Practical Approach to Recurrent Benign Paroxysmal Positional Vertigo

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Benign paroxysmal positional vertigo is the most common vestibular disorder and it has a significant impact on health-related quality of life. The disease is probably caused by the accumulation of lithiasis material from the otolithic membrane of the utricle. Patients experience multiple short vertigo crises lasting seconds when they go to bed or turn over. There are several clinical variants affecting posterior, horizontal or anterior canals and in some cases vestibular lithiasis can occur in two canals simultaneously. Diagnosis is by video-oculographic recording of positional nystagmus during positional tests to identify the canal affected. There are specific treatment manoeuvres for each clinical variant, which a high degree of short-term effectiveness.

Key words: Bening paroxysmal positional vertigo. Vestibular system. Vestibular training.

Abordaje práctico del vértigo posicional paroxístico benigno recurrente
El vértigo posicional paroxístico benigno es el trastorno vestibular más frecuente y tiene un impacto significativo en la calidad de vida relacionada con la salud. Probablemente, la enfermedad se origina por la acumulación de un material litiásico procedente de la membrana otolítica del utrículo. Los pacientes sufren múltiples crisis de vértigo, que duran segundos, cuando se acuestan o se dan la vuelta en la cama. Existen varias formas clínicas que pueden afectar a los conductos posterior, horizontal o anterior y que en algunos casos afectan a dos conductos simultáneamente. El diagnóstico se realiza mediante el registro videooculográfico del nistagmo posicional al realizar las pruebas posicionales para localizar el conducto afecto. Para cada variante clínica hay maniobras terapéuticas específicas con elevada efectividad a corto plazo.

Palabras clave: Vértigo posicional paroxístico benigno. Sistema vestibular. Entrenamiento vestibular.

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder, but its treatments are very effective in the short term. It was described by Robert Barany in 1921,[1] although the clinical symptoms and positional nystagmus were not defined by Charles Hallpike until 1952.[2]

The condition is characterized by episodes of vertigo of short duration associated with a characteristic nystagmus, which enables the identification of the semicircular canal that generates this nystagmus, as the axis of rotation of the eyeball is perpendicular to the plane of the affected canal.[3,4] The diagnosis and treatment through positional manoeuvres have been published in medical journals, and the disorder can be diagnosed and treated in primary care.[5,6]

The posterior semicircular canal is the most common clinical variant, although the anterior and lateral canals can also be affected.[7,8] Its aetiology is attributed to the appearance of fragments from the otolithic membrane of the utricle in the semicircular canals, which may be found free in the endolymph (canalolithiasis) or attached to the dome (glycoprotein matrix located over the neuroepithelium), as described by Harold Schuknecht in 1969, who observed deposits of basophile material in the dome of the posterior semicircular canal.[9] These fragments, called canaliths, are formed by otoconia and protein material and alter the hydrodynamic properties of the endolymph, which responds to movements of the canaliths and produces a strong stimulation of the neuroepithelium of the ampullar crests. The diagnosis of BPPV of the posterior canal is based on the observation of a typical positional nystagmus during the Dix-Hallpike test.[5,6]

The most widely used treatment of BPPV of the posterior semicircular canal is the manoeuvre of particle relocation, which John Epley described and Parnes et al[6] simplified.

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The treatment consists of a sequence of movements of the head and upper body which allow the rotation of the posterior semicircular canal in a plane that moves the canaliths from the canal to the utricle, where they are inactive.[3,10]

The efficacy of this treatment has been demonstrated in several clinical trials,[11] although the natural history of BPPV is not well known and vertigo may reappear after an initially effective treatment. Currently, it is estimated that the annual rate of recurrence is at least 15%, and there is no evidence that the manoeuvre of relocation of particles can reduce recurrences in the long term.[12]

The objective of this systematic review is to provide a formal summary of the knowledge generated from the published literature on BPPV, including some observations based on the author's clinical practice.

