, patients may rarely have anterior canal BPPV. Even though anterior canal BPPV is uncommon accounting for 1-3 % of cases (Heidenreich 2011), it is important to recognize the direction of the vertical component of the provoked torsional nystagmus to make the correct diagnosis. A down-beating vertical component in addition to the torsional nystagmus towards the dependent ear could imply anterior canal rather than posterior canal BPPV (Casani et al 2011, Lopez-Escamez et al 2006, Heidenreich et al 2011). This diagnosis should be considered with caution because down-beating positional nystagmus related to brainstem or cerebellar lesion can produce a similar pattern and should be ruled out. (Fife 2009)

PERFORMING THE DIX-HALLPIKE DIAGNOSTIC MANEUVER

The Dix-Hallpike maneuver is performed by the clinician moving the patient through a set of specified head positions to elicit the expected characteristic nystagmus of posterior canal BPPV (**Figure 1**). (Furman & Cass, 1999; Dix & Hallpike, 1952) Before beginning the maneuver, the patient should be counseled regarding the upcoming movements and that they may experience a sudden onset of intense subjective vertigo, possibly with nausea, which should subside within 60 seconds. Since the patient is going to be placed in the supine position

relatively quickly with the head position slightly below the body, the patient should be oriented so that when placed supine, the head can "hang" with support off the posterior edge of the examination table by about 20 degrees. The examiner should ensure that he/she can support the patient's head and guide the patient through the maneuver safely and securely, without the examiner losing support or balance.

1. The maneuver begins with the patient in the upright seated position with the examiner standing at the patient's side. (Furman & Cass, 1999) If present, the patient's eyeglasses should be removed. We initially describe the maneuver to test the right ear as the source of the posterior canal BPPV.

2. The examiner rotates the patient's head 45° to the right to align the posterior semicircular canal with the mid sagittal plane of the body, and with manual support maintains the 45° head turn to the right during the next part of the maneuver. The patient is instructed to keep the eyes open. Fairly quickly, the examiner moves the patient from the seated to the supine right-ear down position and then extends the patient's neck slightly (approximately 20° below the horizontal plane) so that the chin is pointed slightly upward with the head hanging off the edge of the table (supported by the examiner). The examiner observes the patient's eyes for the latency, duration, and direction of the nystagmus. (Norre & Beckers, 1988; White et al, 2005) 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.

3. After the resolution of the subjective vertigo and the nystagmus, if present, the patient may be slowly returned to the upright position. During the return to the upright position, a reversal of the nystagmus may be observed and should be allowed to resolve.

4. If the initial result for the right side is negative, the Dix-Hallpike maneuver (steps 1-4) should then be repeated for the left side, with the left ear arriving at the dependent position. (Nunez et al, 2000) Again, the examiner should inquire about subjective vertigo and identify objective nystagmus, when present. This completes the Dix-Hallpike test.

The Dix-Hallpike maneuver is considered the gold standard test for the diagnosis of posterior canal BPPV. (Fife et al, 2008) It is the most common diagnostic criterion required for entry into clinical trials and for inclusion of such trials in meta-analyses. (Hilton & Pinder, 2004; Cohen & Kimball, 2005) The lack of an alternative external gold standard to the Dix Hallpike maneuver limits the availability of rigorous sensitivity and specificity data. Although it is considered the gold standard test for posterior canal BPPV diagnosis, its accuracy may vary between specialty and non-specialty clinicians. Lopez-Escamez et al, have reported a sensitivity of 82% and specificity of 71% for the Dix-Hallpike maneuvers in posterior canal BPPV, primarily among specialty clinicians.(Lopez-Escamez et al, 2000) In the primary care setting, Hanley and O'Dowd have reported a positive predictive value for a positive Dix-Hallpike test of 83% and a negative predictive value of 52% for the diagnosis of BPPV.(Hanley & O'Dowd, 2002) Therefore, a negative Dix-Hallpike maneuver does not necessarily rule out a diagnosis of posterior canal BPPV. Because of the lower negative predictive values, it has been suggested that

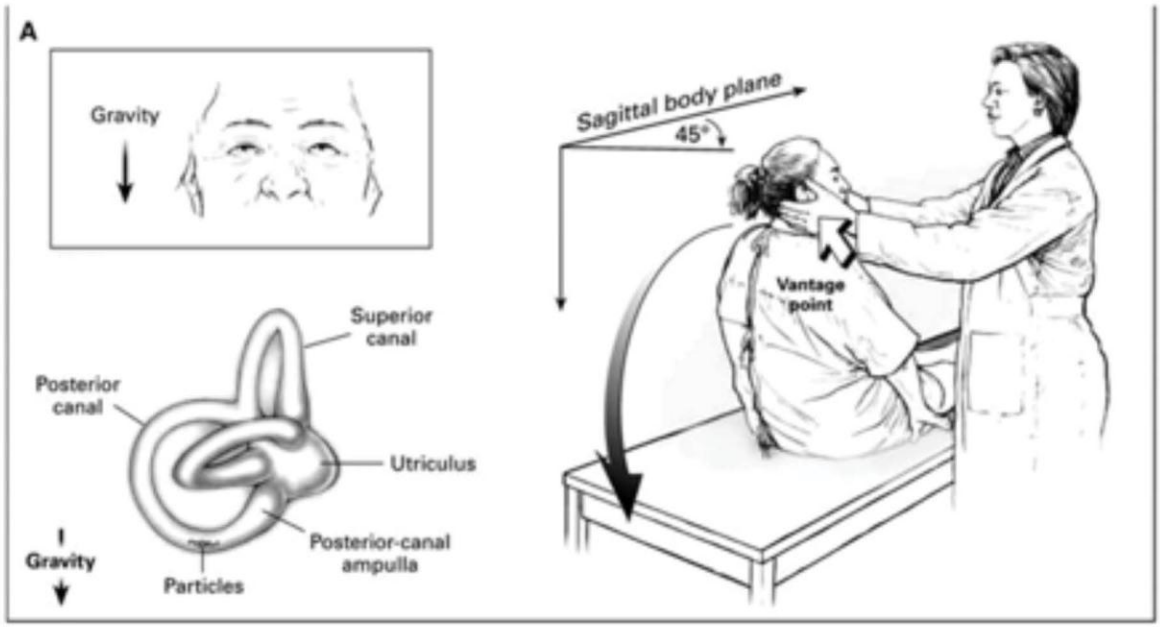
the Dix-Hallpike maneuver may need to be repeated at a separate visit in order to confirm the diagnosis and to avoid a false negative result. (Nunez et al, 2000; Viirre et al, 2005; Norre, 1994)

Factors that may affect the diagnostic accuracy of the Dix-Hallpike maneuver include the speed of head movements during the test, time of day, and the angle of the occipital plane during the maneuver. (Nunez et al, 2000) The Dix-Hallpike maneuver, may in certain circumstances be performed bilaterally in order to determine which ear(s) is(are) involved, particularly if the diagnosis is not clear with the first performance of the maneuver. (Nunez et al, 2000) In a small percentage of cases, the Dix-Hallpike maneuver may be bilaterally positive (i.e. 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. (Katsarkas, 1999)

While the Dix-Hallpike maneuver is the test of choice to confirm the diagnosis of posterior canal BPPV, it should be avoided in certain circumstances. Although there are no documented reports of vertebrobasilar insufficiency (VBI) 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.(Whitney & Morris, 2006) Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity.(Whitney et al, 2005; Whitney & Morris, 2006) Patients who are obese may be difficult for a single examiner to fully support the head through the maneuver and additional assistance may be required. For patients with the above concerns or other physical limitations, special tilting examination tables may allow the safe performance of the Dix-Hallpike maneuver. Such patients

may benefit from referral to more specialized clinicians and/or facilities with additional resources.

Figure 1: **Diagrammatic representation of performance of the Dix-Hallpike maneuver for the diagnosis of posterior canal BPPV (adapted from Fife et al, 2008) In Panel A, the examiner stands at the patient's right side and rotates the patient's head 45° to the right to align the right posterior semicircular canal with the sagittal plane of the body. In Panel B, the examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient's neck 20° so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The arrows in the inset depict the direction of nystagmus in patients with typical benign paroxysmal positional vertigo. A presumed location in the labyrinth of the free floating debris thought to cause the disorder is also shown.**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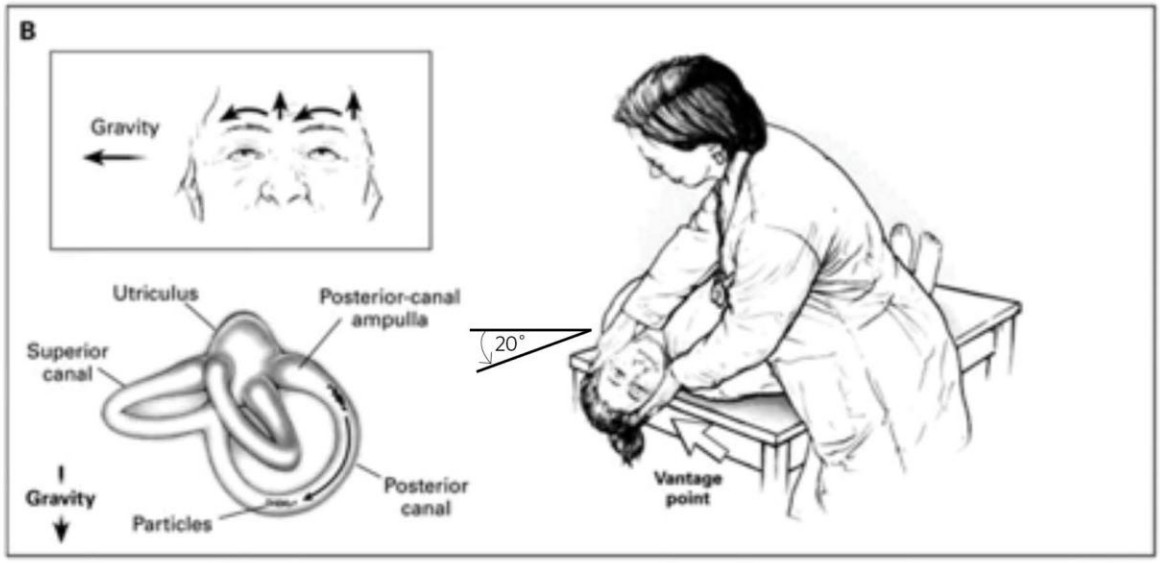
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1b. DIAGNOSIS OF LATERAL (HORIZONTAL) SEMICIRCULAR CANAL BPPV. If the patient has a history compatible with BPPV and the Dix-Hallpike test exhibits horizontal or no nystagmus, the clinician should perform, or refer to a clinician who can perform, a supine roll test to assess for lateral semicircular canal BPPV. *Recommendation based on diagnostic studies with limitations and a preponderance of benefit over harm.*

Action Statement Profile

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

dysfunction, spinal cord injuries, and the morbidly obese

- Policy level: Recommendation
- Differences of opinion: None

Supporting Text

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

Incidence. Lateral semicircular canal BPPV is the second most common type of BPPV. (Imai et al, 2005; Steenerson et al 2005; Moon et al, 2006) Several studies have cited an incidence of approximately 5-22% in populations referred for evaluation and treatment of BPPV. (White et al, 2005; De La Meilleure 1996; Cakir et al, 2006; Hornibrook, 2004; Han et al, 2006; Caruso & Nuti, 2005; Casani 2011). The wide range of incidence of lateral semicircular canal BPPV reported in the literature is probably a function of how soon after the onset of vertigo the patient can be seen at each institution. Lateral semicircular canal BPPV tends to self-resolve more quickly than posterior semicircular canal BPPV (Imai 2005) so clinics seeing patients after more time has elapsed since symptom onset will likely see a lower percentage of the lateral semicircular canal form of BPPV cases and proportionally more posterior semicircular canal.

Lateral semicircular canal BPPV may occur following performance of the canalith repositioning procedure (e.g. Epley maneuver) for an initial diagnosis of posterior semicircular canal BPPV. This transition from posterior semicircular canal BPPV to lateral semicircular canal BPPV is thought to occur as freely mobile calcium carbonate material originating from otoconia of the utricle moves from the posterior semicircular canal to the lateral semicircular

canal (so called "canal conversion"). Since this type of transition is possible but uncommon, clinicians should be aware of lateral semicircular canal BPPV and its diagnosis. (White et al, 2005)

Distinguishing features. Lateral semicircular canal BPPV differs from the more common posterior semicircular canal BPPV in two important ways. First, the nystagmus elicited by the supine roll test in lateral semicircular canal BPPV is predominantly horizontal whereas the nystagmus from the Dix-Hallpike test in posterior semicircular canal BPPV is upbeating and torsional. Second, the vertigo and nystagmus are evoked by turning the head side to side while supine (supine head roll test, Figure 2) whereas vertigo and nystagmus are induced by the Dix Hallpike maneuver in the cases of posterior semicircular canal BPPV. Patients with a history compatible with BPPV (that is, repeated episodes of vertigo produced by changes in head position relative to gravity) who do not appear to have posterior semicircular canal BPPV by Dix Hallpike positioning, should be tested for lateral semicircular canal BPPV. The patient's presenting symptomatic report of positional dizziness due to lateral semicircular canal BPPV is often indistinguishable from posterior semicircular canal BPPV. (Steenerson et al, 2005; Fife 2012)

Supine head roll test (Pagnini-Lempert or Pagnini-McClure roll test). The supine head roll test is the preferred maneuver to diagnose lateral semicircular canal BPPV. (Cakir et al, 2006; Fife 2012; Nuti et al, 1998, Casani 2011) The supine roll test is performed by initially positioning the patient supine with the head in neutral position followed by quickly rotating the head 90° to one side with the clinician observing the patient's eyes for nystagmus (**Figure 2**). After the nystagmus subsides (or if no nystagmus is elicited), the head is then returned to the straight face-up supine position. After any additional elicited nystagmus has subsided, the head

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

Nystagmus characteristics of lateral canal BPPV. Two potential nystagmus findings may occur with this maneuver reflecting two types of lateral semicircular canal BPPV. Both types are so-called direction changing positional nystagmus. That is, the direction of the positional nystagmus changes with changes in the head position. (White et al, 2005; Nuti et al, 1998; Fife 2012; Tirelli & Russolo, 2004)

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

(B) APOGEOTROPIC TYPE: Less commonly, the roll test results in a horizontal nystagmus beating toward the uppermost ear (apogeotropic nystagmus). Upon rolling to the opposite side, the nystagmus will change direction, again beating toward the uppermost ear. It seems likely that when lateral semicircular canal BPPV exhibits the apogeotropic form of nystagmus, the

calcium carbonate debris is located adherent to (cupulolithiasis) or close to the ampulla of the semicircular canal. (Baloh 1993, Casani 2011)

Identifying the affected side. Effective treatments for lateral semicircular canal BPPV are somewhat predicated on knowing which side is affected, although it is recognized that determining the affected side can be complex and may require specialty referral after the initial diagnosis is made. Table 7 outlines some of the methods for determining which side is affected in lateral canal BPPV. The supine roll test is the most commonly utilized method for determining the affected ear in therapeutic trials of lateral semicircular canal BPPV.(Steenerson et al, 2005; Han et al, 2006, Lee 2007, Mandala 2013) Among the two types of lateral semicircular canal BPPV, the geotropic variant is the most common and the most amenable to treatment.(Steenerson et al, 2005; Nuti et al, 1998; Casani et al, 2011) Despite using some of the methods described in Table 7, clear lateralization remains unclear in about 20% of cases (Lee 2007, Fife 2012, Hwang 2015). In such situations, one may simply treat one side and then then other. Alternatively, other testing methods such as the Bow and lean procedure (Table 7) may be applied to add to the diagnosis certainty of side of involvement.

Risk and benefit analysis. Reports of harm or patient injury from the performance of the supine roll test were not identified in the literature review although many authors simply stated that patients who could not tolerate positional maneuvers were excluded. Care should also be exercised in patients with the same exclusionary criteria for the Dix Hallpike maneuver.(Whitney et al, 2005; Whitney & Morris, 2006) The benefit of performing the supine roll test is that it allows clinicians to confirm a diagnosis of lateral semicircular canal BPPV quickly and efficiently.(White et al, 2005; Fife et al, 2008) It also allows clinicians to more accurately and

comprehensively diagnose positional vertigo that is not due to the posterior canal whereas without supine roll testing, patients with lateral semicircular canal BPPV might be diagnostically missed if only traditional Dix-Hallpike testing were done. Further benefit may be realized if the supine roll test is done and the diagnosis recognized obviating unnecessary or unhelpful diagnostic testing.

Figure 2

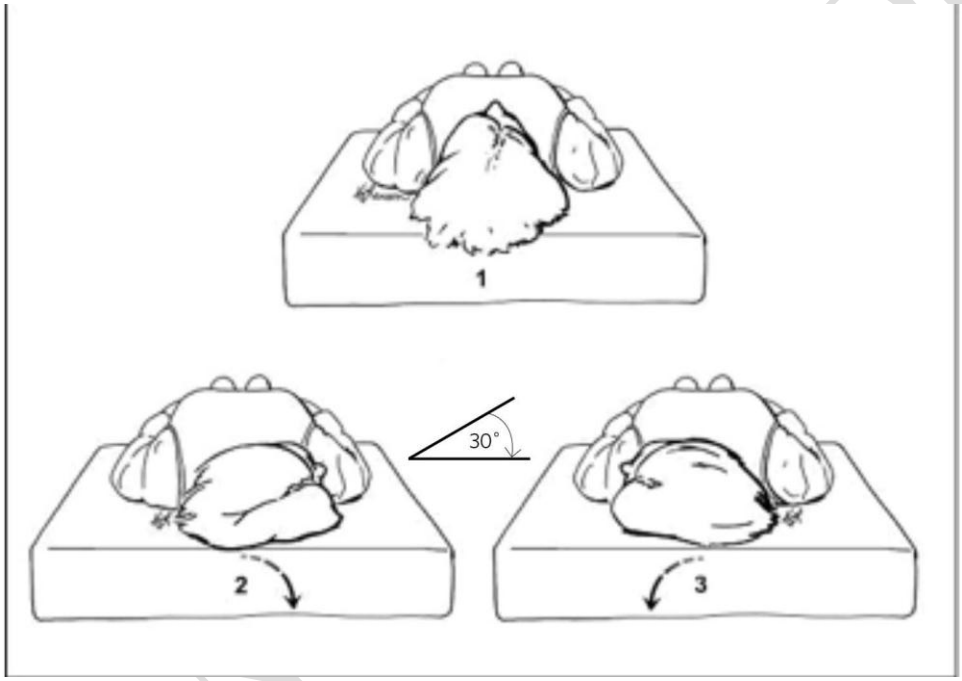

Figure 2: Diagrammatic views of the supine roll test. (1) indicates the patient in the starting neutral position. The patient's head is turned rapidly to the right side (2) examining for characteristic nystagmus. Then the head is returned to the face-up position (1) allowing all nystagmus to subside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

Table 7. Selected methods to determine the affected ear in lateral canal BPPV.

