

Clinical evaluation of posterior canal benign paroxysmal positional vertigo

Titus S. Ibekwe^{1,2}, C. Rogers²

¹Department of Surgery (ENT), College of Health Sciences University of Abuja, Nigeria, ²Communication Sciences and Disorders, University of Cape Town, South Africa

ABSTRACT

Background: Benign paroxysmal positional vertigo (BPPV) is a mechanical peripheral vestibular disorder which may involve any of the three semicircular canals but principally the posterior. In as much as the literature has described theories to explain the mechanism of BPPV and also contains scholarly works that elucidate BPPV; its management remains an enigma to most clinicians. To this end, this work was aimed at outlining an evidence-based best practice for most common form of BPPV. **Materials and Methods:** A systematic review of the literature was conducted between 1948 and June 2011 in PubMed, Embase, Ovid, and Cochrane database through the online Library of the University of Cape Town. Seventy-nine worthy articles that addressed the study were selected on consensus of the two authors. **Conclusion:** There is consensus for the use of canalith repositioning procedures as the best form of treatment for posterior canal canalolithiasis. However, successful treatment is dependent on accurate identification of the implicated canal and the form of lithiasis. Furthermore, clinicians should note that there is no place for pharmacological treatment of BPPV; unless it is to facilitate repositioning.

Key words: Benign paroxysmal positional vertigo, canalith repositioning procedures, canalolithiasis, cupulolithiasis, nystagmus, vertigo

Address for correspondence:

Dr. Titus S. Ibekwe,
University of Abuja and Association
of African Universities Scholar
(Visiting Lecturer and Researcher),
University of Cape Town,
South Africa, PMB 117,
Garki Abuja, Nigeria.
E-mail: ibekwets@yahoo.com

INTRODUCTION

Benign paroxysmal positioning vertigo (BPPV) is a peripheral vestibular disorder involving the semicircular canal usually but not exclusively the posterior semicircular canal (PSCC). Involvement of the lateral (LSCC) and superior (SSCC) semicircular canal has also been described. BPPV, with its characteristic, short-duration vertigo and accompanying nystagmus is provoked by critical provocative movements of the head relative to gravity, often lying down or turning over in bed. Indeed, if the word "bed" comes into the case history, the clinician should always consider BPPV. It should be noted that up to one third of patients will present with an atypical history, so all dizzy patients should undergo provocative positioning. In spite of the site of lesion being in the end-organ, BPPV is not associated with hearing loss or tinnitus; nor are there worrying neurologic symptoms.

Historically, Barany¹ was the first to describe BPPV in a female patient, nine decades ago with typical representation of the above features. He simply attributed his observation to the malfunction of the otolith organ. However, the details in the diagnosis were later defined by Dix and Hallpike² (Dix-Hallpike manoeuvre) in 1952. Subsequently, efforts at better understanding the pathogenesis and management of the disease were made by Schuknecht^{3,4} and others, including Epley.⁵⁻⁹ Essentially, these authors outlined the cupulolithiasis theory, which prompted the first efforts at physiotherapeutic treatment; and then the canalolithiasis theory, which also resulted in new techniques aimed at repositioning.

Despite the scholarly contributions in elucidating BPPV, clinicians still find the management challenging and sometimes indulge in dogmatic practices essentially nonbeneficial to the patients. Following the foregoing, this work is focused on outlining the scientific-based best practices for the treatment of the most commonly encountered class of BPPV.

MATERIALS AND METHODS

The search strategy

A comprehensive literature search in PubMed, Embase, Ovid and the Cochrane data base were conducted (1948–

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June 2011) using the MeSH words “Benign paroxysmal positional vertigo” “Posterior vestibular canal” “Vertigo” and “management” combined in various modes. The initial hit for MEDLINE search yielded 975 and Cochrane library 19 articles. The two authors assessed the articles independently and selected 61 that were considered relevant to the study. A cross referencing of the articles yielded an additional 18 articles. Full articles were retrieved via the online library of the University of Cape Town.