MATERIAL AND METHOD

The following sources of information were used to prepare this review:

– A search in PubMed using the term "BPPV" or "positional vertigo" in English or Spanish, limiting the field to the title (n=420). The search was performed on January 26, 2008

– All reviews published in English or Spanish on positional vertigo included in PubMed from the previous search (n=39)

– Manual search in the list of references of the works identified in the primary search

– Personal or e-mail contact with other specialists with experience in BPPV

– Review of the proceedings of relevant conferences

– Specialized textbooks, monographs, and a doctoral thesis

All the abstracts of the works identified were evaluated and classified according to the basic goal they sought to investigate: epidemiology (1), aetiology or physiopathology (12), clinical presentation (79), diagnosis (86), and treatment (190). Some studies, such as review articles, were not classified in any category.

EPIDEMIOLOGY OF BPPV

The only population-based study, conducted in Germany, has estimated that the lifetime prevalence in the adult population is 2.4% while the annual incidence is of 0.6%.[13] Based on this estimate, about 270 000 people a year would have BPPV in Spain, and this would be the most frequent vestibular disorder. Among the elderly population, the disorder may be even more frequent and unrecognized BPPV could reach a prevalence rate of 9%.[14]

The age of onset is 49 years and the incidence increases over time, and reaches 10% at 80 years of age.[13] The average length of each episode is 2 weeks, although this parameter suffers from wide dispersion and a third of the patients reported that the episodes lasted more than 1 month.[13]

The disease is confined to an isolated episode in 44% of cases, while in 56% the episodes of vertigo are recurrent.[13] In a longitudinal study conducted at the Hospital de Poniente, 30% of patients had a history of recurrent vertigo on the first visit, and 34% of the cases presented a recurrence of BPPV after 1 year of follow up.[14,15]

PHYSIOPATHOLOGY OF BPPV

The hypothesis that attempts to explain the physiopathology of BPPV is based on a combination of clinical and histopathological observations and physiologic experimentation.[16-21] Electron microscopy has confirmed that the canaliths are formed by otoconia.[22] Several of the features of positional nystagmus hinder the development of a unique hypothesis that can explain the variability observed in the nystagmus of different patients: latency (time elapsed from the moment the head is placed in the trigger position until the nystagmus begins), adaptation or fatigue (the velocity of the nystagmus decreases and the interval between nystagmus jerks increases), and habituation or adaptation of the response with the repetition of the test.[20,23]

Initially, it was postulated that some otoconia from the utricle moved up to the dome of the posterior semicircular canal; this situation is called cupulolithiasis.[9] Today it is considered more likely that the particles from the utricle accumulate in the long arm of the posterior semicircular canal, known as canalolithiasis.[24] The particles may agglomerate and act like a piston on the endolymph and cause a shift in the dome, which induces nystagmus when the plane of the semicircular canal is situated so that the force of gravity moves the utricular particles, as during the Dix-Hallpike position.[17] In this case, the semicircular canal becomes a linear acceleration detector, or gravity detector, for the vertical axis and activates the vestibulo-ocular reflex of the posterior canal with the contraction of the ipsilateral upper oblique and contralateral lower rectus muscles.

There is evidence supporting the involvement of the posterior canal, such as: *a*) the fact that surgical section of the inferior vestibular nerve, which innervates the posterior canal, or the blocking of the canal resolve the condition[25]; and *b*) the identification of free particles in the posterior canal in the surgery for its occlusion.[26,27]

Vector analysis of the rotation axis of positional nystagmus[28,29] has shown that other canals, such as the lateral and anterior, may be affected as well as the simultaneous participation of several canals. In addition, studies based on rotation tests outside the vertical axis show utricular dysfunction.[30]

On the other hand, several studies have demonstrated vestibular paresis in some individuals through heat test evidence,[10,16] as well as paresis of the vertical canals.[31,32]

CLINICAL DIAGNOSIS OF BPPV

Patients describe BPPV as an attack of spinning vertigo with a duration of various seconds, precipitated by certain

movements or changes in the position of the head. The movements most commonly reported are turning in bed, the extension of the neck or the inclination of the head forward. Patients can identify the affected side or a head movement that triggers the symptoms (for example, turning in bed to the right, but not to the left, precipitates the crisis, suggesting that the right ear is involved).