Technique or Circumstance		Conclusion regarding the affected ear
Supine roll testing (Figure 2) reveals a direction changing nystagmus that is either geotropic (beating toward the ground) or apogeotropic (beating away from the ground) and is distinctly stronger on one side than the other (Nuti 2005, Lee 2007, Casani 2011, Fife 2012)		Geotropic form: the side with the **strongest nystagmus** is the affected ear; Apogeotropic form: the side **opposite** the strongest nystagmus is the affected ear.
Posterior canal BPPV torsional upbeating nystagmus **converts to strongly horizontal nystagmus (**lateral canal BPPV) during positioning (Fife 2012)		Same ear as was affected by the posterior semicircular canal BPPV
Patient is moved from sitting to straight supine facing up results in transient horizontal nystagmus (**lying-down nystagmus*)** (Casani 2011, Nuti 2005, Lee 2007, Asprella-Libonati 2008, Koo 2006)		Geotropic: Nystagmus beats **away** from the affected ear Apogeotropic: Nystagmus beats **toward** the affected ear
With the patient in the straight supine position, the patient then sits up and the head bends down as a "Head Pitch Test" (head-bending nystagmus) (Hwang 2015, Kim 2012, Asprella-Libonati 2008)		Geotropic: Nystagmus usually beats **toward** the affected ear Apogeotropic: Nystagmus beats **away** from the affected ear (opposite of lying-down nystagmus.)
Bow and lean test (BLT)* in which the direction of nystagmus is noted when the patient bends the head forward facing down (bowing) and when facing upward (leaning). (Lee 2010, Choung 2006)		**Geotropic:** bowing position (face down): nystagmus toward the affected ear leaning position (face up): nystagmus beats **away** from the affected ear. **Apogeotropic:** (reverse of geotropic type) bowing (face down): nystagmus beats away from the affected ear Leaning (face up) nystagmus: beats **toward** the affected ear.

*The supine head roll test will still be needed to determine if there is a pattern of geotropic or apogeotropic direction changing nystagmus.

2a. DIFFERENTIAL DIAGNOSIS: Clinicians should differentiate, or refer to a clinician who can differentiate, BPPV from other causes of imbalance, dizziness and vertigo.

Recommendation based on observational studies and a preponderance of benefit over harm.

Action Statement Profile

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

Supporting Text

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

Despite being the most common cause of peripheral vertigo, (Froehling et al, 2000) BPPV is still often under-diagnosed or misdiagnosed. (von Brevern et al, 2004) Other causes of vertigo which may be confused with BPPV can be divided into otologic, neurologic and other entities. Among patients presenting with dizziness, the frequency of various causes depends on the setting. In a German telephone survey of over 1000 patients with dizziness, BPPV accounted for 8% of cases. (von Brevern 2007) In an analysis of nearly 10,000 US emergency department patients with dizziness, nearly half of patients had a medical (non-vestibular and non-neurological) diagnosis. (Newman Toker 2008) Only a third of patients were given a vestibular-related diagnosis. In a British general practice setting, evaluation of patients presenting with vertigo, BPPV has been found to account for 42% of cases followed by vestibular neuritis (41%), Meniere's disease (10%), vascular causes (3%) and other causes (3%). (Hanley & O'Dowd, 2002) In subspecialty populations, BPPV accounts for 20-53% of patients referred to ENT specialty clinics for dizziness. (Luscher 2014).

The most common diagnoses that require distinction from BPPV are listed in Table 8. These conditions require distinction from BPPV as their natural history, treatment and potential for serious medical sequelae are significantly different from BPPV. Patients with BPPV may not specifically describe true vertigo and may complain of lightheadedness or non-specific dizziness and thus the clinician may need to initially consider a broader differential diagnosis. (Lawson 2005). BPPV has been described as occurring in conjunction with, or as a consequence of, other vestibular disorders as well, such as Meniere's disease and vestibular neuritis. (Karlberg et al, 2000) 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.

Recent studies emphasize that taking a history that focuses on timing and triggers of a patient's dizziness is more important that the specific descriptor that a patient uses (Newman Toker 2007, Kerber 2015, Bisforff 2015, Newman Toker 2015) Timing (acute versus episodic versus chronic) and triggers (discrete trigger versus spontaneous) of the dizziness and its evolution over time defines four distinct vestibular syndromes. (Newman Toker 2015) (Table 9): These include: an acute vestibular syndrome (AVS), triggered episodic vestibular syndrome (t-EVS), spontaneous episodic vestibular syndrome (s-EVS) and chronic vestibular syndrome (CVS). Each of these entities has its own differential diagnosis, with BPPV fitting the t-EVS criteria given its positional trigger and brief episodic occurrences of vertigo.

OTOLOGIC DISORDERS

Whereas BPPV is characterized by acute, discrete episodes of brief positional vertigo without associated hearing loss, other otologic disorders causing vertigo may be differentiated by their clinical characteristics including temporal pattern and the presence or absence of hearing loss. (Kentala & Rauch, 2003) Meniere's disease is characterized by discrete episodic attacks, each attack exhibiting a characteristic clinical constellation of sustained vertigo with fluctuating hearing loss, aural fullness, and tinnitus in the affected ear.(Baloh et al, 1987; Sajjadi 2008) As opposed to BPPV, the duration of vertigo in an episode of Meniere's disease typically lasts longer (usually on the order of hours), is typically more disabling due to both severity and duration and is not triggered by any obligate head position changes. In addition, an associated contemporaneous decline in sensorineural hearing is required for the diagnosis of a Meniere's attack, whereas acute hearing loss should not occur with an episode of BPPV. (Thorp et al, 2003)

Protracted nausea and vomiting are also more common during an attack of Meniere's disease. Meniere's disease would be categorized as an s-EVS.

Acute peripheral vestibular dysfunction syndromes (characterized as an AVS above) such as vestibular neuritis or labyrinthitis present with sudden, unanticipated, severe vertigo with a subjective sensation of rotational (room spinning) motion. If the auditory portion of the inner ear is affected, hearing loss and tinnitus may also occur and clinically this is consistent with labyrinthitis. (Baloh, 2003) 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. (Kentala & Rauch, 2003; Kentala, 1996; Kentala et al, 1999) The vertigo is present at rest (not requiring positional change for its onset) but it may be subjectively exacerbated by positional changes. These acute peripheral vestibular syndromes may also be accompanied by severe levels of nausea, vomiting, sweating, and pallor that are also typically sustained along with the vertigo.

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. (Minor et al, 2001) SCD differs from BPPV in that vertigo is induced by pressure changes and not position changes. SCD syndrome may also present with an associated conductive hearing loss attributable to lower bone conducted thresholds for sound perception, when compared to air conducted thresholds and is diagnosed via computed tomography of the temporal bones, or alternatively, if available, vestibular evoked myogenic potential testing..(Rosowski et al, 2004; Texiheido 2008) Given that SCD would be categorized as a s-

EVS, similarly to BPPV, it should be differentiated from BPPV by its characteristic pressure related trigger (e.g. Valsalva). Similar to SCD, a perilymph fistula can produce episodes of vertigo and nystagmus triggered by pressure, thereby allowing differentiation from BPPV. PLF can occur after surgery involving the middle/mastoid or spontaneously and may be accompanied by a fluctuating hearing loss.

Post-traumatic vertigo can present with a variety of clinical manifestations including vertigo, disequilibrium, tinnitus, and headache. (Marzo et al, 2004, Hoffer 2015) These symptoms can be due to damage of the peripheral or central structures and are often complicated by overlay of depression or anxiety. Post-head trauma vestibular migraine has also been described. (Fife 2015). Although BPPV is most often idiopathic, in specific cases traumatic brain injury is associated with BPPV. (Davies et al, 1995)

NEUROLOGIC DISORDERS

One of the key issues facing clinicians attempting to diagnose the etiology for vertigo is the differentiation between peripheral causes of vertigo (those causes arising from the ear or vestibular apparatus) and central nervous system causes of vertigo. Although at times this may be difficult, several clinical features may suggest a central cause of vertigo rather than BPPV.(Labuguen, 2006; Baloh,1998) Nystagmus findings that more strongly suggest a neurologic cause for vertigo rather than a peripheral cause such as BPPV include: down- beating nystagmus on the Dix-Hallpike maneuver (particularly without the torsional component), direction changing nystagmus occurring without changes in head position (i.e. periodic alternating nystagmus), gaze holding, direction switching nystagmus (e.g., beats to the right with right gaze, and to the left with left gaze) or baseline nystagmus manifesting without provocative

maneuvers (which also could be a manifestation of vestibular neuritis apart from a neurological cause). Failure to respond to conservative management, such as CRP or vestibular rehabilitation should raise concern that the underlying diagnosis may not be BPPV. (Dunniway & Welling, 1998). Among the central causes of vertigo that should be distinguished from BPPV are vestibular migraine, brainstem and cerebellar stroke or transient ischemic attacks (TIAs), and intracranial tumors or disorders, such as multiple sclerosis.

Vestibular migraine (or migraine associated vertigo) is very common with a lifetime prevalence of 3.2% (Lempert 2009) and may account for as many as 14% of cases of vertigo. (Kentala & Rauch, 2003). Diagnostic criteria include: 1) ≥5 episodes of vestibular symptoms lasting 5 minutes to 72 hours, 2) current or history of migraine according to International Headache Society Criteria, 3) ≥ 1 migraine symptoms during at least 50% of the dizzy episodes: migrainous headache, photophobia, phonophobia, visual or other aura, 4) other causes ruled out by appropriate investigations. (Seemungal 2015). It is distinguishable from BPPV by virtue of the diagnostic components enumerated above, which are not associated with classic BPPV. Furthermore, vestibular migraine would be characterized as a s-EVS.

Brainstem and cerebellar stroke are dangerous causes of vertigo. (Kerber 2013) In one series of 240 cerebellar strokes, 10% presented similar to a peripheral vestibular process. (Lee 2006) The onset tends to be more sudden than with neuritis. Physical examination will often disclose other neurological findings relating to the posterior circulation such as dysarthria, dysmetria, dysphagia, sensory or motor loss or findings of a Horner's syndrome. (Kerber 2013)

Another important cause of vertigo is posterior circulation TIA. (Blum 2015) A study of 1141 stroke patients, of which 24% were in the posterior circulation, showed that patients with vertebrobasilar strokes had an odds ratio of 15 to have had prior posterior circulation TIA in the

90 days preceding their stroke. (Paul 2015) Half of these attacks were isolated vertigo and 8% of the patients with vertebrobasilar stroke had a TIA of isolated vertigo. Because TIAs generally last < 1 hour, most patients are asymptomatic on presentation; however, if they were to have symptoms and signs on presentation, they would be the same as those associated with vertebrobasilar stroke.

Intracranial tumors and other brain stem lesions may rarely present with a history and symptomatology similar to those of BPPV. (Dunniway & Welling, 1998). One uncommon, but important, example is central paroxysmal positional vertigo, due to structural lesions (tumors, strokes and MS plaques) generally in the cerebellar vermis or region of the fourth ventricle, which can closely mimic BPPV. (Dunniway & Welling, 1998; Soto-Varela 2013). Multiple sclerosis (MS) patients are more often female, and will nearly always have other worrisome findings such as central nystagmus patterns, internuclear ophthalmoplegia and other abnormalities that localize to the central nervous system. (Pula 2013). Importantly, in patients with known MS, BPPV was found to be a more common cause of acute dizziness than a MS flare. (Frohman 2000 Neurology)

OTHER DISORDERS

Several other non-otologic and non-neurologic disorders may present similarly to BPPV. Patients with panic or anxiety disorders may complain of symptoms of lightheadedness and dizziness. Although these symptoms are usually attributed to hyperventilation, other studies have shown high prevalence of vestibular dysfunction in these patients. (Jacob et al, 1996; Furman et al, 2006). Several medications, such as mysoline, carbamazepine, phenytoin, sedatives, antihypertensive and cardiovascular medications, may produce side effects of

dizziness and/or vertigo and should be considered in the differential diagnosis.

Cervical vertigo has been described as vertigo arising in conjunction with degenerative cervical spine disease. (Bracher et al, 2000) Cervical vertigo may produce similar symptoms to BPPV due to proprioceptive abnormalities arising from cervical spine dysfunction. (Padoan et al, 1998) Symptoms of cervical vertigo may be triggered by rotation of the head relative to the body while in an upright posture (as opposed to vertigo triggered by changes in head position relative to gravity). Orthostatic (postural) hypotension also may produce episodic dizziness or vertigo. The symptoms, however, are provoked by moving from the supine or sitting to the upright position in distinction to the provocative positional changes of BPPV.

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

Table 8: Basic differential diagnosis of BPPV

Otologic disorders	Neurologic disorders	Other entities
Meniere's disease Vestibular neuritis Labyrinthitis Superior canal dehiscence syndrome Post-traumatic vertigo Perilymphatic fistula Inner ear lesions	Vestibular migraine Posterior circulation TIA and stroke Demyelinating diseases Central nervous system lesions Vertebro-basilar insufficiency Central positional vertigo	Anxiety or panic disorder Cervicogenic vertigo Medication side-effects Postural hypotension Various medical conditions (such as toxic, infectious and metabolic conditions)

Table 9: Common causes of acute dizziness: differential diagnosis by timing and triggers category

Acute Vestibular syndrome	**Triggered Episodic Vestibular syndrome**	**Spontaneous episodic vestibular syndrome**	**Chronic vestibular syndrome**
Vestibular neuritis Labyrinthitis Posterior circulation stroke Demyelinating diseases Post-traumatic vertigo	BPPV Postural hypotension Perilymph fistula Superior canal dehiscence syndrome Vertebrobasilar insufficiency Central paroxysmal positional vertigo	Vestibular migraine Meniere's disease Posterior circulation TIA Medication side-effects Anxiety or panic disorder	Anxiety or panic disorder Medication side-effects Post-traumatic vertigo Posterior fossa mass lesions Cervicogenic vertigo (variable)

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

Triggered episodic vestibular syndrome = episodic dizziness that are triggered by specific and obligate actions, usually a change in head or body position. Episodes generally last less than 1 minute.

Spontaneous episodic vestibular syndrome = episodic dizziness that is NOT triggered and which can last minutes to hours.

Chronic vestibular syndrome = dizziness lasting weeks to months or longer

2b. MODIFYING FACTORS: Clinicians should assess patients with BPPV for factors that modify management including impaired mobility or balance, central nervous system disorders, a lack of home support, and/or increased risk for falling. *Recommendation based on observational and cross-sectional studies and a preponderance of benefit over harm.*

Action Statement Profile

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

Supporting Text

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

Although BPPV arises from dysfunction of the vestibular end organ, patients with BPPV often concurrently suffer from comorbidities, limitations and risks that may affect the diagnosis and treatment. Careful assessment of the patient with BPPV for factors that modify management is essential for improved treatment outcomes and ensuring patient safety. The majority of factors that may modify management of BPPV can be identified if the clinician questions patients for these factors and elicits a detailed history, (Rubenstein et al, 2001) including the potential social and economic impact this might have for the patient.

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.(Lawson et al, 2005) In cross-sectional surveys, patients with BPPV demonstrate higher rates of diabetes, anxiety, and history of head trauma.(Cohen et al, 2004) Other case-control studies have also found higher relative rates of migraine (34% in BPPV patients versus 10% in non-dizziness control group), history of stroke (10%, BPPV patients versus 1%, controls), diabetes (14% versus 5%), and hypertension (52% versus 22%).(von Brevern, 2007) Clinicians should assess patients with BPPV for these co-morbidities because their presence may modify management and influence treatment outcomes in BPPV.

One of the major concerns with BPPV and vertiginous conditions in general is the risk for falls and resultant injury. (Gazzola et al, 2006; Agrawal Y et al 2009; Murdin & Schilder 2015) Data from the National Health and Nutrition Examination Survey (NHANES) demonstrated a 12-fold increase in the risk for falls among older individuals who were clinically

symptomatic (reporting dizziness). (Agrawal Y et al 2009) Among community dwelling adults over the age of 65, 1 in 3 fall each year. (Tinetti et al 1988) This creates a tremendous individual and societal burden related to the health care costs of the associated injuries that occur from falling. It is estimated that the costs from falls in the United States exceed $20 billion annually. (Agrawal et al., 2013). In multiple studies concerning the etiology of falls, dizziness and vertigo were deemed the primary etiology 13% of the time, compared to existing balance and gait problems (17%), and person-environment interactions (31%).(Rubenstein, 2006) In a study by Oghalai, 9% of patients referred to a geriatric clinic for general geriatric evaluation had undiagnosed BPPV, and three fourths of those with BPPV had fallen within the 3 months prior to referral.(Oghalai et al, 2000) Thus, evaluation of patients with a diagnosis of BPPV should also include an assessment of risk for falls.(Lawson et al, 2005) In particular, elderly patients will be more statistically at risk for falls with BPPV. An initial falls risk screening might start with questions such as those suggested by the Centers for Disease Control and Prevention in 2015: 1) Have you had a fall in the past year? How many times? Were you injured? 2) Do you feel unsteady when standing or walking? 3) Do you worry about falling? A positive response to questions such as these might then prompt the clinician to conduct a more detailed falls risk assessment or refer to a clinician who can using tools such as the Get Up and Go test (Mathias et al. 1986), Tinetti Balance Assessment (Tinetti et al 1986), Berg Balance Scale (Berg et al, 1992) or others.