Inclusion/exclusion criteria

All original and quality review articles that addressed one or more of these: “historical background,” “Epidemiology,” “pathophysiology” and “management” of the posterior canal BPPV were included. Quality illustrations were adapted following written permission from the publishers, whereas all animal studies (except for reference purposes) and Letters to the Editor were excluded.

Epidemiology

BPPV has been described as the most commonly diagnosed vestibular disorder with a life time prevalence of about 2.4%.¹⁰⁻¹³ It is possible that this may be an under estimation due to unreported cases of sporadic BPPV that resolve spontaneously. There is no evidence-based racial bias; however, there is a strong suggestion of female preponderance^{14,15} and predilection for older age group (5th and 6th decades of life). It is rare in childhood and those aged less than 35 years except for cases of BPPV associated with head trauma.¹⁶⁻¹⁸

Classification

The classification of BPPV can be based on the anatomical location or the etiology. The most commonly involved semicircular canal is posterior; occasionally lateral canal and rarely the superior canal. Research from Korres and colleagues¹⁹ produced a typical distribution of implicated canals in 122 cases of diagnosed BPPV with 90% posterior, 8% lateral and 2% superior. LSSC BPPV may occur either with or without PSSC involvement and most often results from severe head trauma.^{17,20} However, as the type of BPPV that clinicians are most likely to encounter is in the PSSC, the focus of the discussion will be on this form. Primary or idiopathic BPPV is of unknown cause and by far the more

common (about 50%–70%) presentation. In contrast, secondary BPPV is of established causes, as illustrated in Table 1.

Pathogenesis of BPPV

In an attempt to explain the pathogenesis of BPPV, there is need for a brief description of the anatomical structure and spatial orientation of the vestibular organ [Figure 1].

The three semicircular canals are the LSSC (horizontal), PSSC, and SSCC. They lie in perpendicular (right angle) planes to one another thus representing the three dimensional spatial orientation, and sub serves for the angular acceleration. Each is encased in the bony labyrinth, and has a dilated anterior end (the ampulla) containing the patch of neuro-epithelium known as crista. The hair cell of the crista is embedded within the overlying gelatinous cupula and is displaced by the movement of the endolymph. The nonampullated ends of the posterior and superior canals fuse into a common channel (crus commune). Thus the three canals open into the vestibule via five openings specifically into the utricle which lies within the bony vestibule and communicates with the saccule through the utriculosaccular duct. Both the utricle and the saccule contain sensory elements called macula which sub serves

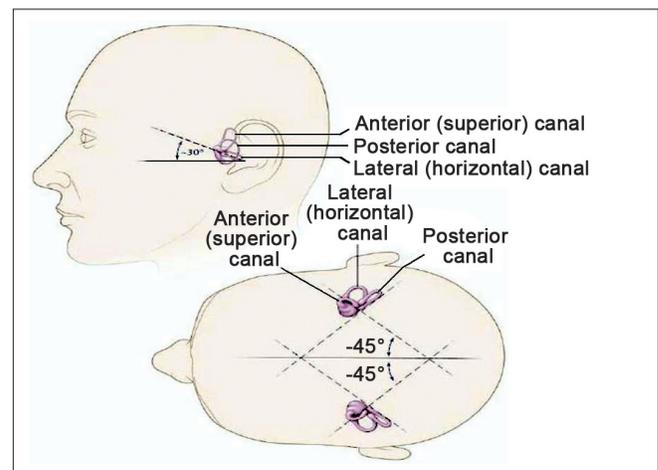


Figure 1: Schematic diagram of the vestibular system showing the anatomical and spatial orientation of the semi-circular canals relative to the head. (Adapted from Ref. 22 and reproduced with permission from Dr. L.S. Parnes parnes@uwo.ca.)