The crisis of vertigo lasts 10-30 s, although some patients perceive it for several minutes. The reason for this variation is probably due to over-evaluation of the duration of the crisis by the patients. Sometimes they present several crises separated from each other, which they report as a single crisis; at other times patients refer to the nausea and dizziness or imbalance that persist for several hours after a crisis, thus complicating the diagnosis. Although 80% speak of a sense of rotation, up to 47% present a feeling of floating.[33] The episodes of vertigo occur for several weeks (23%) or during the course of a day (52%).[33]

Some patients report headaches, nausea, dizziness and, in many cases, sensitivity to movements of the head in all directions. In addition, many patients present anxiety and may develop avoidance behaviours to the movement triggering the vertigo crisis.

Figure 1 shows the diagnostic algorithm for single or recurrent vertigo used at our centre. All patients with a positive Dix-Hallpike (DH) test are evaluated within 7 days to confirm the positional diagnosis through video-oculography. The standardized tests are spontaneous nystagmus, cephalic agitation nystagmus, nystagmus in decubitus, left rotation, right rotation, left DH, right DH, hyperextension in midline, and bithermal caloric test.

Many patients are taking benzodiazepines or sulpiride and report instability, making it necessary to withdraw these drugs before carrying out a proper assessment of any nystagmus, not just positional nystagmus.

BPPV is not a diagnosis that excludes other causes of balance disorders and it can be seen in patients with a history of vestibular neuritis, migraine or Ménière's disease. In our series, 24% of individuals with BPPV presented caloric vestibular hypofunction,[16] which is why a caloric test is systematically carried out in all individuals with BPPV.

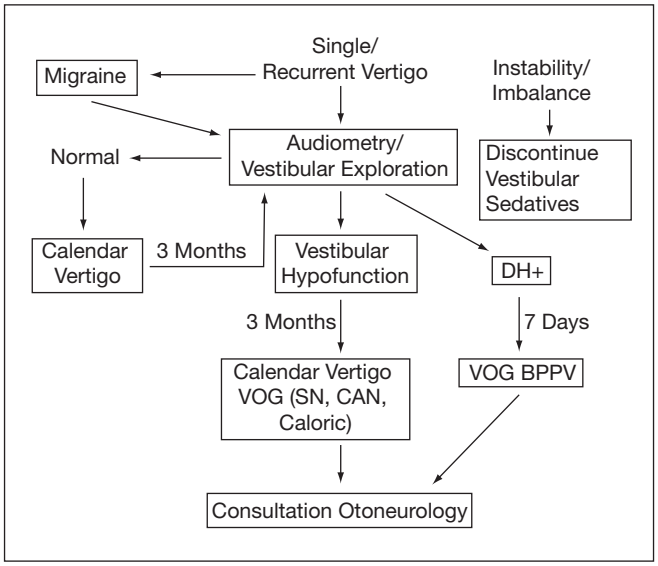

Figure 1. Diagnostic flowchart for patients with balance disorder. CAN indicates cephalic agitation nystagmus; DH+, Dix-Hallpike test; SN, spontaneous nystagmus; VOG, video-oculography.

POSTERIOR SEMICIRCULAR CANAL

The DH manoeuvre or test is used for diagnosis of posterior canal BPPV.[2] Figure 1 represents a schematic sequence of movements of DH. The patient is initially seated facing the front, the head is turned 45º towards the explored ear; next the patient is placed in the supine position, with the head rotated 30º under the horizontal, and the eyes of the patient are observed, in which a typical nystagmus of short latency (1-5 s) and limited duration (typically <30 s) appears. With the eyes in neutral position, the nystagmus has a vertical component, with the rapid phase upwards and a rotational component with the rapid phase towards the affected ear. The direction of nystagmus is reversed when the patient sits down again, and the nystagmus becomes fatigued if the manoeuvre is repeated.[2] The patient describes a feeling of dizziness or collapsing and the intensity of symptoms is not always proportional to the nystagmus response (Figure 2).

Figure 2. Dix-Hallpike test for the left posterior canal.