As noted above, comorbid conditions that occur commonly with BPPV such as a history of stroke or diabetes should also be identified when evaluating patients with BPPV. Patients with a history of stroke or a history of diabetes, particularly with peripheral neuropathy, may already have a pre-existing gait, balance or proprioceptive deficit. (Casellini & Vinik, 2007;

Richardson, 2002; Tilling et al, 2006) The additional symptoms of BPPV may increase their risk for fall and injury. Patients with visual disturbances often lack the ability to correct or compensate for a balance deficit with visual cues, and may also be at increased risk for falls. Possible associations between osteoporosis (osteopenia) and BPPV have also been reported. (Yu et al, 2014) Patients with both conditions may be at greater risk for fractures resulting from falls related to BPPV and 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. (Jonsson et al 2004)

BPPV may occur in the simultaneously with other central nervous system disorders. Patients should be questioned as to the presence of pre-existing central nervous system disorders that may modify the management of BPPV. BPPV may occur relatively commonly after trauma or traumatic brain injury.(Hoffer et al. , 2004; Motin, et al, 2005) Posttraumatic BPPV is most likely to involve the posterior semicircular canal and studies indicate that post-traumatic BPPV is significantly more likely to require repeated CRP (up to 67% of cases) for resolution as compared to non-traumatic forms (14% of cases).(Gordon et al, 2004; Aron M et al 2015) Because post-traumatic 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.(Motin et al, 2005; Ahn S-K et al 2011; Liu 2012) 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 canalith repositioning procedure.(Frohman et al, 2003; Frohman et al, 2000) These studies support that care should be taken to not miss a

diagnosis of BPPV in patients with central nervous system disorders as they may be successfully diagnosed and treated with CRP for BPPV.

Finally, in a small percentage of cases, refractory or persisting BPPV may create difficulties from a psychological and/or social-functional perspective for affected individuals.(Gamiz & Lopez-Escamez, 2004; Lopez-Escamez et al, 2005) Outcomes studies have shown that patients with BPPV exhibit a lower quality of life scores compared to the normative population in multiple subscales of the Short Form-36 quality-of-life outcomes instrument.(Lopez-Escamez et al, 2005; Lopez-Escamez et al, 2003) Patients who have pre-existing comorbid conditions may require additional home supervision in the setting of BPPV.(Whitney et al, 2005) This may include counseling about the risk of falling at home or a home safety assessment

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

Action Statement Profile

- Quality improvement opportunity: Reduce unnecessary testing and costs, reduce unnecessary radiation and radiographic contrast exposure (National Quality Strategy domains: safety, affordable quality care)
- Aggregate evidence quality: Grade C, based on observational studies for radiographic imaging.

- Level of confidence in evidence: Medium
- Benefits: Facilitate timely treatment by avoiding unnecessary testing associated with low yield and potential false positive diagnoses. Avoid radiation exposure and adverse reactions to testing.
- Risks, harms, costs: None.
- Benefits-harm assessment: Preponderance of benefit over harm.
- Value judgments: The panel placed heavy value in the accuracy of the BPPV diagnosis at the outset in that a diagnosis made by appropriate history and Dix-Hallpike is adequate to proceed with management without further testing.
- Intentional vagueness: None.
- Role of patient preferences: None.
- Exceptions: Patients who have separate indications for radiographic or vestibular testing aside from confirming a diagnosis of BPPV.
- Policy level: Recommendation against.
- Differences of opinion: None

Supporting Text

The purpose of this statement recommending against radiographic imaging is to optimize patient care, promote effective diagnosis and therapy, and reduce variations in care. The committee chose to focus on radiographic imaging in BPPV (as opposed to other diagnostic measures that can be employed) as the cost of diagnostic imaging can be significant, its use common and there is a body of literature available examining its use in BPPV from which to draw conclusions. The diagnosis of BPPV is based on the clinical history and physical

examination. Routine radiographic imaging is unnecessary in patients who already meet clinical criteria for the diagnosis of BPPV (Table 6). Further radiographic may have a role in diagnosis if the clinical presentation is felt to be atypical, if Dix-Hallpike testing elicits equivocal or unusual nystagmus findings or if additional symptoms aside from those attributable to BPPV are present, suggesting an accompanying modifying central nervous system or otologic disorder.

Radiographic imaging, most commonly central nervous system imaging using magnetic resonance or computed tomographic techniques, is commonly obtained in the evaluation of a primary symptom complaint of vertigo. However, routine imaging is not useful in the diagnosis of BPPV because there are no radiological findings characteristic of or diagnostic for BPPV.(Turski et al, 1996; Turski et al, 2006) This is likely due to fact that the pathology presumed to occur in BPPV within the semicircular canals occurs at a microscopic level which is beyond the resolution of current neuroimaging techniques.(Parnes et al, 2003) 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.(Colledge et al, 1996; Day et al, 1990). In a retrospective cohort study of 2374 patients MRI testing was not contributory to the clinical diagnosis of BPPV and neuroimaging has been shown to be of little value (Grill et al 2014).

Radiographic imaging of the central nervous system 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 neurologic signs on physical examination that are not typically associated with BPPV. Such symptoms include abnormal cranial nerve findings, visual disturbances, severe headache, among

others. It should be noted that intracranial lesions causing vertigo are rare. (Hanely et al, 2001) Potential lesions causing vertigo identifiable on central nervous system imaging include cerebro-vascular disease, demyelinating disease or an intracranial mass and these findings are most often located in the brainstem, cerebellum, thalamus or cortex.(Hanely et al, 2001) In small case series, positional vertigo and nystagmus have been associated with neuro-vascular compression of the VIIIth cranial nerve, vestibular schwannoma, Arnold Chiari malformation, and a variety of cerebellar disorders.(Brandt & Dieterich, 1994; Jacobson et al, 1995; Kumar et al, 2002)

In contrast to BPPV, such conditions are quite rare and typically present with additional neurologic symptoms in conjunction with the vertigo. Routine neuroimaging has not been recommended to discern these conditions from the more common causes of vertigo. (Gizzi et al, 1996) The costs of routine imaging in cases of BPPV are not justified given that it does not improve diagnostic accuracy in the vast majority of BPPV cases. Therefore, neuroimaging should not be routinely used in the diagnosis of BPPV.

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

Action Statement Profile

- Quality improvement opportunity: Reduce unnecessary testing and costs (National Quality Strategy domains: safety, affordable quality care)
- Aggregate evidence quality: Grade C, based on diagnostic studies with limitations in referred patient populations and observational studies for vestibular testing.

- Level of confidence in evidence: Medium
- Benefits: Facilitate timely treatment by avoiding unnecessary testing associated with low yield and potential false positive diagnoses. Avoid patient discomfort from nausea and vomiting from vestibular testing. Reduced costs from unnecessary testing.
- Risks, harms, costs: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: None
- Intentional vagueness: None
- Role of patient preferences: None
- Exceptions: Patients who have separate indications for vestibular testing aside from confirming a diagnosis of BPPV
- Policy level: Recommendation against
- Differences of opinion: None

Supporting Text

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

Vestibular function testing involves a battery of specialized tests which primarily record nystagmus in response to labyrinthine stimulation and/or voluntary eye movements. The components of the vestibular function test battery identify abnormalities in ocular motility as

well as deficits in labyrinthine response to position change, caloric stimulation, rotational movement, and static positions (sitting and supine). Caloric testing is an established, widely accepted technique which 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 (Fife et al, 2000). There are other tests which may also be considered. Postural stability testing allows for assessment of the impact of vestibular dysfunction on balance. Vestibular evoked myogenic potentials (VEMP) (ocular and cervical) provides information about the utricle and saccule, respectively. Video head impulse testing allows for assessment of the function of each semicircular canal. Some or all of these test components may be included in a vestibular test battery. These tests are useful in the evaluation of vestibular disorders that may not be evident from the history and clinical examination, and may provide information for quantification, prognostication and treatment planning (Gordon et al, 1996).

The diagnosis of BPPV is based on the clinical history and physical examination with a positive result on the Dix-Hallpike test. Fortunately, this can be accomplished by a trained clinician without specialized testing equipment and an appropriate canalith repositioning procedure (CRP) can be implemented immediately. In a retrospective chart review of 100 consecutive patients referred for vestibular assessment, Phillips et al, (2009) estimated a 9% reduction in referrals for this specialized testing could be realized if the initial provider obtained a thorough case history and completed a Dix-Hallpike test. Comprehensive vestibular testing is unnecessary in patients who already meet clinical criteria for the diagnosis of BPPV (Table 6). This does not imply that use of video-oculographic technology

with or without recording should not be used when available to help in identification and differentiation of types of BPPV.

Comprehensive vestibular testing may have a role in diagnosis if the clinical presentation is felt to be atypical, if Dix-Hallpike testing elicits equivocal or unusual nystagmus findings, if the diagnosis is unclear, or if additional symptoms aside from those attributable to BPPV are present, suggesting an accompanying modifying central nervous system or otologic disorder. It may also be beneficial when multiple concurrent peripheral vestibular disorders are suspected (Baloh et al, 1987; Kentala, 1996; Kentala et al, 2000).

In cases of BPPV where the nystagmus findings are suggestive but not clear, there may be benefit to using video-oculographic recordings of nystagmus associated with posterior canal BPPV, as the eye can be enlarged on a screen for detail, and may be replayed for further study or second opinion. In a small percentage of cases, patients with a history of positional vertigo but unclear nystagmus findings may undergo vestibular function testing. Among complex patients referred for subspecialty evaluation of BPPV, such atypical or unclear nystagmus findings may approach 13% in patients with diagnoses suspicious for BPPV (Bath et al, 2000).

BPPV is relatively frequently associated with additional vestibular pathology. Symptoms associated with an underlying, previously present, chronic vestibular dysfunction may persist following appropriate treatment for BPPV, even if the treatment is effective in resolving the specific complaint of positional vertigo. For example, in highly selected subsets of patients referred for subspecialty evaluation of BPPV, additional otopathology and/or vestibulopathy has been identified in 31% to 53% of BPPV patients (Baloh et al, 1987; Roberts et al, 2005;

Korres & Balatsouras, 2004). Abnormalities of the cervical VEMP have been reported in 25.8% to 34.8% of patients with BPPV (Hong et al, 2008; Longo et al, 2012). Lee et al. (2013) found that 50% of patients with recurrent BPPV had abnormalities on either cervical or ocular VEMP which was significantly more than the 15% of patients with non-recurrent BPPV. These VEMP abnormalities have been interpreted as suggestive of more complicated otolith dysfunction in some patients with BPPV and this negatively impacts quality of life for these patients (Hoseinabadi et al, 2015). These results have typically been measured for patients referred to specialty care centers such as audiology, neurology, or otolaryngology and may be higher than expected for patients seen by first-line, non-specialty clinicians. Vestibular disorders that have been associated with BPPV include Meniere's Disease, viral vestibular neuritis and labyrinthitis (Karlberg et al, 2000; Hughes & Proctor, 1997). Vestibular function testing may be obtained when these additional diagnoses are suspected based on signs or symptoms in addition to those of BPPV.

In patients with vestibular pathology in addition to BPPV, canalith repositioning procedures appear to be equally effective in resolving the positional nystagmus associated with BPPV, but complete symptom resolution is significantly less likely in this patient population. In one study, 86% of patients with BPPV without associated vestibular pathology reported complete resolution of symptoms after CRP versus only 37% reporting complete resolution when additional vestibular pathology was present (Pollak et al, 2002). Thus, patients with suspected associated vestibular pathology *in addition* to BPPV may be a subset who benefit from the additional information obtained from vestibular function testing. Similarly, 25% to 50% of patients with separate recurrences of BPPV are more likely to have associated vestibular pathology (Del Rio & Arriaga, 2004; Lee et al, 2013) and therefore

patients with recurrent BPPV may be candidates for vestibular function testing which could lead to additional targeted management

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

4a. REPOSITIONING PROCEDURES AS INITIAL THERAPY: Clinicians should treat, or refer to a clinician who can treat, patients with posterior canal BPPV with a canalith repositioning procedure. *Strong recommendation based on systematic reviews of randomized controlled trials and a preponderance of benefit over harm.*

Action Statement Profile

- Quality improvement opportunity: To promote effective treatment of posterior canal BPPV ((National Quality Strategy domain: promoting effective prevention/treatments)
- Aggregate evidence quality: Grade A, based on systematic reviews of randomized controlled trials.
- Level of confidence in evidence: High for otolaryngology or subspecialty settings. Lower in primary care settings where evidence is more limited.

- Benefits: Prompt resolution of symptoms with a relatively low number needed to treat ranging from 1 to 3 cases.
- Risks, harms, costs: Transient provocation of symptoms of BPPV by the procedure. Risk for falls due to imbalance after the procedure. No serious adverse events reported in RCTs.
- Benefits-harm assessment: Preponderance of benefit over harm.
- Value judgments: High value ascribed to prompt resolution of symptoms and the ease with which the CRP may be performed.
- Intentional vagueness: None
- Role of patient preferences: Moderate.
- Exceptions: Patients with physical limitations including cervical stenosis, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, retinal detachment, carotid stenosis and spinal cord injuries may not be candidates for this procedure or may need specialized examination tables for performance of the procedure.
- Policy level: Strong recommendation
- Differences of opinion: None.

Supporting Text

The purpose of this statement is to provide evidence for and promote the specific use of canalith repositioning procedures (CRP) as the initial treatment to resolve symptoms and disability secondary to posterior and lateral canal BPPV. There is high quality and compelling evidence that patients diagnosed with posterior and lateral semicircular canal BPPV should be offered expeditious treatment with CRP. These are specific and distinct from

habituation/movement exercise such as the Cawthorne-Cooksey exercises or Brandt-Daroff exercises. Treatment of BPPV with CRPs consistently eliminates the disabling vertigo and can also improve quality of life, and reduce the risk of falling.

POSTERIOR CANAL BPPV TREATMENTS

There are two distinct basic types of CRP for posterior canal BPPV (1) the canalith repositioning procedure (commonly referred to as the Epley maneuver) and (2) the liberatory maneuver (commonly referred to as the Semont maneuver). Where previous therapeutic exercises were based on habituation, these maneuvers work directly on either freeing/liberating the adhered otoconia on the cupula (cupulolithiasis) and/or by moving free floating otoconia (canalithiasis) out of the involved semicircular canal and back into the vestibule. There is significant evidence for the efficacy of both procedures for BPPV in the posterior semicircular canal and steadily advancing evidence for lateral semicircular canal.

Treatment with canalith repositioning procedure (CRP) or "Epley maneuver"

CRP was first described by Epley in 1992. (Epley, 1992). Patients are moved sequentially through a series of head position changes, designed to utilize gravity to move free-floating particles through the alignment of the posterior semicircular canal back into the vestibule, thereby relieving the pathologic stimulus which had been producing the vertigo in BPPV. Figure 3 depicts the CRP for posterior semicircular canal BPPV. There are over 20 years of evidence to support CRP for this indication although many studies were non-randomized case series (Lynn et al, 1995; Li, 1995; Lempert et al, 1997; Wolf et al, 1999; Lopez-Escamez et al, 1999; Asawavichianginda, et al, 2000; Froehling et al, 2000; Sherman & Massoud, 2001; Angeli et al, 2003; Yimtae et al, 2003; Change et al, 2004; White et al, 2005;

Woodworth et al, 2004; Teixeira & Machado, 2006). Most studies used symptom resolution as the primary outcome, but more recently conversion to a negative provocative procedure [Dix-Hallpike has been reported. A 2010 meta-analysis of the CRP (Prim-Espada et al 2010), found that patients treated with CRP had a 6 1/2 times greater chance of improvement in clinical symptoms relative to controls [OR of 6.52 (95%CI 4.17-10.20)] and similar likelihood of negative Dix-Hallpike maneuver [OR 5.19 (95% CI, 2.41-11.17)].

The 2014 updated Cochrane Collaborative Review (Hilton & Pinder, 2014), included 11 trials (745 patients) and reported that CRP is more effective compared to sham maneuvers or controls. Complete resolution of vertigo occurred significantly more often in the CRP treatment group when compared with sham or control [OR 4.42, (95% CI, 2.62 to 7.44)]. Conversion from a positive to a negative Dix-Hallpike was more likely in the CRP treatment group than the sham or controls [OR 9.62 (95% CI, 6.0 to 15.42)]. Importantly, a single CRP is over ten times more effective than a week of three times daily Brandt-Daroff (BD) Exercises [OR 12.38, 95% CI, 4.32 to 35.47)]. The randomized prospective clinical trial specifically cited in the Cochrane review (Amor-Dorado JC et al 2012) showed that by day 7 the Dix-Hallpike was negative in 80.5% of CRP versus 25% in the BD group. Differences between the groups remained statistically significant at one month. Bruintjes (Bruintjes TD, 2014), looked at CRP versus sham maneuver over long term (12 months). They found that both conversion too negative Dix-Hallpike [91% versus 46% (p=0.001) and perceived disability (p=0.003) as assessed by the Dizziness Handicap Inventory (DHI) significantly favored CRP.

The CRP is most commonly performed in the outpatient setting by a clinician after the diagnosis of posterior semicircular canal BPPV has been confirmed. (Fife et al, 2008) Patients should be informed that nausea, occasional vomiting and/or a sense of falling may arise during

the CRP.

(Uneri, 2005) Patients who previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuver may be offered antiemetic prophylaxis 30-60 minutes prior to CRP.