Table 1: Etio-pathological associations of secondary benign paroxysmal positioning vertigo

Causes/association	Possible mechanism
Head trauma ^{17,20,21}	Dislodge of otolith materials following impact to the head. May be unilateral or bilateral
Vestibular neuritis ²²⁻²⁵	Believed to be mainly from the inflammatory response from viral attack on the vestibular nerve/apparatus leading to degeneration and dislodgement of the otolith
Ménière's disease ²⁶⁻²⁹	The hydroptic changes associated with Ménière's disease might lead to structural damage to maculae of the utricle and saccule or cause partial obstruction to the posterior canal membranous labyrinth
Otitis media ^{30,31}	Similar to the mechanism involved in Ménière's disease however, this cascade of reaction might be triggered by the diffusion of toxins into the endolymph via the round and oval windows
Otosclerosis ^{31,32}	Degenerative changes on the utricle and saccule leading the release of otoliths
Inner ear surgery ^{32,33}	Damage to the utricle during surgery with resultant release of otoliths

for linear acceleration and deceleration. The details of the functional anatomy of the vestibular system could be obtained from standard otoneurology text books.³⁴

Two main theories, namely cupulolithiasis and canalolithiasis have been propounded over time in an attempt to explain the pathogenesis of BPPV.

(a) Cupulolithiasis theory: This marked the first attempt by Schuknecht at explaining the phenomenon of BPPV in 1969.⁴ He opined that the degeneration of the utricular otolithic membrane occurs following trauma, ischemia, infections, and other insults of the vestibular organ which results in the release of “otoconia” which gets deposited at the PSSC. This alters the specific gravity of cupula thus converting it to a gravity receptor and thereby provoking a rotatory nystagmus on rapid change of the head position.

(b) Canalolithiasis theory: Described by Hall *et al.*³⁵ in 1979 and given further prominence by Brandt and Stedding⁸ (1993), who disputed the cupulolithiasis and other theories. They proposed that otoconial debris floats freely in the PSSC rather than getting attached to the cupula. Rapid changes in the head position in respect to gravity causes downwards movement of this otoconial “clots” of debris and this induces the endolymphatic flow and cupular deflection resulting in the typical features of BPPV. The time lag in the aggregation of the otoconial floaters into ‘clots’ could account for the latent period elicited in BPPV.

Currently, it is believed that canalolithiasis theory explains most of the classical features of BPPV and coexists with cupulolithiasis. Therefore, both theories are thought to be relevant in the pathogenesis of BPPV.

Natural history, course, and diagnosis of BPPV

Case history of BPPV

Patients describe repeated, positionally-induced, sudden onset, brief episodes of vertigo, usually lasting thirty seconds or less in duration. Movements which may trigger episodes include turning over in bed; getting in and out of bed; bending over and then straightening up; extending the neck to look up, such as reaching for an item from a shelf (top-shelf vertigo); or hanging up washing.³⁶ Turning the head from side to side while standing does not provoke symptoms, as the PSSC is not stimulated by this action.³⁷ Associated symptoms may include nausea and vomiting if the BPPV is severe; and nonspecific dizziness similar to motion sickness that may last throughout the day.³⁷⁻³⁹ A fear of falling backward is an almost unique complaint associated with BPPV.⁴⁰ Patients may also complain of unsteadiness of gait and postural instability during the active and inactive phases of BPPV, and occasionally after treatment.⁴¹ The description given is often one of “walking on pillows” and may be classified as otolithic vertigo.⁴⁰

The presence of vertigo may become a psychologically disabling symptom for several reasons. First, vertigo

is difficult to identify or to see physically, leaving patients unable to locate its source, and thus prone to somatization.⁴² Second, attacks of BPPV may be frightening and potentially dangerous, for instance should an attack occur when climbing a ladder.^{40,41} Furthermore, episodes may be unpredictable, and thus fear of an attack and a tendency to anxiety and panic is well established.⁴²⁻⁴⁴ Third, research has suggested that a significant decline in the quality of life occurs with the onset of BPPV.⁴⁵⁻⁴⁸

Natural course of BPPV

It is thought that clinicians do not appreciate the natural course of BPPV, in particular its recurring nature.^{49,50} BPPV tends to present with clusters of episodes in a limited period of time (the “active” phase), followed by an interval of no attacks (the ‘inactive’ period) before recurring again.^{40,51,52} Periods of remission may be variable over many years.