HORIZONTAL SEMICIRCULAR CANAL

The observation of a horizontal nystagmus with changes of direction when placing the patient in the supine position or performing the DH allows a diagnosis of BPPV of the horizontal semicircular canal to be established. To explore

this canal, the patient is placed in the supine position and the head is turned 90º toward the explored ear (McClure manoeuvre).[34] A horizontal nystagmus appears, with no vertical component, geotropic with the rapid phase towards the explored ear in most cases. Next, the head is turned toward the other ear, and a horizontal nystagmus in the opposite direction to the previous one, ie, geotropic, will be identified. This nystagmus indicates that the canalith is free in the lateral canal, a condition known as canalolithiasis and the most prevalent. In some cases, a non-geotropic nystagmus with shifting direction may appear, indicating that the lithiasis is attached to the dome (cupulolithiasis).[35]

A horizontal canal nystagmus presents different kinetic characteristics when compared to the vertical torsional nystagmus of the posterior canal. This horizontal nystagmus is of shorter latency (0-3 s), the intensity is higher, the duration may be greater than 1 min and the adaptation of the response or fatigue is lower than that observed in positional nystagmus of the posterior canal.[36] One feature that helps identify a non-geotropic nystagmus is the existence of a null point or position of the head in which there is no sign of nystagmus.[37] This point can be identified in decubitus (rotating the head until the nystagmus changes direction) and seated (by bending over 30º).

ANTERIOR SEMICIRCULAR CANAL

The anterior variation is considered the least common form of BPPV, with a frequency of 1%-24%.[26,36,38-40] The low incidence of lithiasis in the anterior canal is attributed to the anatomical characteristics of the labyrinth. Particles in the anterior canal can be eliminated because the posterior arm of the anterior canal descends directly into the common crus and the utricle. It is characterized by a positional nystagmus with downward movements, with a small torsional geotropic or non-geotropic component in response to DH[29] or to the hanging head manoeuvre.[39] Stimulation of the anterior canal generates an ocular movement response with contraction of the ipsilateral upper rectus muscle and the contralateral inferior oblique muscle, which produces the downwards vertical nystagmus.

Diagnosis is reached with positional tests: the DH manoeuvre stimulates the posterior and anterior vertical canals. The manoeuvre of hyper-extension of the head in decubitus (hanging head) on the midline may be useful to demonstrate a downward-beating vertical nystagmus, indicative of BPPV of the anterior canal.[39] The differential diagnosis between the 2 vertical variations is based on the direction of the vertical component in the rapid phase of the nystagmus response during the DH.[40]

Spontaneous or downward positional nystagmus has been observed in the Arnold-Chiari malformation, lesions of the posterior fossa or multiple systemic atrophy, with 3 forms of presentation: Parkinsonian, cerebellar, and autonomic (Shy-Drager type). This is why it should be mandatory to conduct magnetic resonance imaging with gadolinium to rule out a lesion of the central nervous system in these cases.[39]

BPPV IN VARIOUS SEMICIRCULAR CANALS

Lithiasis can occur simultaneously in several semicircular canals, which can hamper diagnosis and treatment. The observation of positional nystagmus with a vertical component during the Dix-Hallpike test on both sides must lead to caution, because the involvement of two canals is likely. In these cases, the manoeuvre described by Steddin et al[41] may be useful to differentiate unilateral BPPV of the posterior canal which is similar to bilateral nystagmus.

The use of video-oculography to record positional nystagmus in patients with BPPV has allowed identification of the rotational axis of the nystagmus and the identification of the canals involved in its generation.[28,29] In addition, this technology has made it possible to demonstrate that the incidence of atypical positional nystagmus in individuals with BPPV is higher than was previously estimated.[42] Atypical positional nystagmus includes anterior and horizontal canal variations, as well as multiple positional nystagmus (observed in several positional tests), indicating the simultaneous existence of lithiasis in several semicircular canals.[36]

Our series of cases with video-oculographic recording found 41% with unilateral BPPV of the posterior canal; 21% of cases with lithiasis of the horizontal canal; and 17% of individuals with involvement of the anterior canal. Furthermore, 20% presented multiple positional nystagmus during positional testing, reflecting an injury involving several canals; 5 cases were bilateral BPPV of the posterior canal; and another 2 patients presented downward-beating positional nystagmus in the DH test towards the right and left, as well as in the cephalic hyperextension manoeuvre, consistent with bilateral BPPV of the anterior canal. However, 7 individuals among our cases presented positional nystagmus with changing direction comprising vertical and horizontal components which could not be explained by the involvement of a single semicircular canal. These patients with multiple positional nystagmus presented changing patterns of nystagmus during the follow-up, confirming the complexity of these cases.[36]