Treatment with the liberatory maneuver (LM) or "Semont"

The liberatory (Semont) maneuver, developed by Semont et al (Semont et al, 1988), (depicted in Figure 4) utilizes both inertial and gravity forces to move patients briskly down into a side lying position (involved side) and then through a rapid 180-degree arc to their uninvolved side. As with all CRP, the LM was designed to move the debris from the posterior semicircular canal back into the vestibule by principally breaking the canaliths free from adherence to the cupula (cupulolithiasis) and/or reposition free floating canaliths (canalithiasis). Early studies looking at the LM have demonstrated its effectiveness over sham treatments with initial success rates similar to CRP (Cohen & Kimball, 2005), and better than medication treatment (Salvinelli et al, 2003) or Brandt Daroff exercises (Soto Varel et al, 2001). Recent Cochrane Collaborative Review (Hilton & Pinder, 2014) showed no difference when comparing effectiveness of LM with CRP. Chen et al (Chen Y et al, 2012) demonstrated the short-term effectiveness of the LM in a double-blind randomized trial with conversion to negative Dix-Hallpike on the fourth day in 85% of patients treated LM versus 14% in control group (p=0.001). Some authors advocate the LM over CRP in cases of resistant BPPV, however research is lacking to demonstrate a benefit of LM in this subgroup.

Table 10 summarizes recent RCTs evaluating CRP for posterior semicircular canal BPPV. Of note, treatment effects between CRP and control patients tended to diminish over time. *The*

majority of RCTs for CRP continue to take place in specialized or tertiary *clinical setting*s, which may limit the generalizability of these results. For example, in the Munoz 2007 RCT, investigators were unable to demonstrate a significant benefit for the CRP based on symptomatic outcome in a primary care setting, although the conversion to a negative Dix-Hallpike at one week was more likely in the CRP group than those treated with sham maneuvers (Munoz et al, 2007). Since 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.

Considerable variability exists in terms of the number of times the CRP is applied for the initial treatment of BPPV, even across RCTs (Froehling et al, 2000; Lynn et al, 1995; Yimtae et al, 2003). 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. (Lynn et al, 1995) Even further variability exists among published case series for CRP. (Ruckenstein, 2001; Sekine et al, 2006; Prokopakis et al, 2005). A rapid systematic review in 2014 (Reinink,2014) concluded that multiple studies with high relevance and moderate risk of bias show a benefit of multiple treatments with the CRP in patients with BPPV who are not fully cleared. Specifically, in studies reviewed, 32%-90% of patients cleared in the first treatment session, 40-100% after second treatment session, 67%-98% after the third treatment session, 87%-100% after the fourth treatment session, and 100% in studies in which patients received 5 treatment sessions. Based on a review of the literature, it was not possible to determine the optimal number of treatments with the CRP however there is a demonstrated beneficial effect of multiple treatment sessions in patients with persistent nystagmus following the initial maneuver.

With respect to complications of treatment, CRP is associated with mild and generally self-limiting adverse effects in about 12% of those treated. (Fife et al, 2008) Some patients may experience an immediate falling sensation within 30 minutes after the maneuver and may benefit from counseling prior to the maneuver (Ear Nose Throat J. 2005 Feb;84(2):82, 84-5.). Serious complications from the CRP have not been identified in multiple randomized controlled trials. The most commonly encountered complications include nausea, vomiting, fainting and conversion to lateral canal BPPV during the course of treatment (so called "canal switch or conversion"). Canal conversion occurs in about 6-7% of those treated with CRP (Yimtae et al, 2003; Herdman & Tusa, 1996) underscoring the importance of recognizing the lateral canal variant of BPPV and need for more unique and different CRP. Another potential side effect after the CRP is postural instability that can last 24 hours with a tendency to fall backwards or forwards. 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. (Sridhar & Panda, 2005)

LATERAL (HORIZONTAL) SEMI-CIRCULAR CANAL BPPV CRP TREATMENTS

Evidence is mounting for the effectiveness of unique repositioning procedures based on semi-circular canal involvement. Although such evidence exists, the complexities associated with determining the affected side and subtype (geotropic versus apogeotropic) of the lateral canal BPPV may limit the ease of applicability of such procedures since it is paramount to determine the sidedness prior to CRP treatment in lateral canal BPPV. Nonetheless, the panel felt that information on the use of these procedures would be valuable to include as the panel anticipated increased knowledge of this type of BPPV over the next guideline update cycle.

Given that any CRP for BPPV is a direct application of anatomy of the semi-circular canal with respect to gravity, lateral semicircular canal BPPV is usually unresponsive to canalith repositioning procedures used for posterior semicircular canal BPPV, but is being found responsive to other maneuvers intended to move the displaced otoconia in the unique plane of the lateral semicircular canal. Lateral semicircular canal BPPV exists in two forms, geotropic form or apogeotropic. The best researched and most clinically responsive form is the geotropic form. CRP effectiveness specific to the lateral semicircular canal were initially described in 1996 (Lempert & Tiel-Wilck, 1996; Herman & Tusa, 1996; Fife, 1998) with the first maneuver reported as 270-360 degree "Barbeque roll" in the plane of the lateral semicircular canal (White et al, 2005; Prokopakis et al, 2005). (Figure 5) A subsequent maneuver, termed the Gufoni maneuver, was developed by Gufoni in 1998 (original publication in English by Appiani and colleagues in 2001(Appiani GC et al 2001), which involves laying sideways onto the uninvolved side and then turning the head into the terminal nose down position. (Figure 6) As with the CRP for the posterior semicircular canal, either maneuver may be performed in the outpatient setting after a diagnosis of lateral semicircular canal BPPV has been made with the supine roll test (Figure 2).

Several cohort studies and case series have reported response rates from 50% to 100% using the barbecue roll maneuver to treat lateral semicircular canal BPPV (geotropic form) (White et al, 2005; Fife et al, 2008; Nuti et al, 1998; Tirelle & Russolo, 2004; Casani et al, 2002; Prokopakis et al, 2005; Fife, 1998; Lempert & Tiel-Wilck, 1996; Appiani et al, 1997; Asprella Libonati, 2005; Chiou et al, 2005). Lateral semicircular canal BPPV may spontaneously remit more quickly than other forms of BPPV. (Moon et al, 2006; Sekine et al, 2006). There have also been several recent randomized controlled studies on both forms of lateral semicircular canal

BPPV. (Casani, 2011; Kim JS et al, 2012; Kim JS et al, 2012b; Van den brock, 2014) Casani (Casani et al 2011) demonstrated the effectiveness of these two types of CRP's in treating the geotropic form of lateral semicircular canal BPPV, comparing the results of the barbecue maneuver plus forced prolonged positioning (resting in bed for at least 12 hours with the head turned toward the unaffected ear) versus the Gufoni maneuver in a randomized prospective clinical trial with 81% success versus 93%, respectively, as determined by absence of vertigo and nystagmus on the supine roll test at follow-up examination. A study by Kim in 2012 for geotropic lateral semicircular canal BPPV with 170 consecutive patients in 10 nationwide dizziness clinics in Korea (Kim JS et al 2012), reported that after a maximum of 2 maneuvers on the initial visit day, both the barbeque roll and Gufoni maneuver were better than sham maneuvers at both one hour and one month after treatment (69%, 61%, and only 35% respectively). In the Kim study for the apogeotropic lateral semicircular canal BPPV (Kim JS et al 2012b) statistically significant results were also noted for specific CRP (modified Gufoni or therapeutic headshaking) over sham maneuvers at 73%, 62%, and only 35% for both immediate and long-term outcomes. A recent systematic review of the Gufoni maneuver for the treatment of geotropic form of lateral semicircular canal BPPV (Van den brock et al, 2014), found the Gufoni maneuver was easy to perform and more effective than sham maneuver or vestibular suppressants.

Forced prolonged positioning, as mentioned in the previously discussed Casani study, is another treatment that has been found effective for lateral semicircular canal BPPV. This involves laying for an entire night on the uninvolved side (for the geotropic form) or the involved side (for the Apogeotropic form). It may be performed either alone or concurrently with other maneuvers (Casani, 2011). The effectiveness based on case series ranged from 75-90%. (Casani

et al, 2002; Appiani et al, 1997; Chiou et al, 2005; Vannucchi et al, 1997) Other lesser-known maneuvers such as the Vannucchi-Asprella liberatory maneuver (Asprella Libonati, 2005; Appiani et al, 2005,) have also been reported as effective in uncontrolled studies.

In summary, variations of the barbecue roll maneuver or Gufoni maneuver appear moderately effective for the geotropic form of lateral semicircular canal BPPV. Other methods are not supported by randomized controlled trials. For the apogeotropic form of lateral semicircular canal BPPV, there is only a single randomized control trial (Kim, 2012) providing insufficient evidence to recommend a preferred CRP.

SELF-ADMINISTERED CRP

CRP (Epley) and the liberatory maneuver have both been modified for self-administration by patients for the treatment of BPPV (Radtke et al, 1999; Radtke et al, 2004). Self-administered CRP appears to be more effective (64% improved) than self-treatment with Brandt Daroff exercises (23% improvement) (Radtke et al, 1999) Another trial reported that self-administered CRP (Epley) resulted in 95% resolution of positional nystagmus 1 week after treatment compared to 58% for patients self-administered liberatory maneuver (Semont) maneuver ($p<0.001$). (Radtke et al, 2004). No comparison studies have been published from which to make recommendations regarding self-treatment versus clinician-administered treatment of BPPV.

Table 10: RCTs evaluating the effectiveness of Epley vs. control/placebo; or Epley vs. Brandt-Daroff or Semont for posterior canal BPPV

Reference	Time point of assessment	Improved in treatment group n/ N	Improved in control group n/ N	Endpoint	P value	Odds Ratio (95% CI)

Amor Dorado 2012	*1 week*	*33/41 (80%) Epley*	*10/40 (25%) BD*	*Negative Dix-Hallpike: Epley vs BD exercises*	*P<0.001*	*12.38 [4.34, 35.47]*
	1 month	*92.00%***	*42.50%**	*Negative Dix-Hallpike: Epley vs BD exercises*	*p<0.001*	
Bruintjes 2014	12 months	20/22 (91%)	10/22 (45%)	Negative Dix-Hallpike: Epley vs control or placebo	p<0.001	12.00 [2.24, 64.28]
	1 month	21/22 (96%)	8/22 (36%)	Negative Dix-Hallpike: Epley vs control or placebo	p<0.001	
Froehling 2000	1-2 weeks	16/24 (67%)	5/26 (19%)	Negative Dix-Hallpike:(Epley vs control or placebo)	P=0.020	3.20 [1.00, 10.20]
Liang 2010	7 days	42/43 (98%)	34/44 (77%)	cured*(E pley vs control or placebo)	p<0.05	12.35 [1.51, 101.36]

Lynn 1995	2 weeks	16/18 (89%)	4/15 (27%)	Negative Dix-Hallpike:(Epley vs control or placebo)	P<0.033	22.00 [3.41, 141.73]
Mazoor 2011	*1 week*	*22/30 (73%) Epley*	*21/30 (70%) Semont*	*Negative Dix-Hallpike: (Epley vs Semont)*	*p=0.08*	*1.18 [.38, 3.63]*
	4 weeks	*28/30 (93%) Epley*	*25/30 (83%) Semont*	*Negative Dix-Hallpike: (Epley vs Semont)*	*P=0.30*	
Munoz 2007 (Primary care setting)	Immediate	13/38 (34%)	6/41 (14%)	Negative Dix-Hallpike:(Epley vs control or placebo)	p=0.04	3.03 [1.01, 9.07]
von Brevern 2006	24 hours	28/35 (80%)	3/31 (10%)	Negative Dix-Hallpike:(Epley vs control or placebo)	P<0.001	37.33 [8.75, 159.22]
Xie 2012 (Primary care setting)	7 days	54/58 (93%)	11/45 (24%)	Cured*:(Epley vs control or placebo)	p<0.05	41.73 [12.29, 141.65]
Yimtae 2003	1 week	22/25 (88%)	13/20 (65%)	Negative Dix-Hallpike: (Epley vs control or placebo)	P=0.005	3.95 [0.87, 17.99]

	4 weeks	16/25 (64%)	7/20 (35%)	Negative Dix-Hallpike: (Epley vs control or placebo)	P=0.336	3.3 [1.0 - 11.3]

RCT: Randomized Controlled Trials

BD: Brandt Daroff

CI: Confidence

OR: Odds ratio

**Cured: outcomes reported as a composite measure of symptom resolution and Hallpike test result)*

***: Raw values not given in article*

All RCTs completed in secondary or tertiary care otolaryngology settings except where designated

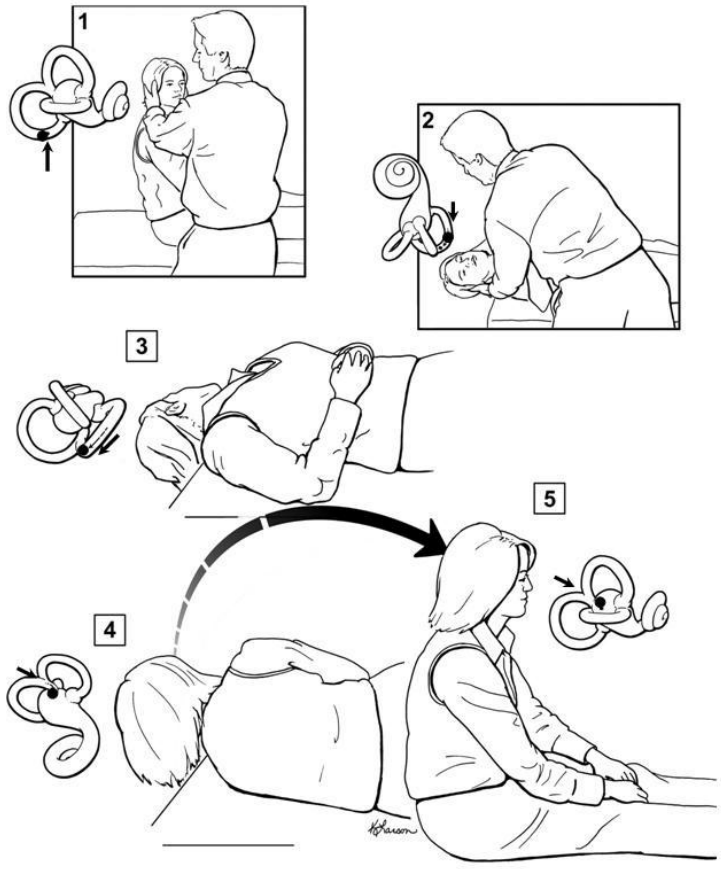

Figure 3: Depiction of the canalith repositioning maneuver (Epley maneuver) for right ear posterior semicircular canal BPPV (refer to table 11 for description).

Table 11: Stepwise sequence for the performance of the canalith repositioning maneuver (see Figure 3)

Step	Action

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

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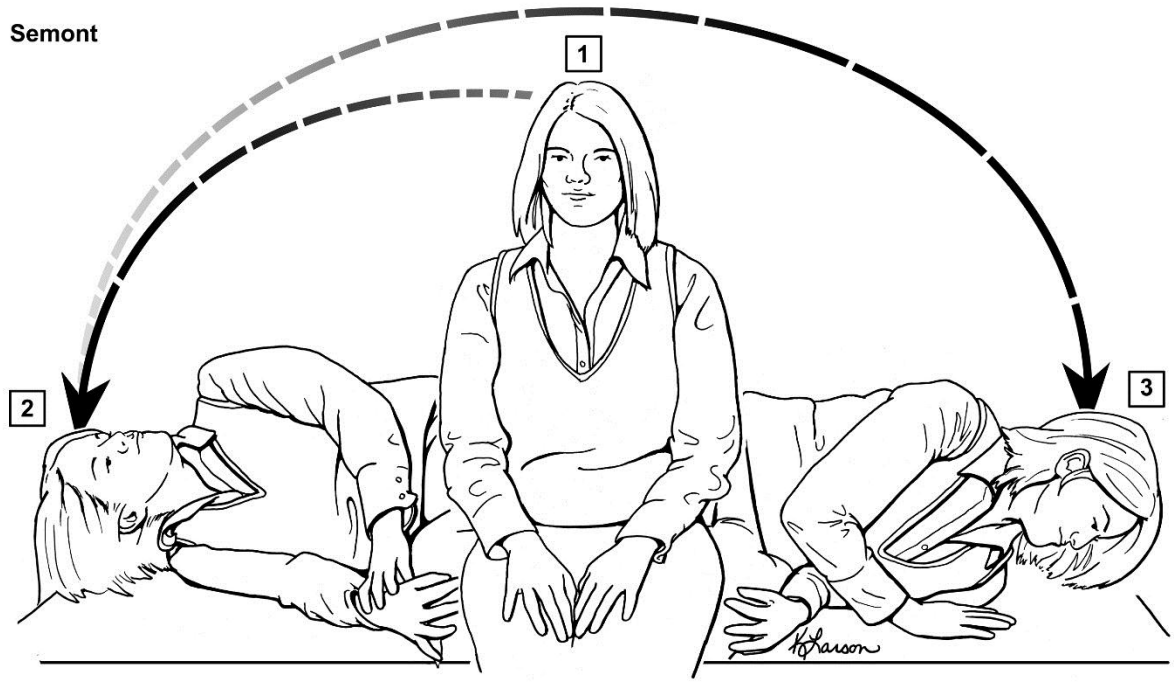

Figure 4. Semont Liberatory Maneuver for treatment of right posterior semicircular canal BPPV (see Table 12 for description).

Table 12: Stepwise description of the performance of the Semont liberatory maneuver (right ear affected)

Step	Description
1	Start with the patient sitting on a table or flat surface with head turned away from the affected side.
2	Quickly put the patient into the side-lying position, toward the affected side with the head turned up. Nystagmus will occur shortly after arriving at the side-lying position. Keep the patient in this position until at least 20 seconds after all nystagmus has ceased (some recommend up to 1-2 minutes).