Epley (1993) suggested that awaiting spontaneous resolution was not desirable as the nature of BPPV may be severely incapacitating. As BPPV may be concomitant with, and perhaps mask, other otologic or neurologic disease, which may only be revealed once the BPPV has been resolved; treatment of BPPV should occur as soon as possible.⁵³ Finally, with research suggesting that 30% of untreated cases of BPPV were still symptomatic one year after diagnosis, the likelihood of spontaneous remission is not so high as to withhold other treatment options.⁵¹

Diagnosis of BPPV: Dix Hallpike Maneuver

Diagnosis of PSSC BPPV rests on recognition of characteristic positioning nystagmus in a patient with a typical history of positional vertigo.³⁷ The Dix Hallpike Maneuver (DHM) is the test most commonly used to induce signs and symptoms of BPPV, and is regarded as the gold standard and critical for accurate diagnosis.⁵⁴ It is illustrated in Figure 2. Note that the head is positioned prior to assuming a supine position.



Figure 2: Dix-Hallpike maneuver to the left. (Reproduced with permission from Dr. A. K. Vats, <http://dizziness.webs.com/bppv.htm>.)

However, there are issues with the DHM's use as gold standard.⁹ The DHM is not 100% sensitive due to the nature of BPPV which cycles through acute and clinically silent phases. If a patient is examined during a sub-clinical period, symptoms and signs may not be provoked.^{54,55} In addition, an appraisal of the literature has revealed that estimates of specificity are lacking.⁵⁴ In spite of some concerns regarding the use of the DHM as gold standard, it is mandatory in cases with a strong history of BPPV.⁵⁴ Due to BPPV's common association with other vestibular lesions;²⁶ and the fact that up to one third of patients with BPPV may present with an atypical history;^{56,57} it is argued that careful examination including a DHM is essential in all patients who complain of dizziness or imbalance. Moreover, in complex cases with more than one subtype of dizziness, successful treatment of BPPV with a canalith repositioning maneuver (CRP), may resolve one aspect of the symptomatology.

The response to a DHM reveals the key features of canalolithiasis. As the patient reclines with the head hyper-extended over the end of the table, the clot of canaliths will move in an ampullofugal direction, which in turn displaces the cupula due to a plunger effect created by the clot moving in the narrow semi-circular canal.^{58,59} The inertia of the canaliths, in addition to the time taken for them to settle in the most dependent part of the canal, helps to explain the latent period for the onset of symptoms and signs noted after having assumed the supine position.^{20,59} Cupular deflection produces an excitatory response, which in turn leads to vertigo and torsional nystagmus in the plane of the posterior canal.²⁰ The nystagmus builds up to a crescendo and then declines quickly as the endolymph drag stops when the canalith mass reaches the limit of its descent and the cupula moves back to its neutral position.⁴⁰ Upon the patient assuming a seated position at the end of the Dix-Hallpike maneuver, reversal nystagmus occurs as the mass of particles has moved in the opposite direction, creating nystagmus in the same plane, but opposite direction.⁴⁰ The torsional nystagmus, which is the result of the neural firing in response to gravity, is difficult to suppress with fixation; so simple observation of the patient's eyes will confirm the response.⁶⁰⁻⁶² However, in mild cases Frenzel's lenses may allow better visualization of the nystagmus and infrared video goggles are suggested if they are available.^{60,63-70} Patients who have a history of BPPV with a negative DHM should be tested with the supine roll in order to exclude lateral semicircular canal BPPV.⁹ However, note that a subset of patients will have a history of BPPV but will test negative for both the DHM and supine roll.⁵⁴ These patients should be evaluated on another occasion, preferably when they are complaining of symptoms.