TREATMENT OF BPPV

Treatment of BPPV has changed dramatically over the past 25 years. In the seventies, patients were given vestibular sedatives for the symptoms and warned not to perform the movements that caused the vertigo. In 1980, Brandt et al[43] suggested a few habituation exercises, with which the patient repeated the movements that caused the bout of vertigo, so as to achieve adaptation in the response of the vestibular nuclei. These positional habituation exercises, which cause the release and dispersion of particles in the dome or free in the canal, are poorly tolerated by many patients and do not prevent recurrence of crises.

At present, the treatment of BPPV is designed to transport the lithiasis particles from the affected semicircular canal to the utricle. The manoeuvres were initially described by Semont et al[44] in 1988 and Epley[3] in 1992, independently.

Semont et al proposed a manoeuvre based on the acceleration of the head by displacements carried out on an examining table to obtain dispersion of the particles and this became known as the particle releasing manoeuvre.[44] The results published initially were excellent, but many clinicians find it difficult to perform this manoeuvre in elderly and obese patients.

The manoeuvre described by Epley and the subsequent modifications by Parnes et al,[4] known as particle repositioning manoeuvre, has become the most popular treatment used for BPPV (Figure 3). The manoeuvre was initially done with sedation and the use of a mastoid vibrator. In the Epley manoeuvre, patients are moved sequentially into 5 positions, with the objective of shifting the canaliths by the force of gravity from the canal back towards the utricle. In practice, a modified version of the manoeuvre with three positions, called the particle repositioning manoeuvre, is used, eliminating sedation and the mastoid vibrator. Table summarizes the diagnostic and therapeutic manoeuvres used for BPPV at our centre.

Figure 3. Particle repositioning manoeuvre described by Epley for the treatment of BPPV of the left posterior canal.
S: starting position with the patient sitting; 1: left Dix-Hallpike position; 2: cephalic rotation from left to right; 3: right lateral decubitus with the head rotated to the right; 4: sitting with head rotation from the right to the midline; 5: head with slight anterior flexion.

TREATMENT OF POSTERIOR CANAL BPPV

A meta-analysis that included 3 clinical trials of high methodological quality has demonstrated a high effectiveness for the Epley manoeuvre, evaluated through the negativization of DH in the short term (odds ratio [OR] = 5.67; 95% confidence interval [CI], 2.21-14.56, favourable to the treatment).[11] There is no evidence that the Epley manoeuvre provides long-term resolution of the symptoms.

Figure 3 shows the examination of the labyrinth from the left side. The process begins in a sitting position (S), with the head rotated 45º toward the side under examination, next, the head and torso are moved as in the DH manoeuvre (1), and the canaliths move by gravity within the posterior canal; this position is maintained for 1-2 min. Next, the head is turned toward the right side (2), with the head hanging and the neck extended, causing the particles to move, until the patient lies with his or her face toward the floor (3), and this movement causes the particles to enter the common crus of the anterior and posterior canals. Finally, the patient sits (4) and the dispersed particles enter the utricle with a final flexion of the neck (5). John Epley proposed that the manoeuvre should be repeated until nystagmus is no longer induced, as a means of testing the efficiency of the treatment. However, this is debatable, since the repetition of the sequence produces an adaptation of the nystagmus response known as fatigue.

Although for many years patients were advised to avoid the decubitus position during the 48 h after treatment, to prevent the particles re-entering the canal, this measure does not appear to increase effectiveness.