3	Quickly move the patient back up and through the sitting position so that he or she is in the opposite side-lying position with head facing down (head did not turn during the position change). Keep the patient in this position for about 30 seconds (some recommend 2-10 minutes).
4	At a normal or slow rate, bring the patient back up to the sitting position.

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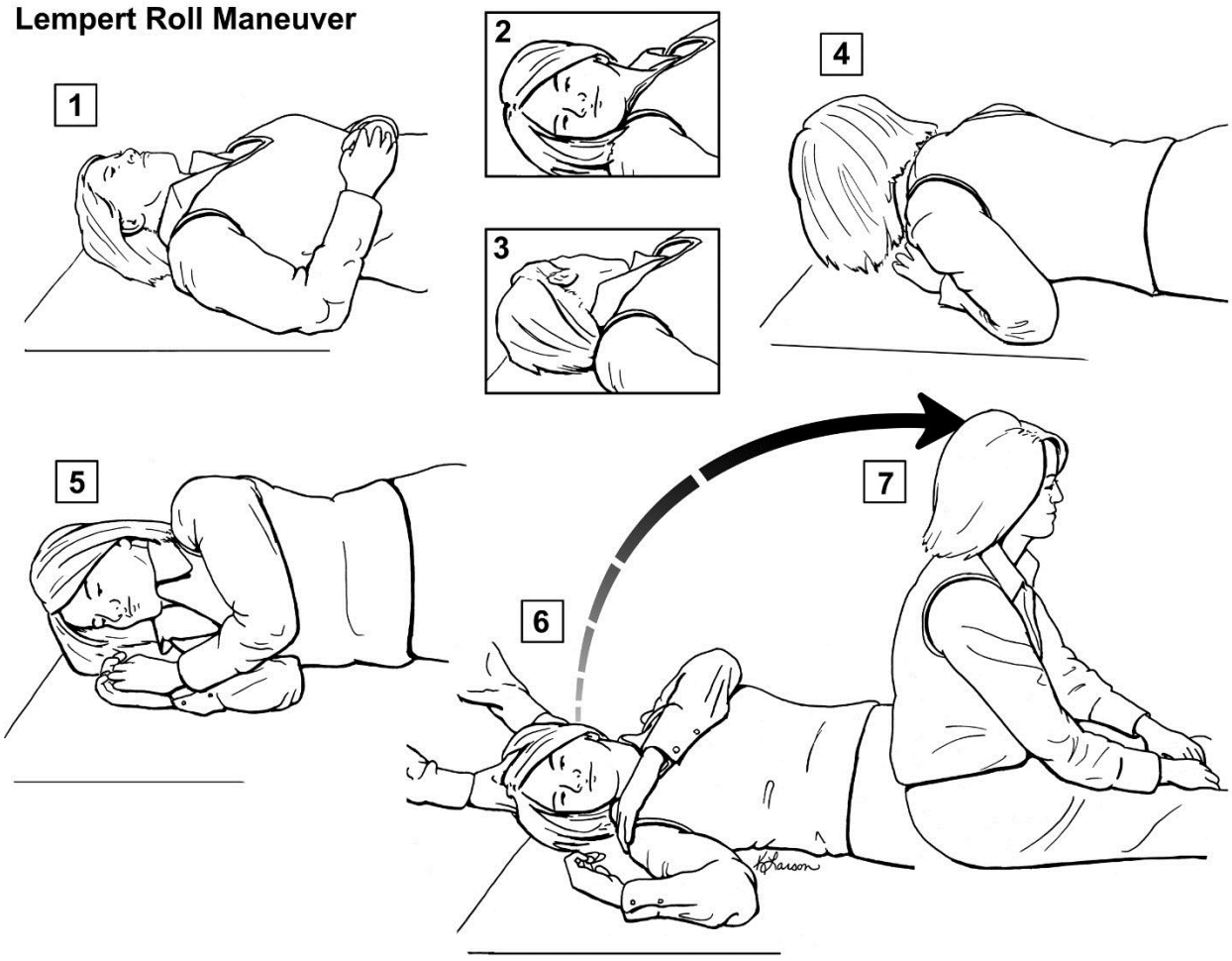


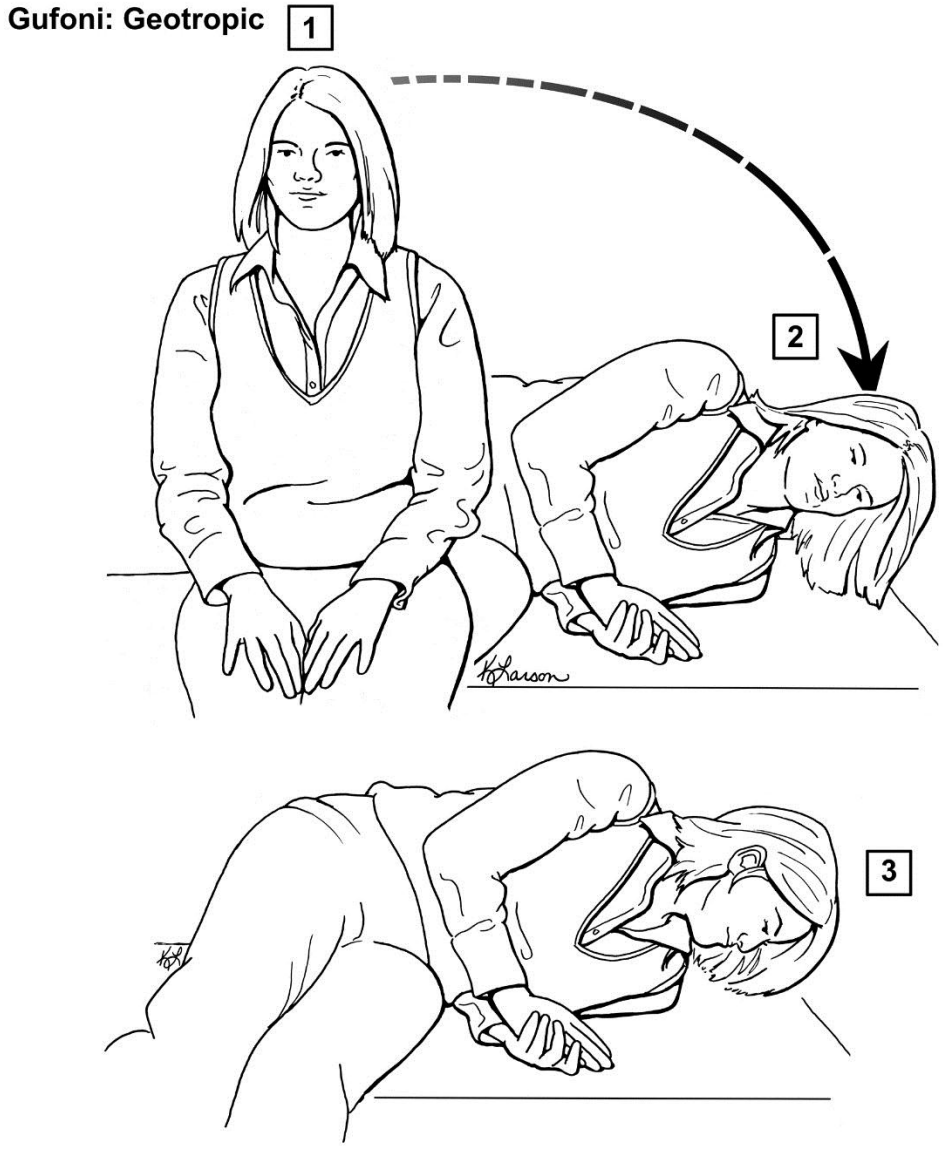
Figure 5. The Lempert 360-degree roll maneuver (sometimes referred to as the barbecue roll maneuver) for the treatment of right lateral SSC BPPV-geotropic type.

Table 13: Stepwise description of the performance of the Lempert 360° roll maneuver (barbecue roll maneuver) for the treatment of right lateral canal BPPV-geotropic type

Step	Description*
1	Starting from the supine position OR
2	Some recommend rolling to start on the involved side

3	Roll his/her head (or full body) to the unaffected side.
4	Keep rolling in the same direction until his/her head is completely nose down or prone. Some recommend ending the maneuver here and returning to sit (270-degree roll) as anatomically the debris is repositioned.
5,6,7	As originally published, however, complete the final roll (full 360) and return to sitting.

*Each position pictured is held for 15-30 seconds or until nystagmus stops.



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1419 **Figure 6. Gufoni maneuver for treatment of right-sided lateral semicircular canal BPPV-**

1420 **geotropic type (see Table 14 for description).**

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Table 14: Gufoni maneuver for treatment of right-sided lateral semicircular canal BPPV-geotropic type.

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

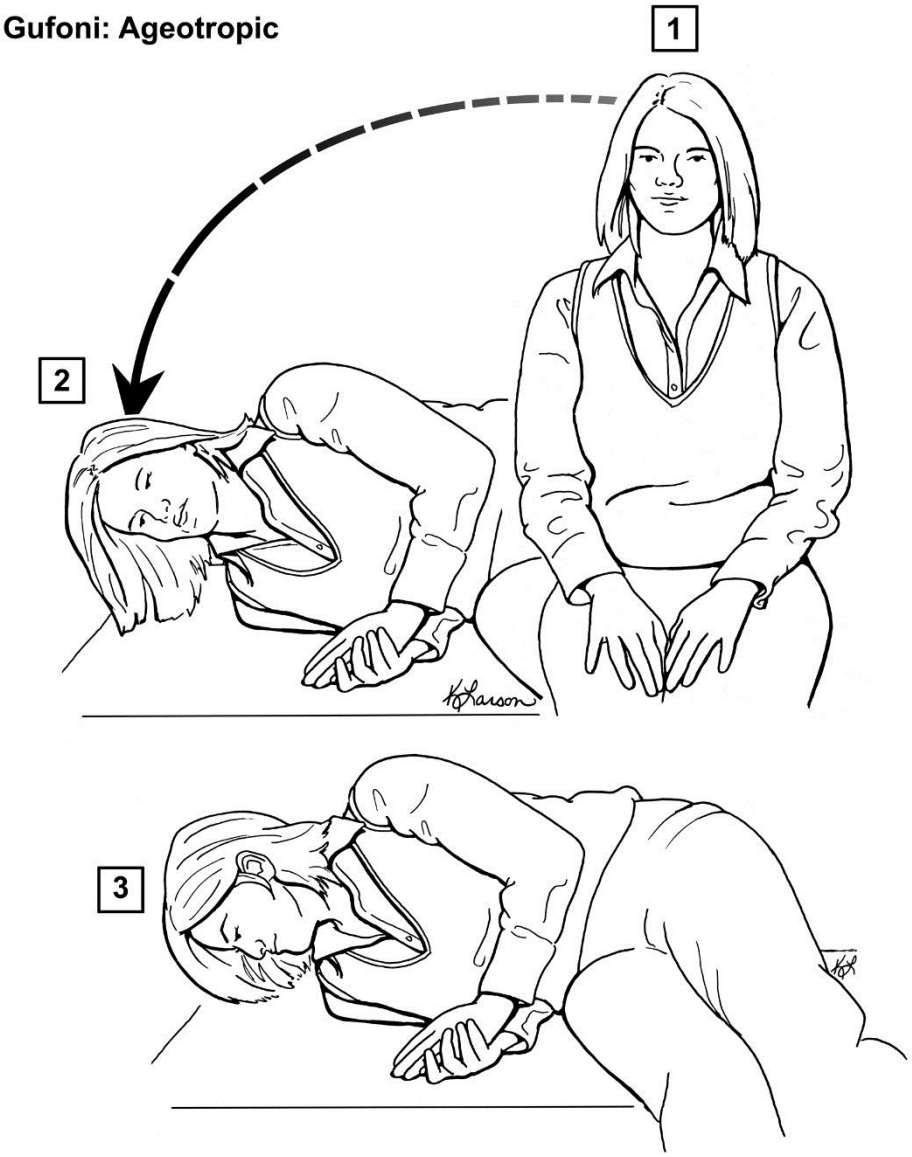


Figure 7. Gufoni maneuver for treatment of right-sided lateral semicircular canal BPPV-Apogeotropic type (See Table 15 for description).

Table 15: Gufoni maneuver for treatment of right-sided lateral semicircular canal BPPV-Apogeotropic type.

Step	Description

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

4b. POST-PROCEDURAL RESTRICTIONS: Clinicians should not recommend post- procedural postural restrictions after canalith repositioning procedure for posterior canal BPPV. *Strong recommendation against restrictions based on randomized controlled trials with minor limitations and a preponderance of benefit over harm.*

Action Statement Profile

- Quality improvement opportunity: Avoidance of unnecessary interventions, engaging patients, decreasing use of ineffective treatments (National Quality Strategy domain: coordination of care)
- Aggregate evidence quality: Grade A
- Level of confidence in evidence: High
- Benefits: Faster return to normal lifestyle, reduced anxiety, less sleep or work interruption, reduced musculoskeletal discomfort, reduced cost (e.g., of cervical collars)
- Risk, harm, cost: Potential risk for increased failure risk in a small subset of patients
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: None
- Intentional vagueness: The generic term restrictions is used but that can include sleeping upright, laying on the involved side, use of a cervical collar, or any type of restriction
- Role of patient preferences: Small
- Exclusions: None
- Policy level: Strong Recommendation Against
- Differences of opinion: Several panel members had only medium confidence in the evidence

Supporting Text

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

As canalith repositioning maneuvers grew in acceptance as a favored treatment choice for

BPPV, clinicians often advised patients regarding various post-maneuver restrictions. The rationale has been that mobile otoconial debris returned to the vestibule during treatment may move back into the semicircular canal if patients do not carefully avoid certain movements and positions. The actual restrictions vary among clinicians and even among reports describing research in this area. Common restrictions include avoidance of the following: sleeping without elevation of the head, sleeping with the treated ear in a dependent position, vertical head movement, etc. Soft cervical collars have been used to help remind patients to avoid certain head movements. Again, there is lack of clarity on exactly which positions and head movements should be avoided or for how long these limitations should be recommended. Some authors have reported that complications including neck stiffness are observed when patients are given these types of restrictions (De Stefano et al, 2011).

Comparison of studies, in particular the treatment arms for RCTs, reveals similar response rates whether or not post-treatment postural or activity restrictions are observed (i.e., Massoud & Ireland, 1996; Roberts et al, 2005; De Stefano et al, 2011; Balikci & Ozbay, 2014) There are at least nine investigations which indicate no effect. There are two investigations that report statistically significant benefit of using post-maneuver restrictions (Cohen & Kimball, 2004; Cakir et al, 2006).

Devaiah and Andreoli (2010) conducted a meta-analysis using data from six investigations with 523 patients meeting all inclusion criteria. Using this analysis, they found no effect when outcome of the patients from the two groups were compared. The authors state their findings contradict recommendations that post-maneuver head restrictions are necessary to maintain the effectiveness of BPPV maneuvers. This finding contrasts with a more recent systematic review by Hunt et al, 2012 which identified nine studies for further analysis of effects

of postural restrictions on BPPV treatment efficacy. They included data from 528 patients from the nine trials. Their results indicated benefit of using postural restrictions which provided a statistically significant improvement in outcome when the pooled data were considered. Still, the authors note a small effect size and state the statistically significant effect only highlights a small improvement in treatment efficacy. Since this report was published, there have been two additional investigations which report no significant effect of post-maneuver restrictions on BPPV treatment outcome (Toupet et al, 2012; Balikci & Ozbay, 2014).

Overall, there is insufficient evidence to recommend post-maneuver restrictions for most patients with posterior semicircular canal BPPV who are treated with a CRP. The clinician must bear in mind that these published investigations specifically excluded patients with BPPV and concomitant vertiginous disorders such as Meniere's disease, migraine, vestibular neuritis, etc. Patients with bilateral and/or multicanal involvement were also excluded. There is a small subset of patients with BPPV who will present with frequently recurring BPPV. That group was also not investigated in these reports. It is possible some of these groups may benefit from post-maneuver restrictions and this may be considered by the clinician in select cases.

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

Action Statement Profile

- Quality improvement opportunity: Decreased costs due to less intervention and incorporating patient preferences. (National Quality Strategy domains: engaging patients, affordable quality care)

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

Supporting Text

The purpose of this statement is to provide evidence and rationale for the use of "observation" as a treatment option for patients with known BPPV, including the use of waiting

times prior to canalith repositioning procedure (CRP) for acute episodes or recurrences of BPPV, especially when contra-indications to treatments or a history of adverse consequences from prior treatments for BPPV are present or as per stated preferences by the patient. Delaying referrals for specialty evaluations and/or vestibular rehabilitation are also included within the category of "observation", until such time that they are mutually agreeable with all involved.

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

In order to consider observation as an option in the management of BPPV, the natural history of BPPV needs to be understood. BPPV is a common, often self-limiting condition, but it can be either acute as a single episode, chronic and/or persisting. Although BPPV can manifest along all ages of the lifespan, it is relatively rare in children with steady and dramatic increase after age 40. Prevalence in patients over the age of 60 is 7x greater than ages 18-39. (von Brevern et al 2007). The cumulative lifetime incidence of BPPV was almost 10% by age 80 in one population-based survey from Germany, although the diagnoses were made by historic criteria alone, with no confirmation by the Dix-Hallpike maneuver. (Von Brevern, et al., 2007) The natural history of BPPV is usually one of eventual resolution of symptoms in most patients.

In several studies, the spontaneous rate of symptomatic resolution of BPPV ranges from 27-38% (Hilton 2014). Similarly, review of a recent commentary in a Cochrane Report states, the "…successful resolution of BPPV with no treatment except observation in 35% - 50% of patients indicates the rate of spontaneous recovery as part of the natural history of this condition." (Burton, 2012)

Adverse effects associated with CRP may influence decisions to avoid or delay treatment for BPPV, in favor of observation. However, adverse effects from CRP are infrequently reported. There are usually no serious adverse effects of treatment reported, although the rates of nausea during the repositioning maneuver varied from 16.7% to 32%. (Hilton & Pinder, 2014) In addition, some patients were unable to tolerate CRP because of cervical spine problems, while others complained of headache or pain in the neck after treatments. Patients with any of the relative contraindications cited elsewhere in this report, including cervical spondylosis, known cervical disk disease, and/or unstable cardiac conditions, may be candidates for observation rather than active treatment.