Differential diagnosis

Positioning testing establishes the diagnosis and also helps differentiate BPPV from positional vertigo of central origin and other forms of vertigo. Classically positive results from the DHM suggesting a peripheral site of lesion will meet

the following criteria: Linear-rotatory nystagmus with fast phase toward the undermost ear; reversal of direction of the nystagmus on sitting; duration of nystagmus and accompanying vertigo is usually less than 1 min. The nystagmus and vertigo will present in a crescendo-decrescendo pattern. The duration of the latent period prior to the onset of signs of nystagmus and symptoms of vertigo will vary depending on the type of lesion; cupulolithiasis (about 2 s) and canalolithiasis (up to 30s).

Positional vertigo is nearly always a benign condition that can be treated easily at the bedside, but in rare cases it can be a symptom of a central lesion, particularly one near the fourth ventricle. Central positional nystagmus is nearly always purely vertical (either up-beating or down-beating), and there are usually associated neurologic findings. The lesions in central PPV are often found dorsolateral to the fourth ventricle or in the dorsal vermis. This localization, together with other clinical features (associated cerebellar and oculomotor signs), generally allows for differentiation between central PPV from BPPV.³⁵

Contraindications to DHM

Humphriss and colleagues⁷¹ raised concerns about possible contraindications to the DHM. Examples of pathologies which could have serious consequences include atlantoaxial subluxation and occipito-atlantal instability; in which the application of the DHM may result in compression of the medulla and brainstem and possible vascular compromise. In patients with neck pain, the clinician should hold the patient's head securely with both hands and avoid moving the patient by the shoulders in order to avoid neck injury.⁷⁰ Prepositioning the head at a 45° angle from the sagittal plane before any downward movement is essential and reduces the risk of injury.⁷⁰ It is standard clinical practice to ask the patient for a brief history of neck or spinal problems prior to performing the DHM. Clinicians testing obese patients or those with limited mobility may require assistance in order to fully support the patient.⁹ Humphriss *et al.*, suggested that there is good evidence that the DHM is a safe procedure. The side-lying test⁷¹ is an effective alternative for patients unable to undergo the standard DHM; although it has not been subject to as much research as the DHM.

Treatment modalities of BPPV

Management options range from counseling the patient, but not giving any treatment while spontaneous resolution is awaited; physical exercises and repositioning maneuvers and in intractable cases, surgery (posterior canal occlusion procedure or singular neurectomy).⁵¹ Counseling is an important first step in treatment. Many patients, due to the frightening nature of the attacks, have marked serious illness worry and require extensive reassurance about the essentially benign nature of their disorder.³⁷ Whatever the treatment option selected, more than half of the patient population will have at least one exacerbation after their initial remission.^{37,51} The likelihood of relapse should be

discussed with the patient in order to reduce anxiety should a relapse occur.⁴⁰ Indeed, repositioning procedures should be presented to the patient as a treatment and not a cure of their BPPV.

Clinical guidelines do not support the use of medication to treat BPPV as there is no evidence that they are effective.^{9,72,73} It should be noted that vestibular suppressants may retard central processes of compensation as well as increase the risk of falling.⁹ However, medication such as promethazine HCl (Phenergan) may be indicated prior to treatment by repositioning maneuvers in patients with severe nausea or vomiting.⁷⁴

Brandt-Daroff exercises

A sequence of exercises designed by Brandt and Daroff were the first effective physical therapy exercises specifically designed for the treatment of BPPV.⁴¹ Brandt-Daroff exercises are a sequence of rapid, lateral head and trunk tilts that are repeated serially to diminish vertigo. Although the precise mechanism for relief of BPPV is not clear, it is thought that debris may be dislodged or dispersed into an area where it can no longer trigger symptoms.⁷⁵ Exercises are demonstrated to the patient who follows a program at home over a period of time (usually 10–12 days) until s/he becomes asymptomatic.⁷⁵ Self-administered Brandt-Daroff exercises are less effective than a canalith repositioning procedure in the treatment of posterior canal BPPV; have a low success rate and should not be used as initial treatment.^{46,73}