TREATMENT OF THE LATERAL SEMICIRCULAR CANAL

Numerous techniques have been described for the treatment of horizontal canal BPPV, although the effectiveness has not been evaluated in random clinical trials. The simplest is the prolonged positional manoeuvre, developed by

Diagnostic and Therapeutic Manoeuvres for Benign Paroxysmal Positional Vertigo[a]

Clinical Variation	*Clinical Diagnosis*	*Video-Oculography*	*Treatment*
Posterior	Ipsilateral DH	Spontaneous nystagmus, cephalic agitation nystagmus, nystagmus in decubitus, left rotation,right rotation, left DH, right DH, hyperextension of midline in decubitus and bithermal caloric test[b]	Ipsilateral Epley
Horizontal	Bilateral DH. Bilateral head rotation		Lempert
Anterior	Ipsilateral/bilateral DH. Hyperextension of the midline		Contralateral Epley
Multiple	Bilateral DH. Head rotation. Hyperextension of the midline		Most symptomatic canal

[a]DH indicates Dix-Hallpike test.
[b]25% of patients with benign paroxysmal positional vertigo presented canalicular paresis.[16]

Vannucchi et al.[45] The patient is placed in lateral decubitus position with the affected ear facing upwards for 12 h. In the 35 patients in this series, the efficiency reached 90%, although 6 of these patients resulted in posterior canal BPPV.

Barrel rotation was described by Epley. It implies patients turning 180º on the examination table.[46] The starting position is the supine position, turning until the prone position is reached and then incorporating with the help of the knees and arms. This determines the movement of particles within the horizontal canal towards the utricle.

Lempert et al[47] suggested the barbecue manoeuvre, which is the technique most commonly used in Spain for horizontal canal BPPV. Here, the patient starts with the head turned completely towards the affected ear. The patient is turned quickly from the affected ear, increasing 90º up to a total of 270º with the head sustained in each position for 1 min. This manoeuvre causes the particles to migrate directly into the utricle by inertia or gravity. The barbecue manoeuvre was originally described only for the variation with geotropic nystagmus, but can also be used for the treatment of the non-geotropic form.

TREATMENT OF THE ANTERIOR SEMICIRCULAR CANAL

The experience accumulated on the treatment of ASC BPPV is scant and the works published are small series of patients, thus bringing their findings into question. In anterior canal disorders, the repositioning techniques normally used are the Epley manoeuvre or the Epley manoeuvre contralateral to the ear involved.[40,48]

HEALTH-RELATED QUALITY OF LIFE IN BENIGN PAROXYSMAL POSITIONAL VERTIGO

Quality of life is defined as the perception by individuals of their life situation in the cultural context and the values according to which they live; this includes a broad spectrum of domains such as health, economic resources, employment status, social relationships and leisure activities.[49] Health-related quality of life (HRQOL) is used to refer to the portion of the quality of life determined by the health of an individual.

We designed a prospective study in a series of 50 individuals with posterior canal BPPV to assess the outcome of the treatment and HRQOL in patients with BPPV of the posterior semicircular canal treated by the particle relocation manoeuvre.[50] All patients were treated with the specific manoeuvre and recurrences were evaluated through the DH test at 30, 180, and 360 days after treatment. The results were evaluated in terms of the negativization of DH and the scores obtained in the questionnaires SF36 (a general health questionnaire)[51] and DHIS (a specific, abbreviated questionnaire for vertigo and dizziness),[52] translated and adapted for Spanish-speakers.[53] DH was negative in 80% (40/50) of individuals within 30 days; 10, 7, and 5 patients experienced a positive DH after 30, 180, and 360 days respectively. Persistent BPPV was observed in 5% (2/50) cases after a year of follow-up, despite repetition of the treatment. Recurrences (DH+ after successful treatment) were observed in 7.5% (3/50) of patients after 6 months and 1 year, and the effectiveness of treatment was 88% after 1 year of follow-up.[50]

The HRQOL perceived by individuals with BPPV, as assessed by the SF36 questionnaire is worse than the standardized mean for the Spanish population in all dimensions, except for vitality.[54] After treatment, patients improved their scores on the SF36 and DHIS questionnaires, indicating a recovery of HRQOL after 30 days. Scores in the physical dimensions of the SF36 improved from the 30th day until 1 year. Likewise, the scores on the DHIS were significantly better after treatment ($P<.001$).[50]

As a result, patients with BPPV experience a decline in HRQOL that recovers after treatment. Although recurrences were observed in 7.5% of individuals, they did not affect the quality of life after 6 months or 1 year and the improvement of social and physical functions as well as perceived mental health, remained after treatment.[50]

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