There was no consensus present among the guideline panel members regarding the optimal duration of observation for patients with symptomatic BPPV. However, the panel strongly favored initial treatment with CRP, particularly in subsets of patients who are either at higher risk for falls or are reporting more disabling symptoms given the high success rates detailed in section 4a. For example, there is evidence in the elderly, the most common age group to experience BPPV, that BPPV has not only a significant impact on health-related quality of life that improves with CRP (Gamiz, 2004), but that unrecognized (or untreated) BPPV has significant associated morbidity (impaired ADL/IADL capacity and fall prevalence at 78% versus 35%, p=0.026 with odds ratio of 6.2 (95% CI 1.2-31). (Oghalai, et al 2000) Additionally,

BPPV can be a triggering event for more chronic disabling dizziness in patients who are more distraught/anxious (Heinrichs et al, 2007) for which timely treatment is indicated. Widespread adoption of CRP for treatment of BPPV has yet to be seen, despite CRP's documented efficacy. Some authors are already citing the poor utilization of CRP as indicators of sub-optimal treatment quality patterns in primary care. (Kerber, 2015) However, if cases of BPPV are not as severe among those patients seen in primary care settings, compared to those patients visiting subspecialty clinics or emergency departments (spectrum bias or selection bias), then observation may become a more suitable treatment option within primary care settings. Waiting for recurrence or persistence of what would be expected to be self-limited BPPV symptoms may be one possible option to make the routine use of CRP and vestibular rehabilitation services a more rational and cost-effective policy. More research is needed to resolve the influence of a potential spectrum bias and the possible impact upon clinical trials, especially in those that include observation as a viable option.

The natural history of lateral canal BPPV is less well-defined than that of posterior canal BPPV. Some authors have commented that lateral canal BPPV may be prone to more rapid spontaneous resolution than posterior canal BPPV. (Moon et. al., 2006; Sekine et. al., 2006) One study of untreated patients with posterior canal BPPV determined a mean interval from onset of symptoms to spontaneous resolution to be about twice that of those patients with lateral canal BPPV (39 days; n=69 vs. 16 days; n=34), and the mean time between the onset of vertigo in lateral canal BPPV to spontaneous symptom resolution was about 16 days. (Imai, et. al., 2005) Although repositioning maneuvers have shown success in lateral canal BPPV, the available high quality comparative data regarding treatment versus observation, such as RCTs, are limited in this subtype of BPPV (Sekine K. et al, 2006) Thus, observation as a management strategy for

patients with lateral canal BPPV remains as a rational option. More research is needed for the interventional management of lateral canal BPPV.

In summary, observation is an option for the management of posterior canal semicircular canal BPPV and lateral semicircular canal BPPV in some patients. Observation offers the potential benefits of avoiding provocation of new symptoms and any discomfort associated with the repositioning maneuvers themselves, or with vestibular rehabilitation. There may also be cost savings from decreased rates of referral for vestibular rehabilitation or CRP. Patients who elect observation should be informed about the possibility of longer duration of symptoms when compared to patients receiving active treatment maneuvers. There is also a potential for higher recurrence rates of another episode of BPPV with the observation option. Patient education materials may be offered to those electing the observation approach to BPPV. (Furman, et al, 2013)

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

Action Statement Profile

- Quality improvement opportunity: Offer additional therapy for patients with additional *impairments, who fail initial CRP attempts,* who are not candidates for *CRP and/or* who refuse CRP. Promoting effective therapy and increased patient safety (National Quality Strategy domains: safety, promoting effective prevention/treatment)
- Aggregate evidence quality: Grade "B", based on subset analysis of a SR and limited

RCTs.

- Level of confidence in evidence: Medium
- Benefits: Offer additional therapy for patients with additional impairments; prevention of falls, improved return of natural balance function.
- Risks, harms, costs: No serious adverse events noted in published trials. Transient provocation of BPPV symptoms during rehabilitation exercises. Potential for delayed symptom resolution as compared to CRP as a sole intervention. Need for repeated visits if done with clinician supervision. Cost of therapy.
- Benefits-harm assessment: Relative balance of benefits and harm.
- Value judgments: The panel felt that vestibular rehabilitation, as defined in this guideline, may be better as an adjunctive therapy rather than a primary treatment modality. Subsets of patients with preexisting balance deficit, CNS disorders or risk for falls may derive more benefit from VR than the patient with isolated BPPV.
- Intentional vagueness: Non-specification of type of VR nor timing (initial vs adjunctive) of therapy
- Role of patient preferences: Large.
- Exceptions: Patients with physical limitations such as cervical stenosis, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries.
- Policy level: Option.
- Differences of opinion: None

Supporting Text

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

Vestibular rehabilitation (VR) has been defined as physical maneuvers or exercise regimens to treat dizziness and balance disorders. VR has long been recognized as an effective method for managing peripheral vestibular deficits (Cawthorne, 1944; Cooksey, 1946; Dix, 1979; Whitney & Sparto, 2011; Hillier & McDonnel 2011; McDonnel & Hillier 2015) by promoting habituation, adaptation, central compensation mechanisms and more recently *mechanical repositioning.* Thus, VR is not a single specific protocol but it refers to a broad designation of therapies that include CRP itself, as well as habituation exercises, exercises for gaze stabilization, balance retraining and facilitation of sensory and motor integration, gait retraining, fall prevention, relaxation training, conditioning exercises, functional and occupational skills retraining, and patient and family education.(Herdman et al, 2000; Telian & Shepard, 1996; Whitney & Rossi, 2000, Hall et al. 2016 (in press), McDonnell et al 2015) For the purpose of this key action statement, VR is being more narrowly defined as any additional therapy beyond isolated CRP for patients who either fail initial CRP attempts, are not candidates for CRP, have additional impairments, and/or who refuse CRP.

Two movement/habituation-based VR treatment protocols with respect to BPPV deserve specific mention, as they are well defined in the literature and often adopted in clinical practice. These are the Cawthorne-Cooksey exercises and the Brandt-Daroff exercise. The Cawthorne and Cooksey (Cawthorne, 1944) exercises consist of a series of eye, head and body movements in a hierarchy of increasing difficulty intended to provoke vestibular symptoms. Cawthorne-

Cooksey type exercises begin with simple head movement exercises performed in the sitting or supine position and progress to complex activities including walking on slopes and steps with eyes open and closed and sports activities requiring eye-hand coordination. These exercises theoretically fatigue the vestibular response and force the central nervous system to compensate by habituation to the stimulus (Han et al, 2011). The Brandt and Daroff exercise was developed specifically for BPPV and involves a sequence of rapid lateral head/trunk tilts repeated serially to promote loosening and ultimately dispersion of debris toward the vestibule. (Brandt & Daroff, 1980; Brandt et al, 1994) In this exercise, the patient starts in a sitting position moving quickly to the right side lying position with head rotated 45 degrees facing upward. This position is maintained until the vertigo stops. The patient then moves rapidly to a left side lying position with head rotated 45 degrees facing upward.

Several studies have compared movement/habituation-based VR to CRP in the treatment of posterior canal BPPV. In a RCT of 124 patients randomized to CRP (Epley or modified LM), Brandt-Daroff exercises, vestibular habituation exercises, or sham, both habituation routines were more effective than sham. (Cohen & Kimball, 2005; Hillier & Hollohan, 2007) However CRP was found more effective than both habituation routines. (Cohen & Kimball, 2005; Hillier & Hollohan, 2007) Soto Varela comparatively analyzed a total of 106 BPPV patients randomly assigned to receive Brandt-Daroff habituation exercises, or one of two CRP (LM or the Epley maneuver) (Soto Varela et al, 2001). At the one-week follow-up, patients treated with CRP (LM and Epley maneuvers) experienced resolution rates of 71-74% compared to only 24% with the Brandt & Daroff exercise. More recently, Toledo found in 2000 that CRP (LM specifically) was superior to Cawthorne-Cooksey exercises at both 15 days and at 3 months (Toledo, 2000). In the 2015 Cochrane review of VR for unilateral peripheral vestibular dysfunction, McDonnell &

Hillier, reported not only a significant effect of VR over control or no intervention (OR of 2.67 @ 95% CI ,1.85-3.86) but that CRP was found to be superior to movement/habituation-based VR (e.g. Cawthorne-Cooksey, Brandt-Daroff) with OR of .19 (95% CI .07-.49, odds ratio for improvement with VR versus CRP). Concluding statements from the Cochrane review support intuitive thought that the primary intervention for patients with BPPV should be maneuvers (CRP) that directly treat the condition, e.g. mechanical repositioning but that other aspects of movement/habituation-based VR may further aide and support long term functional recovery. (McDonnel & Hillier, 2015, Amor-Dorado et al 2012)

Although there is evidence that movement/habituation VR should not be considered as a substitute for CRP in the initial treatment of BPPV, there is a role for VR as adjuvant therapy in the management of selected patients with BPPV. BPPV can result in significant residual complaints of more generalized dizziness (abnormal motion sensitivities not associated with provocation of nystagmus) and definable abnormal postural control with heightened fall risk even after CRP has successfully resolved paroxysmal positional nystagmus (Di Girolamo S et al 1998; Giacomini P et al 2002). There is a statistically significant increased risk for persistent postural abnormalities in the elderly in general (Blatt PJ et al 2000) where multifactorial comorbid impairments may be present. A randomized control trial found that individuals with BPPV who were treated with CRP and additional VR exercises (balance/habituation) had significantly improved measures of overall gait stability compared to those that had received isolated CRP (Epley) for their BPPV (Chang et al. 2008). Additionally, this study documented that increased balance performance was achieved in patients only when additional movement/habituation-based VR was administered. BPPV has also been noted to trigger the development of more chronic disabling dizziness which was originally described as Phobic

Postural Vertigo (Brandt T 1996) and more recently Chronic Subjective Dizziness (CSD) or Persistent Perceptual Postural Dizziness (PPPD) for which VR appears to offer critical additional improvement. (Staab, 2012) If balance and motion tolerance doesn't improve in a timely manner in patients treated successfully with CRP, then further clinical assessment and VR is often not only indicated but necessary to complete healing and optimal resolution of disability.

Historically, VR is offered as either a home exercise-based standardized progression or more specialized and individually tailored exercise, termed customized VR. Where home exercise-based VR programs (e.g. Cawthorne-Cooksey exercises) are most often provided as a handout to a patient during initial consult with no anticipated follow-up and limited education and instruction, customized VR is usually prescribed by a therapist who individually tailors the exercises based on patient specific impairments/tolerance with the anticipation of follow-up to progress the routine. Evidence for the benefits of customized VR over home exercise-based VR have been shown in early studies (Horak FB et al 1992; Shepard & Telian 1995). Although larger randomized controlled studies are needed, customized VR has the potential to improve outcomes of BPPV. When delivered by a VR specialist, customized VR can provide secondary assessment that can gather further diagnostic information and can provide individualized modifications to the CRP (e.g. more ideal positioning with use of a trendelenburg table in patients with limited ROM). in cases of resistive forms of BPPV or complicating co-morbidities customized VR can offer an exercise prescription that is more comprehensive e.g. combinations of liberatory, habituation, more specific balance and gait retraining techniques. Examples of comorbidities that can often require customization include cervical stenosis, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord injuries. Additionally, patients with BPPV

but with other co-morbid otologic or neurologic disorders may benefit from customized VR since they may have other vestibular, mechanical or neurological deficits that require more comprehensive and customized rehabilitation.

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

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

Action Statement Profile

- Quality improvement opportunity: Decreased use of unnecessary medications with potentially harmful side effects. Reduced costs. National Quality Strategy domains: safety, promoting effective prevention/treatment, affordable quality care)
- Aggregate evidence quality: Grade C based on observational and cross-sectional studies.
- Level of confidence in evidence: Medium
- Benefits: Avoidance of adverse effects from or medication interactions with these

medications. Prevention of decreased diagnostic sensitivity from vestibular suppression during performance of the Dix-Hallpike maneuvers.

- Risks, harms, costs: None.
- Benefits-harm assessment: Preponderance of benefit over harm.
- Value judgments: To avoid harm from ineffective treatments. The panel felt that data regarding harms and side effects from non-BPPV populations with vertigo would be applicable to the BPPV patient population.
- Intentional vagueness: The panel recognized that there most likely is a very small subgroup of patients with severe symptoms who may need vestibular suppression until more definitive treatment can be offered (e.g. CRP) or immediately before and/ or after treatment with CRP.
- Role of patient preferences: Small.
- Exceptions: Severely symptomatic patients refusing other treatment options and patients requiring prophylaxis for CRP.
- Policy level: Recommendation against.
- Differences of opinion: None

Supporting Text

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

The symptoms of vertigo, due to many different underlying etiologies, may commonly be treated with medications. Clinicians may prescribe pharmacologic 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 pharmacologic therapies for vertigo may be broadly termed vestibular suppressant medications. (Hain & Uddin, 2003; Hain & Yacovino, 2005)

Several categories of vestibular suppressant medications may be used to treat a variety of vestibular disorders in general. Among these, the most often considered are the benzodiazepine and antihistamine drug classes. Benzodiazepines, such as diazepam and clonazepam, have anxiolytic, sedative, muscle relaxant, and anticonvulsant properties derived from potentiating the inhibitory effect of the gamma-amino butyric acid (GABA) system. In prolonged dizziness, these medications can reduce the subjective sensation of spinning, but also can interfere with central compensation in peripheral vestibular conditions. Antihistamines, 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-5HT3 antagonist. Lastly, anticholinergic medications such as scopolamine block acetylcholine, a widespread central nervous system transmitter, and help with motion sickness by reducing neural mismatching. (Hain & Uddin, 2003; Hain & Yacovino, 2005)

Conversely, vestibular suppressant medications have the potential for significant harm. All of these medications may produce drowsiness, cognitive deficits, and interference with driving or operating machinery.(Ancelin et al, 2006; Hebert et al, 2007; Barbone et al, 1998; Engeland et al, 2007; Jauregui et al, 2006) Medications used for vestibular suppression,

especially psychotropic medications such as benzodiazepines, are a significant independent risk factor for falls.(Hartikainen et al, 2007) The risk of falls increases in patients taking multiple medications and with the use of medications such as antidepressants.(Lawson et al, 2005; Hien le, et al, 2005) The potential for polypharmacy when adding vestibular suppressants further exposes the elderly to additional risk.(Landi et al, 2007) Educational programs to modify a practitioner's use of such medications can result in a reduction of falls.(Pit et al, 2007)

There are other potential harmful side effects of vestibular suppressants. Benzodiazepines and antihistamines interfere with central compensation for a vestibular injury. (Hanley et al, 2001; Baloh, 1998a; Baloh, 1998b) 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 (Ancelin et al, 2006) and on GI motility, urinary retention, vision and dry mouth in the elderly. (Rudolph et al, 2008)

There is no evidence in the literature to suggest that any of these vestibular suppressant medications are effective as a definitive, primary treatment for BPPV, or effective as a substitute for repositioning maneuvers.(Frohman et al, 2003; Hain & Uddin, 2003; Carlow, 1986; Cesarani et al, 2004; Fujino et al, 1994) Some studies show a resolution of BPPV over time with medications, but these studies, however, follow patients for the period of time during which spontaneous resolution would typically occur.(Sacco et al, 2014, Woodworth et al, 2004; Salvinelli et al, 2004; Itaya et al, 1997; McClure & Willet, 1980) In one double blind controlled trial e comparing diazepam, lorazepam and placebo, all groups showed a gradual decline in symptoms with no additional relief in the drug treatment arms.(McClure & Willett, 1980) A small study compared particle repositioning maneuvers to a medication alone treatment arm and found that particle repositioning maneuvers had substantially higher treatment responses (78.6%-

93.3% improvement) compared to medication alone (30.8% improvement) at two weeks follow-up.(Itaya et al, 1997) The data reinforced previous data which also indicated superiority of vestibular training for BPPV over medication use alone.(Fujino et al, 1994) Similar findings were noted when comparing canal repositioning maneuvers to betahistine where patients randomized to canal repositioning maneuvers had faster physical and mental recovery than their pharmacologic counterparts. (Maslovara et al 2012). A more recent study showed that patients who underwent the Epley maneuver alone recovered faster than those who underwent the Epley maneuver and concurrently received a labyrinthine sedative. (Sundararajan et al, 2011) Also, the addition of an antihistamine to canal repositioning maneuvers demonstrated no change in the dizziness handicap inventory score. (Kim et al 2014).

However, more recent studies have shown that there may be some pharmacological benefit in select patients. In one randomized study, the addition of a benzodiazepine to canal repositioning maneuvers significantly decreased the functional and emotional scores of the dizziness handicap inventory but did not affect the physical score compared to patients who were treated with canal repositioning maneuvers alone suggesting a role in treating psychological anxiety secondary to BPPV. (Jung et al 2011). In one trial, betahistine has been shown to be effective in reducing symptoms in patients older than 50, with hypertension, with symptom onset < 1month, with brief attacks under 1 minute when used concurrently with canal repositioning maneuvers. (Guneri 2012) A general lack of isolated benefit from vestibular suppressants and inferiority to particle repositioning maneuvers indicate that clinicians should not routinely substitute pharmacologic treatment of symptoms associated with BPPV in lieu of other more effective treatment modalities. However, when used judiciously in conjunction with canal repositioning maneuvers, pharmacologic therapy may have a role.

In summary, vestibular suppressant medications are not routinely recommended for treatment of BPPV, other than for the short term management of autonomic symptoms such as nausea or vomiting in a severely symptomatic patient. Examples of potential short term uses include patients who are severely symptomatic yet refuse therapy or patients who become severely symptomatic after a CRP. Antiemetics may also be considered for prophylaxis for patients who have previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuvers and in whom a CRP is planned. If prescribed for these very specific indications, clinicians should also provide counseling that the rates of cognitive dysfunction, falls, drug interactions, and machinery and driving accidents increase with use of vestibular suppressants.