Semont's Liberatory maneuver

The Semont's Liberatory maneuver for cupulolithiasis is characterized by extremely rapid movement as the patient is moved in a cart-wheeling motion from the dependent Dix-Hallpike position to the opposite side through sitting [Figure 3].⁴⁶ The speed of the movement is necessary as both inertia and gravity act to remove any particles adhering to the cupula and to prevent the particles falling back toward the ampulla.^{46,51}

The maneuver is aggressive and potentially distressing and harmful to patients, especially those with mobility problems.^{74,76} Evidence-based reviews have suggested that the Semont is possibly effective or more effective than no treatment or Brandt-Daroff exercises treatment for BPPV. The authors noted that there is not enough evidence to establish the relative efficacy of the Semont to the canalith repositioning procedure.^{9,73}

The remaining ranges of therapeutic maneuvers designed to settle BPPV all rest on acceptance of the canalolithiasis hypothesis; that is, that the canaliths are free-floating in the posterior semi-circular canal and are thus able to move freely when the canal is orientated appropriately.^{40,51} Treatment of choice for BPPV is the use of repositioning maneuvers in order to achieve complete remission from attacks of vertigo [Figure 4]; improve quality of life and

reduce fall risk.^{9,38,77,78} While treatment strategies for BPPV were hailed as the most important breakthrough in the field of neurotology in the past twenty five years;⁵⁸ others have queried whether the progress made in the successful diagnosis and treatment of BPPV has been reflected in medical practice.⁴⁸

Epley's Canalith repositioning procedure

The CRP uses gravity to settle cases of posterior canal canalolithiasis. The procedure starts in the supine Dix-Hallpike position with the affected ear undermost. The head is slowly rolled through moderate extension to the

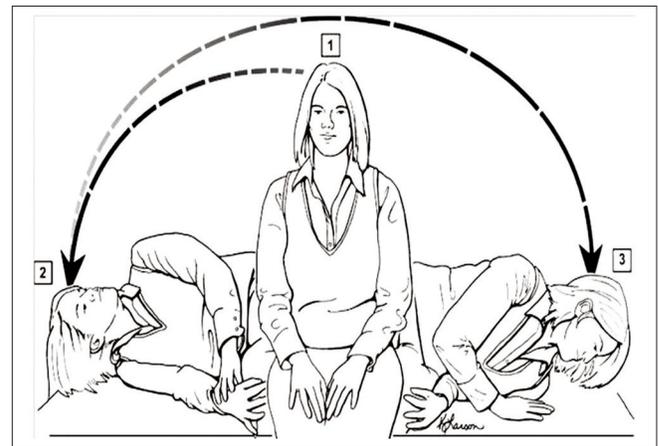


Figure 3: The Semont's manoeuvre for right-sided BPPV. Steps: (1) Patient is seated in the upright position; then the head is turned 45° toward the left side, and the patient is then rapidly moved to the side-lying position as shown in position (2) This position is held for approximately 30 s, and then the patient is rapidly moved to the opposite side-lying position without pausing in the sitting position and without changing the head position relative to the shoulder, resulting in position (3) This position is maintained for 30 s and then the patient gradually resumes the upright sitting position. (Adapted from Ref. 75 and reproduced by permission from Gwen Johnson, Wolter Kluwer Health Imprints, www.LWW.com.)