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

Action Statement Profile

- Quality improvement opportunity: Obtain outcomes data for treatment of BPPV; ability to assess treatment effectiveness. (National Quality Strategy domains: safety, engaging patients, coordination of care, promoting effective prevention/treatment)
- Aggregate evidence quality: Grade C studies with known significant failure rates for an observation option and lower failure rates for CRP.
- Level of confidence in evidence: Medium
- Benefits: Increased accuracy of BPPV diagnosis. Identify patients initially treated

with observation who have persistent symptoms and may benefit from CRP or vestibular rehabilitation to hasten symptom resolution.

- Risks, harms, costs: Cost of reassessment.
- Benefits-harm assessment: Preponderance of benefit over harm.
- Value judgments: Panel valued ensuring the accuracy of diagnosis that may be enhanced by follow-up and capturing patients who could benefit from treatment or re-treatment to improve symptom resolution. Panel valued the potential importance of outcomes measures in the overall healthcare data environment.
- Intentional vagueness: The term reassess could represent various types of follow-up including phone calls from office staff or other methods to document outcome.
- Role of patient preferences: Small
- Exceptions: None.
- Policy level: Recommendation.
- Differences of opinion: Some panel members felt there is value in return visits to establish symptom resolution or to document objective improvement. Most other panel members felt that phone contact versus open-ended follow-up if symptoms persist or recur is sufficient.

Supporting Text

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

Importance of Patient Reassessment

Patients with BPPV, regardless of the initial treatment option, will have variable responses to therapy (Cohen and Kimball 2005). The response to therapy may depend on several factors including the accuracy of diagnosis, the duration of symptoms prior to the diagnosis, and patient compliance with the prescribed therapy (Hilton & Pinder 2004, Rupa 2004). It is important to reassess patients because those who continue to have vestibular symptoms remain at risk for falls, have decreased quality-of-life, and other consequences of unresolved BPPV. Furthermore, patients with continued vestibular symptoms should be reassessed for an accurate diagnosis and evaluated for further treatment needs.

The most effective treatment for BPPV is CRP. Recent studies have shown that the vast majority of patients are adequately treated with 1-2 CRP (79.4-92.7%) (Amor-Dorado et al. 2012; Balikci 2014; Bruintjes et al. 2014; Badawy et al. 2015). However, 12.8-15.3% of patients will require a second CRP, and 5.1% will be classified as treatment failures after 2 CRPs. (Amor-Dorado et al. 2012; Balikci 2014; Bruintjes et al. 2014; Badawy, et al. 2015).

If initial therapy fails, the patient should be reassessed for BPPV diagnosis accuracy. Symptoms of central nervous system disorders may mimic BPPV, and these conditions would not respond to BPPV treatments. In cohort studies, the rates of false positive diagnosis for BPPV subsequently found to be central nervous system lesions *after failed treatment* with CRP ranges from 1.1-3% (Dal, Ozlüoğlu et al. 2000, Rupa 2004). Thus, persistence of symptoms after initial management requires clinicians to reassess and reevaluate patients for other etiologies of vertigo. Conversely, resolution of BPPV symptoms after BPPV-targeted initial therapy, such as CRP, would corroborate and provide further evidence as to an accurate diagnosis.

Definition of Treatment Failure

In order to define a BPPV treatment failure, a failed outcome criterion as well as an appropriate time interval for reassessment needs to be defined. In clinical trials, successful BPPV treatment outcomes are traditionally defined as subjective symptom resolution and/or conversion to a negative Dix-Hallpike test (Hilton and Pinder 2004, Woodworth et al. 2004, Teixeira and Machado 2006).

Although conversion to a negative Dix-Hallpike test may have the advantage of being a more objective reassessment compared with subjective symptom resolution, it also carries the disadvantage of requiring a repeat clinical visit, which is associated with direct and indirect costs. The alternative of a symptom-based reassessment allows practitioners to use clinical judgment regarding the most appropriate follow-up modality for individual patients, including telephone communication, electronic communication, or office-based re-examination. Symptom-based assessment of treatment resolution should be detailed enough to distinguish those patients whose symptoms have decreased or minimized because of positional avoidance (who may not be treatment successes) from those with true symptom resolution (Woodworth, et al. 2004). If the patient was initially diagnosed and treated in an acute care setting (e.g. an emergency room or urgent care clinic), their primary care provider or specialist would be a suitable provider to reassess the patient.

Definition of Time Interval

There is no widely accepted time interval to assess patients for treatment failure. Therapeutic BPPV trials report follow-up assessments for treatment outcomes at 40 hours, 2 weeks, 1 month, and up to 6 months. However, the most common follow-up interval is within or at 1 month (Hilton and Pinder 2004, Woodworth, Gillespie et al. 2004, Teixeira and Machado

2006). Spontaneous symptom resolution at 1 month ranges from 20-80% (Lynn, Pool et al. 1995, Froehling, Bowen et al. 2000, Yimtae, Srirompotong et al. 2003, Sekine, Imai et al. 2006, von Brevern, Seelig et al. 2006, Munoz, Miklea et al. 2007). At the 1-month reassessment, patients should be evaluated for further interventional treatment for unresolved BPPV as well as reassessed for accurate diagnosis (Lynn, Pool et al. 1995, Froehling, Bowen et al. 2000, Yimtae, Srirompotong et al. 2003, Sekine, Imai et al. 2006, von Brevern, Seelig et al. 2006, Munoz, Miklea et al. 2007).

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

7b. EVALUATION OF TREATMENT FAILURE: Clinicians should evaluate, or refer to a clinician who can evaluate, patients with persistent symptoms for unresolved BPPV and/or underlying peripheral vestibular or central nervous system disorders.

<u>Recommendation</u> based on observational studies of diagnostic outcomes in patients with BPPV and a preponderance of benefit over harm.

Action Statement Profile

- Quality improvement opportunity: Capture missed or erroneous diagnoses; offer re-treatment to those patients with early recurrence of BPPV or failed initial CRP (National Quality Strategy domain: safety, promoting effective prevention/treatment)
- <u>Aggregate evidence quality:</u> Grade A for treatment of observation failure and Grade B for CRP failure based on RCT and SR examining treatment responses and failure rates.
- <u>Level of confidence in evidence:</u> Medium
- <u>Benefits:</u> Expedite effective treatment of patients with persistent BPPV and associated co-morbidities. Decrease the potential for missed serious medical conditions that require a different treatment algorithm.
- <u>Risks, harms, costs:</u> Costs of re-evaluation and the additional testing incurred.
- <u>Benefits-harm assessment</u>: Preponderance of benefit over harm.
- <u>Value judgments</u>: Valued comprehensive treatment of not only BPPV but associated conditions that affect balance and function. The panel also valued expeditiously treating cases of persistent BPPV following observation or vestibular rehabilitation with a CRP as more definitive therapy.
- <u>Intentional vagueness</u>: Characterization of persistent symptoms was intentionally vague to allow clinicians to determine the quality a degree of symptoms that should warrant further evaluation or re-treatment.
- <u>Role of patient preferences:</u> Small.
- <u>Exceptions</u>: None

- Policy level: Recommendation
- Differences of opinion: None

Supporting Text

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

Patients with persistent symptoms of vertigo, dizziness, or unsteadiness after initial therapy for BPPV are classified as treatment failures. Treatment failures require re-evaluation for the following reasons: 1) persistent BPPV may be present and responsive to additional maneuvers; 2) co-existing vestibular conditions may be present that can be identified and treated; and 3) serious central nervous system disorders may simulate BPPV and need to be identified. (Furman & Casss, 1999; Rupa, 2004; Furman & Cass, 1995)

PERSISTENT BPPV

Patients with BPPV who initially are treated with observation may fail to resolve spontaneously. Also, based on failure rates of vestibular rehabilitation or a single-session CRP ranging from 8-50%, a significant number of patients initially managed with vestibular rehabilitation or CRP will have persistent BPPV after initial therapy, also indicating a treatment failure.(Furman & Cass, 1999; Hilton & Pinder, 2004; Cohen & Kimball, 2005; Teixeira &

Machado, 2006; von Brevern et al, 2006; Amor-Dorado et al, 2012; Helminski et al., 2010; Bruintjes et al, 2014; Hillier & McDonnel, 2011; Hilton & Pinder, 2014; McDonnell & Hillier, 2015; Prim-Espada, 2010; van Duijn et al, 2014) As such, re-evaluation of a treatment failure is advisable and should include obtaining a history of vertigo and determining if the vertigo is provoked by positional change relative to gravity (i.e. lying down in bed, rolling over, bending down or tilting their head back), which then suggests persistent BPPV. As with the original diagnostic criteria, the Dix-Hallpike test should be repeated to confirm the diagnosis of BPPV. If the Dix-Hallpike maneuver is still positive, repeat canalith repositioning maneuvers can then be performed as a preferred treatment. The rate of successful treatment of BPPV reaches 90-98% when additional repositioning maneuvers are subsequently performed. (Brocchetti et al, 2003; Beynon et al, 2000; Reinink et al, 2014) Therefore, the CRPs are the treatment of choice for initial BPPV treatment failures deemed to be due to persistent BPPV. For treatment failures refractory to multiple CRP, surgical plugging of the involved posterior semicircular canal or singular neurectomy have a greater than 96% success rate; however, the quality of data supporting these interventions precludes the ability to make definitive recommendations for their utilization (Fife et al, 2008).

A similar approach may be adopted for the re-evaluation of persistent symptoms of vertigo after an initial diagnosis of lateral canal BPPV. The supine roll test should be repeated and if characteristic nystagmus is elicited, a CRP appropriate for lateral canal BPPV may be repeated as well. There are limited data regarding the management of treatment failures after CRP for lateral canal BPPV since this condition seems to respond more consistently to CRP and it also has a higher spontaneous resolution rate. (Tirelli & Russolo, 2004; Sekine et al, 2006; Fife, 1998; Asprella Libonati, 2005; van den Broek, 2014) Some studies indicate cure rates of

86-100% with up to four CRP treatments in lateral canal BPPV. (Casani et al, 2002; Chiou et al, 2005) Further sub-analysis suggests that the apogeotropic variant of lateral canal BPPV may be more refractory to therapy. (White et al, 2005; Casani et al, 2002; van den Broek, 2014)

A small percentage of patients initially diagnosed and treated for lateral canal BPPV may experience a "canal conversion". In these cases, initially lateral canal BPPV may transform into posterior canal BPPV in up to 6% of cases.(Nutietal, 1998; Tirelli & Russulo, 2004) Similarly, a small fraction of patients (also approximating 6%) initially presenting with posterior canal BPPV may after treatment transition to lateral canal BPPV.(Yimtae et al, 2003; Herdman & Tusa, 1996) 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.(Jackson et al, 2007) In addition, 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. (Rupa, 2004) Finally, it is possible that initial treatment was not properly directed to the involved canal thus increasing the chance of persistent symptoms. Thus, reassessment of persistent positional vertigo in BPPV should include examination for involvement of other semicircular canals other than that which was originally diagnosed.

CO-EXISTING VESTIBULAR SYSTEM DYSFUNCTION

A BPPV treatment failure may be subsequently found to be a case manifesting vertiginous symptoms that are: provoked by head and body movements in general (i.e. not primarily provoked by positional changes relative to gravity), unprovoked (i.e. spontaneous) episodes of vertigo occurring while not moving, or in fact, a constant unsteadiness. These

specific findings should be identified by clinicians as such findings suggest the presence of vestibular system dysfunction associated with, or in addition to, the initially treated BPPV.

In a study by Monobe, treatment failure of the CRP was most commonly seen in patients with BPPV secondary to head trauma or vestibular neuritis.(Monobe et al, 2001) Since vestibular neuritis and head trauma are both frequently associated with vestibular dysfunction, the cause of persistent symptoms following treatment of BPPV is likely related to widespread dysfunction within the vestibular system in this setting.(Bergenius et al, 1999) Because BPPV is more common in patients with Meniere's disease and migraine, vestibular system dysfunction associated with these disorders can lead to prolonged symptoms of BPPV, greater chance for recurrence BPPV and increased risk for falls, particularly in older persons.(Gordon et al, 2004; Roberts et al, 2005; Hughes & Proctor, 1997; Dornhoffer & Colvin, 2000; Uneri, 2004; Kayan & Hood, 1984) In addition, BPPV not associated with other otologic or neurological disease can still be associated with an underlying impaired vestibular function and affected individuals are more likely to have incomplete resolution of symptoms even if their Dix-Hallpike testing normalizes with CRP.(Pollak et al, 2002) Finally, transient vestibular dysfunction can also occur following repositioning maneuvers. Evidence suggests that balance function continues to be affected between 1-3 months post repositioning maneuvers and that some patients may need additional balance therapy (i.e., counseling, vestibular rehabilitation) in order to prevent falls and decrease their fear of falling after the vertigo from BPPV has resolved.(Blatt et al, 2000; Chang et al, 2006; Giacomini et al, 2002; Black & Nashner, 1984) Thus, re-evaluation of BPPV treatment failures should include a search for these associated conditions.

When co-existing vestibular system dysfunction is suspected, additional testing should be considered. This may include audiometric testing to screen for Meniere's disease and 8th nerve

pathology such as acoustic neuroma, vestibular function testing to detect central and peripheral vestibular dysfunction, and CNS imaging to detect CNS pathology. Such subsequent testing will need to be tailored to the clinical presentation and clinicians should exercise their clinical judgment. Vestibular rehabilitation has been shown to be an effective treatment for vestibular symptoms due to the potentially persistent vestibular dysfunction associated with BPPV and may reduce fall risk. (Angeli et al, 2003)

CNS DISORDERS MASQUERADING AS BPPV

While vertigo of central origin is frequently associated with neurological symptoms such as gait, speech, and autonomic dysfunction, it is important to recognize that, rarely, central nervous system disorders can masquerade as BPPV. (Bertholon et al, 2002) 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 explaining BPPV treatment failure was found in 3% of patients. (Dal et al, 2000)

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.(Smouha & Roussos, 1995) Patients with symptoms consistent with those of BPPV who do not show improvement or resolution after undergoing the CRP, especially after 2 or 3 attempted maneuvers, or those who describe associated auditory or neurologic symptoms should be evaluated with a thorough neurological examination, additional CNS testing and/or magnetic resonance imaging of the brain and posterior fossa to identify possible intracranial pathologic conditions.(Dunniway & Welling, 1998; Buttner et al, 1999)

8. EDUCATION: Clinicians should educate patients regarding the impact of BPPV on their safety, the potential for disease recurrence and the importance of follow-up.

Recommendation based on observational studies of diagnostic outcomes and recurrence in patients with BPPV and a preponderance of benefit over harm.

Action Statement Profile

- Quality improvement opportunity: Education allows patients to understand the implications of BPPV on quality of life and patient safety, especially falls. (National Quality Strategy domains: safety, engaging patients, promoting effective prevention/treatment)
- Aggregate evidence quality: Grade C based on observational and cross-sectional studies of recurrence and fall risk.
- Level of confidence in evidence: Medium
- Benefits: Increased awareness of fall risk potentially decreasing injuries related to falls. Increased patient awareness of BPPV recurrence which allows prompt intervention.
- Risks, harms, costs: None.
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: None.
- Intentional vagueness: None.
- Role of patient preferences: None.
- Exceptions: None.
- Policy level: Recommendation.
- Differences of opinion: None.

Supporting Text

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

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

Although BPPV generally responds well to treatment, there is a significant rate of BPPV recurrence after initial resolution or clinical cure. Most trials of BPPV maintain limited follow up, rarely beyond 3 months. In the few trials of BPPV with longer term follow-up, the rate of recurrent BPPV (that is, BPPV symptoms manifesting again after a symptom free period) is reported to be 5-13.5% at 6-months follow up.(Macias et al, 2000; Sridhar & Panda, 2005) At

one year after treatment, the rate of recurrence has been reported at a slightly higher rate of 10-18% (Prokopakis et al, 2005; Sakaida et al, 2003) The recurrence rate continues to increase over time and may be as high as 36% (Hilton et al, 2014) Patients with BPPV after trauma are likely to demonstrate an even higher recurrence rate of their BPPV.(Gordon et al, 2004)

Thus, clinicians should be aware of the recurrence risk of BPPV and should counsel patients accordingly. Counseling will likely have several benefits. These include earlier recognition by patients of recurrent BPPV, allowing earlier return for CRP or 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 that BPPV places them at greater risk for falls. (Brandt & Dieterich, 1993) This may be particularly applicable for patients with pre-existing balance disorders or vestibular deficits and a separate onset of BPPV. The propensity for falling may actually be a significant motivating factor for patients to be referred for evaluation and management of BPPV. (Lawson et al, 2005) The risk of falls and fear of falls are significant considerations in the management of the elderly who suffer from chronic dizziness. (Gazzola et al, 2006) In study of 120 elderly patients with chronic vestibular disorders, 36.7% carried the diagnosis of BPPV. Fifty-three percent of subjects had fallen at least once in the past year, and 29.2% had recurrent falls. (Gazzola et al, 2006) Other authors have confirmed a relatively high rate of BPPV and associated falling tendencies in the elderly. (Oghalai et al, 2000; Imbaud Genieys, 2007)

Practically speaking, clinicians should counsel patients and their families regarding the risk of falls associated with BPPV. This is particularly important in the elderly and frail who

may be more susceptible to serious injury as a result of falling. Such counseling could include assessment of home safety, activity restrictions and the need for home supervision until BPPV is resolved. (Rubenstein, 2006) Patients may be particularly vulnerable in the time interval between initial diagnosis of BPPV and definitive treatment when they are referred to another clinician for CRP or vestibular rehabilitation. Counseling should therefore occur at the time of initial diagnosis. The direct costs of such counseling are anticipated to be minimal and will enhance patient and public safety and avoid potential post-traumatic sequelae.

Finally, patients should be counseled regarding the importance of follow-up after the diagnosis of BPPV. Patients initially treated with observation should be counseled that if BPPV fails to resolve spontaneously, effective therapies such as the CRP may then be undertaken, particularly if an observation option is initially elected. Also, patients should be educated about atypical symptoms (subjective hearing loss, gait disturbance, non-positional vertigo, nausea, vomiting, etc.) whose occurrence or persistence after resolution of the primary symptoms of BPPV warrant further clinical evaluation. (Rupa, 2004) As noted, such symptoms, particularly when un-masked by the resolution of BPPV may indicate an underlying or concurrent vestibular or central nervous system disorder

Table 16. Patient Information: Frequently Asked Questions

Question	Answer
What is BPPV?	Benign Paroxysmal Position Vertigo (BPPV) is the most common inner ear problem and cause of vertigo or false sense of spinning. BPPV is both a specific diagnosis and a specific description of the disorder. It is "benign" because it is not life-threatening despite, at times, the alarming intensity & severity of symptoms. It is

	"paroxysmal" because it comes on suddenly and then eases in brief distinct spells. It is "positional" because it is triggered by certain head positions or movements. And finally, it is "vertigo" because of the sense of spinning motion often associated with the distinct attacks.
What causes BPPV?	BPPV is caused by displaced crystals or otoconia that have become unglued from their normal settled location in the center pouch of the inner ear and are now free floating and/or stuck on delicate sensors in the wrong or canal part of the inner ear. Where the crystals are a normal part of our inner ear and help us with balance and motion perception when they are in the "pouch", they can create intense false messages of spinning when they are moving in the canals. BPPV symptoms therefore are literally caused by these *crystals dropping* or the *sensors hanging* in these very sensitive canals. The most intense part of the BPPV symptoms are directly related to how long it takes the crystal/sensor to settle after a person moves or changes head/body position. In other words, as the crystals move/settle, your brain is being given powerful (false) messages that you are violently spinning when all you have done is perhaps laid down or rolled over in bed.
What are common symptoms and how can BPPV affect me?	Although everyone will experience BPPV uniquely, the most common symptoms are distinct triggered spells of vertigo or spinning sensations that are most often accompanied by nausea (occasionally vomiting) and/or a severe sense of disorientation in space or instability. These symptoms will last most intensely for seconds to

	minutes however can leave some people feeling a persistent sense of vaguer dizziness and instability. In some people, especially seniors, BPPV can present more as an isolated sense of instability brought on by position change e.g. sitting up, looking up, bending over and reaching. BPPV does not cause constant severe dizziness that is unaffected by position or movement. BPPV can NOT affect your hearing or produce fainting. The natural course/history of BPPV is to lessen in severity over time and so people will often report that the severity of their very first BPPV spinning episode will be the worse their symptoms will ever be.
How common is BPPV?	BPPV is very common. It is more common in older people. Many of us will experience it at some time in their life.
What caused my BPPV?	The vast majority of cases of BPPV occur for no reason however it can occasionally be associated with trauma, migraine, other inner ear problems, diabetes, osteoporosis, and prolonged time lying in bed (e.g. preferred sleep side, surgical procedures, illness)
How is BPPV diagnosed?	Normal medical imaging (e.g. scans, X-rays) or medical laboratory testing cannot show or confirm BPPV however simple bedside testing can help to confirm the diagnosis. The bedside testing requires an examiner to move a person's head into a specific position that makes the crystal move (e.g. hanging head slightly off the edge of the bed or rolling the person's head while lying in bed) which provokes a distinct characteristic eye movement that the examiner will be able to

	see and characterize to confirm the diagnosis. The most common tests are called either the Dix-Hallpike test or supine roll test.
Can BPPV be treated?	Yes. The good news is, that although medications are not indicated other than for relief of immediate distress, e.g. nausea, the vast majority of cases can be corrected with a bedside mechanical repositioning maneuvers that take only a minute or two to complete and have high success rates (around 80%) with only 1-3 treatments. These bedside mechanical repositioning maneuvers are designed to literally guide the crystals back to their original location in the inner ear. These maneuvers are often performed at the same time the bedside diagnostic testing is being performed however you can also be referred to a professional (e.g. medical provider, audiologist or therapist) who can perform these maneuvers. Being referred to a professional is particularly indicated if you have any of the following: severe disabling symptoms, you are a senior with history of past falls or fear of falling, and/or you have difficulty maneuvering (e.g. joint stiffness especially in your neck and back and/or weakness). You can also be taught and learn how to perform these maneuvers by yourself with supervision which is called "self-repositioning".
Is there any down side to BPPV repositioning	During the actual BPPV treatment there can be some momentary distress from vertigo, nausea and feelings of disorientation characteristic of your usual BPPV episodes. Following the treatment,

treatments?	some people report their symptoms start too clear almost immediately, however frequently people will report some degree of persistent motion sickness-type symptoms and mild instability that can take a few hours to resolve to more rarely a few days to gradually clear.
Can BPPV go away on its own?	There is evidence that left untreated, BPPV can go away within weeks. However, remember that while the crystal is out of place, in addition to feeling sick and sensitive to motion, your unsteadiness can make you at increased risk for falling so you need to take precautions to not fall. If you are a senior or have another underlying balance disorder, there is particularly increased risk for injury and more pronounced disability and because of this, seniors are encouraged to seek more timely and professional help to resolve symptoms.
How do I know my BPPV is effectively treated?	The strong positionally-provoked spinning vertigo that has been distinctly provoked with position changes should be dramatically if not completely resolved, with a steady resolution of even more vague complaints and mild instability over the next few days to couple of weeks.
How long will it take before I feel better?	Even after successful repositioning/treatment of BPPV some people can feel some mild residual sensitivities to movement and generalized unsteadiness that can take a few days to a few weeks to gradually resolve. It is important to follow up with your medical provider or therapist if your symptoms of dizziness/instability do not resolve in a timely manner (days to couple weeks). If you are a senior with a

	history of falls or fear of falling, there is evidence that some of the instability that was initially caused by the BPPV may need further exercises or balance therapy to completely resolve your complaints and fall risk.
Is there anything I should or shouldn't do to help my BPPV?	Yes. You need to take precautions that you don't fall as your balance will be "off" and you will feel increased sensitivity to movement until the BPPV has be successfully treated and healed. After your BPPV has been stabilized with a repositioning maneuver and your symptoms are steadily resolving, it is important to resume normal activities that you can safely tolerate as the gradual exposure to motion and movement will help to speed final residual healing.
Can BPPV come back and/or can I prevent it?	Unfortunately, BPPV is a condition that can re-occur periodically however individual risk for recurrence can vary dramatically from relatively low risk (rare experiences in lifetime) to a higher vulnerability or risk which is often caused by some secondary factor (e.g. traumatic causes, other inner ear or medical conditions, aging). Medical research has not found any way to prevent recurrences of BPPV however if it does come back or recur it is as treatable with as high success rates.

What happens if I'm still experiencing persistent symptoms following my initial treatments?	There are a number of reasons your initial treatment could have failed. 1. It is not uncommon to need more than one repositioning session to get the crystals back in their proper place, so further trials may be the only thing you need. 2. There are a number of different forms or types of BPPV which can require more specialized or customized treatment. The most common self-treatment is designed for only the most common form of BPPV. There are however a number of other treatment techniques available dependent on the different types and forms of BPPV. 3. BPPV can occasionally be in more than one canal and/or side at the same time and this would require multiple treatments to resolve. 4. If your initial attempts at repositioning have failed, particularly if you have only tried is self-repositioning, having a professional who specializes in BPPV complete the maneuver may allow for more effective repositioning. It can be difficult to achieve the most accurate positioning, where a professional may be able to achieve more optimal positioning and/or use adaptive equipment. 5. There can be some significant left-over or residual dizziness even after the BPPV crystals have been properly repositioned. This dizziness may require more time (few days to couple of weeks) or may need and/or be appropriate for a different exercise/movement routine. It is VERY important that if you are having persistent symptoms, you follow-up with your healthcare provider who may be able to refer you for further testing to confirm your diagnosis and/or

	offer further treatment options.
Resources:	Vestibular Disorders Association (VEDA): INFO@vestibular.org 5018 NE 15th Ave., Portland OR 97211, (800) 837-8428

IMPLEMENTATION CONSIDERATIONS:

The complete guideline is published as a supplement to *Otolaryngology-Head and Neck Surgery* which will facilitate reference and distribution. An executive summary will be published highlighting key recommendations from the guideline to facilitate information dissemination. Portions of the guideline will be presented at various clinical meetings including planned presentation in as a mini seminar at the annual meeting of the American Academy of Otolaryngology-Head and Neck Surgery. Existing brochures and publications by the AAO-HNSF will be updated to reflect the guideline recommendations. A visual depiction of the anticipated diagnostic and therapeutic treatment algorithm that arises from the current guideline's recommendations is presented in Figure 8. This treatment algorithm emphasizes the diagnosis and evidence-based treatment of BPPV with canalith repositioning procedures. Members of the panel will be representing the guideline at their specialty societies for possible

presentation and endorsement.

Because the guideline presents recommendations for an office-based diagnosis of BPPV based on positional maneuvers, an anticipated barrier to implementation is clinician unfamiliarity with the Dix-Hallpike maneuver and with the supine roll test. In addition to the descriptive and diagrammatic representations of the diagnostic tests, Web-based video links will be provided to the reader illustrating performance of these maneuvers as well as video representations of the expected diagnostic nystagmus findings, especially in the case of lateral canal BPPV. This may also be assisted by a laminated teaching card describing 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.

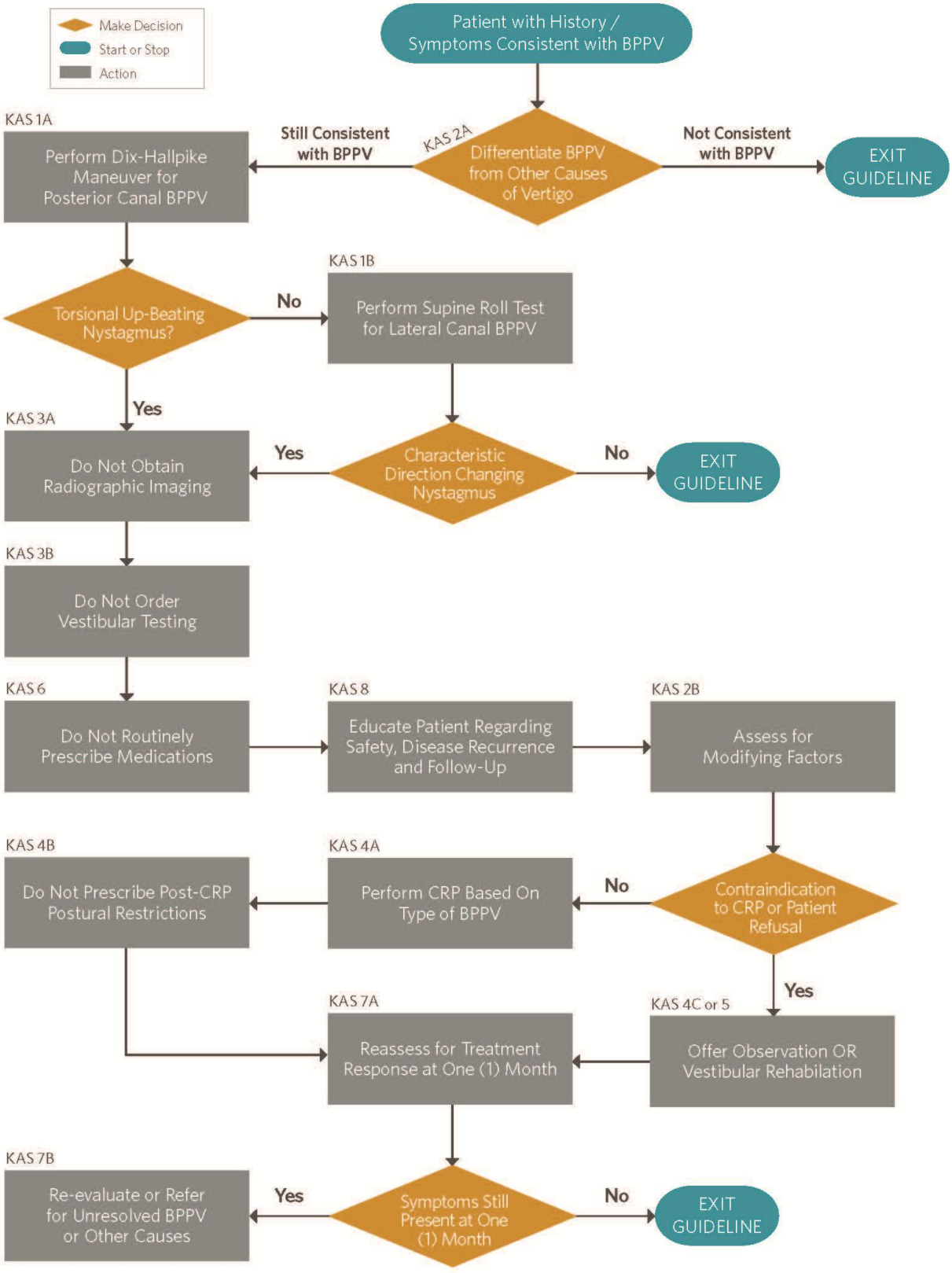
Another barrier to implementation of this guideline is potential clinician or patient preference for the ordering of diagnostic tests to evaluate vertigo. Because the differential diagnosis of vertigo may be vast and at times complex, clinicians may feel obligated to order diagnostic testing such as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

counseling on proper follow-up may offset these issues.

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

Finally, patients may seek what are perceived to be simpler solutions such as medication therapy for BPPV. Given that medication therapy has not been shown effective in the treatment of BPPV, clinicians will need to educate patients that these medications offer more harm than benefit. Additional education of patients will be required in the form of handouts or brochures that inform patients of the risks associated with symptomatic BPPV including risks for falls, recurrence of BPPV and treatment options. Algorithms for fall assessment and home safety assessment will allow clinicians to stratify patients as to these risks. (Rubenstein et al, 2001)

Figure 8: Algorithm showing the relationship of guideline key action statements


Make Decision
Start or Stop
Action
Patient with History / Symptoms Consistent with BPPV
KAS 2A
Differentiate BPPV from Other Causes of Vertigo
Still Consistent with BPPV
Not Consistent with BPPV
EXIT GUIDELINE
KAS 1A
Perform Dix-Hallpike Maneuver for Posterior Canal BPPV
Torsional Up-Beating Nystagmus?
No
KAS 1B
Perform Supine Roll Test for Lateral Canal BPPV
Yes
KAS 3A
Do Not Obtain Radiographic Imaging
Yes
Characteristic Direction Changing Nystagmus
No
EXIT GUIDELINE
KAS 3B
Do Not Order Vestibular Testing
KAS 6
Do Not Routinely Prescribe Medications
KAS 8
Educate Patient Regarding Safety, Disease Recurrence and Follow-Up
KAS 2B
Assess for Modifying Factors
KAS 4B
Do Not Prescribe Post-CRP Postural Restrictions
KAS 4A
Perform CRP Based On Type of BPPV
No
Contraindication to CRP or Patient Refusal
Yes
KAS 7A
Reassess for Treatment Response at One (1) Month
KAS 4C or 5
Offer Observation OR Vestibular Rehabilitation
KAS 7B
Re-evaluate or Refer for Unresolved BPPV or Other Causes
Yes
Symptoms Still Present at One (1) Month
No
EXIT GUIDELINE

RESEARCH NEEDS

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

1. Conduct diagnostic and cost-effectiveness studies to identify which subsets of patients, based on specific history or physical examination findings, should be submitted for additional vestibular testing and/or radiographic imaging in the setting of presumed BPPV.
2. Diagnostic and cost-effectiveness studies evaluating the utility and costs of audiometry in the diagnostic evaluation of BPPV are needed.
3. Determine whether education and application of clinical diagnostic criteria for BPPV will change physician behavior in terms of anticipated decreases in ordering of diagnostic tests.
4. Determine the optimal number of CRPs and the time interval between performance of CRP's for patients with posterior canal BPPV.
5. Cost-effectiveness studies for the potential advantages of earlier intervention based on earlier diagnosis and earlier symptom resolution with expedient CRP's for BPPV are needed. Both direct healthcare and global economic costs require assessment.
6. Extended cohort studies with longer follow-up to determine if measures such as self-performance of CRP or longitudinal vestibular rehabilitation decrease recurrence rates for BPPV or complications from BPPV such as falls.
7. Determine whether vestibular therapy after the CRP offers additional benefits over CRP alone in select patient populations.
8. Studies on the functional impact of BPPV as they relate to home safety, work safety and

absences and driving risks.

9. Epidemiological studies on the rates of falls with BPPV as an underlying cause/diagnosis.
10. Assess the impact of BPPV on quality of life for those affected using general QOL and/or dizziness specific QOL metrics.
11. Develop and validate a disease specific quality of life measure for BPPV to assess treatment outcomes.
12. Perform studies to evaluate the effect of structured versus "as needed" follow up regimens on the outcomes of patients with BPPV.
13. Clarify and standardize the terms used to describe repositioning maneuvers for BPPV of the lateral canal to enable meaningful comparison of their efficacy.
14. Perform studies to evaluate the effectiveness of mastoid vibration in the treatment of BPPV.
15. Epidemiological studies to characterize the relative risk of factors associated with the development of BPPV such as osteoporosis, dental procedures and other devices that deliver cranial vibrations (massage devices, motorized toothbrushes, etc.).
16. Identify patient and treatment related risk factors for the development of recalcitrant BPPV.
17. Perform studies to evaluate the sensitivity, specificity and predictive values of the available exam maneuvers to determine the presence and laterality of BPPV affecting the anterior semicircular canal.
18. Perform studies to characterize the accuracy of diagnostic maneuvers for posterior and lateral canal BPPV and to evaluate the treatment outcomes for patients with BPPV seen in non-specialty settings.

DISCLAIMER

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

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DISCLOSURES

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