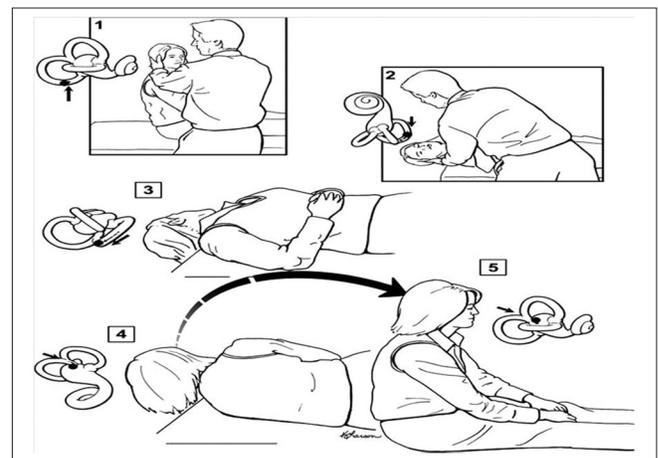


Figure 4: Epley's Canalith repositioning procedure. (Adapted from Ref. 75 and reproduced with the permission of Gwen Johnson, Wolter Kluwer Health Imprints, www.LWW.com.)

unaffected side and maintained in this position briefly, before the patient is rolled into a side-lying position with the head turned 45° down toward the floor [Figure 4]. In the final position, the patient may develop a brief episode of vertigo and nystagmus, which is indicative of debris moving inside the PSSC. With the head deviated to the unaffected side, and the head pitched down, the patient is slowly brought to the sitting position.⁷⁴ Following Epley's pioneering work; subsequent researchers adopted a single treatment approach and did not perform multiple repositioning within one session.^{79,80} Further modifications have included the abandonment of vibration, premedication and post-CRP movement restrictions.⁸¹

CRP is a safe and effective procedure that is recommended to patients with PSSC BPPV.^{9,73} However, practitioners are cautioned to ensure proper training in order to perform a DHM as well as treatment procedures for BPPV.⁸¹ Complications can result in the form of a canal conversion in between 2.5% and 6% of cases— where the BPPV moves from the PSSC to the LSSC. Thus, clinicians should be able to recognize and treat these variants.^{46,82} Another event that may occur during the CRP is “canalith jam.” This occurs as a transient burst of nystagmus that persists irrespective of head position, accompanied by intense vertigo.⁴⁰ It is assumed that the canaliths jam as they migrate from the wider ampulla to the narrower segment of the canal. Treatment is given immediately by repositioning the crus by inversion, allowing gravity to reverse dense debris out of the jam.^{83,84}

Self-treatment strategies

New self-treatment strategies based on the CRP and Semont's maneuvers have been developed.⁸⁵ Analysis has shown that self-treatment with a CRP is more effective than self-treatment with the Semont, and the patients had difficulty in performing the Semont correctly at home. In spite of suggestions that self-treatment strategies based on the CRP would be appropriate as an adjunct to office-based procedures,⁸⁶ clinicians should ensure that patients are appropriate candidates and are able to perform the exercises correctly.

CONCLUSION

Benign paroxysmal positional vertigo is a peripheral vestibular disorder first described by Barany (1921), which theoretically involves any of the three semicircular canals but principally the posterior. Several attempts have been made at explaining the mechanism/pathogenesis of the disease; however, none appears to be all inclusive. Most cases of BPPV are idiopathic and the rest associated with some secondary disorders.

Although BPPV is a common disorder and there are excellent prescribed guidelines for its diagnosis and treatment; practitioners should avoid using a “shotgun” approach and ensure treatment is tailored specifically to the type of

BPPV and the canal in which it occurs. Furthermore, the only way to assess the outcome of treatment and lack of complications is to perform a repeat DHM after treatment. Therefore, there is no rationale to treat and discharge BPPV patients without further follow-up.

Finally, evidence has shown that canalith repositioning procedure remains gold standard in the treatment of BPPV except for very occasional intractable cases that might require surgery. The best clinical practice counsels strongly against the use of medications in management of primary BPPV except for treatment of associated pathologies or symptomatic management of nausea/emesis.

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