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Benign Paroxysmal Positional Vertigo: An Overview

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Abstract: This study consists of a general review of benign paroxysmal positional vertigo and nystagmus. The main etiopathogenesis, diagnosis, and treatments are evoked. The author describes his experience on the subject.

Key Words: benign paroxysmal positional vertigo; diagnosis; etiopathogenesis; treatment

Benign paroxysmal positional vertigo (BPPV) has a sudden onset that is provoked by a certain position or appears in a determined position. This type of vertigo produces a nystagmus called *benign paroxysmal positional nystagmus* (BPPN). This disease occurs frequently and constitutes approximately 50% of acute vertigo complaint in my practice.

Barany [1] was the first to evoke BPPV in 1921, and Dix and Hallpike [2] described the characteristic torsional nystagmus in response to provocative positional testing (which was later named in their honor in 1952). In 1998, I published an article entitled "A state of the art" on this subject in the review of the Royal Belgian Ear Nose and Throat Society [3]. A report was presented by Sauvage et al. [4] at the Société Française d'Oto-rhino-laryngologie et de Chirurgie de la Face et du Cou in October 2007 and published by that society. In February 2008, 800 references about the subject were found in PubMed (Medline) on the Internet.

ETIOPATHOGENESIS

Several hypotheses have been evoked to explain the mechanism of BPPV and BPPN.

Lithiasis

Schuknecht [5] defined cupulolithiasis in demonstrating basophilic deposit on the cupula of the posterior semicircular canal. Kornhuber [6] posited the possibility of mechanical disturbances in a semicircular canal as the origin of the BPPN (e.g., blood clotting, a group of desquamated cells in the endolymph or perilymph). Hall et al. [7] first proposed that fragments of the otoconia floated in the endolymph to produce the BPPN; he called this pathology *canalolithiasis*.

Gordon [8] raised the possibility of an air plug floating in the semicircular canal. This theory is not supported by some characteristics of this nystagmus: It does not explain, for example, the direction of the nystagmus toward the higher ear when a patient reaches body rotation of 180 degrees. Brandt and Steddin [9] compared arguments for and against canalo- and cupulolithiasis.

In Support of Cupulolithiasis

The argument in favor of cupulolithiasis centers around a single histological finding of debris that seems to be attached to the cupula (Fig. 1). Those who argue against cupulolithiasis point to the absence of BPPV attacks with slow head tilt (>6 sec) and of typical vertigo with linear head accelerations.

In Opposition to Cupulolithiasis

The direction and intensity of induced nystagmus and vertigo do not reflect the position of a "heavy" cupula relative to gravity. Additional factors include a short duration of positioning nystagmus (<1 min) with the head motionless; clinical fatigability with repetitive positioning maneuvers; a spontaneous course with varying severity of the attacks; efficiency of physical therapy with unpredictable remission phases and relapses; and lack of compatibility with nystagmus direction in horizontal BPPV.

In Support of Canalolithiasis

Some argue in favor of canalolithiasis because of its compatibility with all clinical features of BPPV and nystagmus and with all arguments against cupulolithiasis (Fig. 2). Positive factors include the positioning of the

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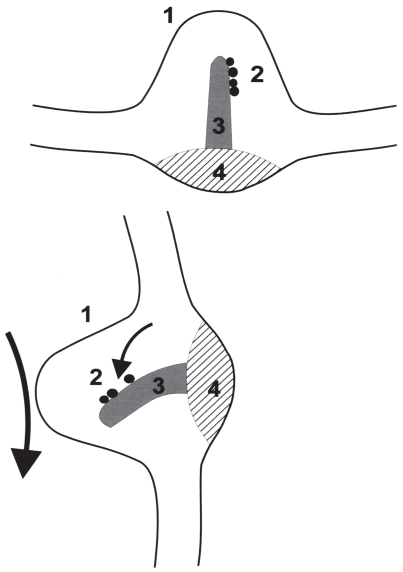

Figure 1. Cupulolithiasis: *1*, semicircular canal ampulla; *2*, fragments of otoconia on cupula of ampulla; *3*, cupula; and *4*, sensory cells.

nystagmus toward the uppermost ear as induced by 180-degree head tilt, providing indirect proof of canalolithiasis. Supporters of canalolithiasis emphasize its compatibility with features of horizontal BPPV and nystagmus.

In Opposition to Canalolithiasis

Those disagreeing with the implication of canalolithiasis cite histological findings of deposits in the semicircular canals in asymptomatic patients and the sudden onset of the disease (absence of slow buildup of clot and symptoms). All these theories, however, do not explain all instances of BPPN, such as those observed after alcohol or heavy water ingestion.

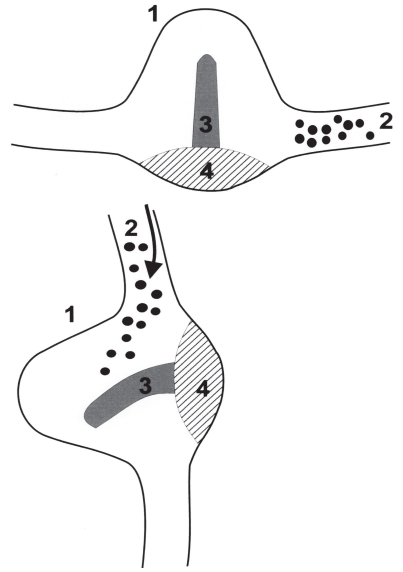

Figure 2. Canalolithiasis: *1*, semicircular canal ampulla; *2*, fragments of otoconias; *3*, cupula of the ampulla; and *4*, sensory cells.

Other Hypotheses

Some observations cast doubt on the role of canalolithiasis in the production of BPPN. Welling et al. [10] searched for the presence of particulate matter in the posterior semicircular canal of patients with and without a clinical history of BPPV. These authors compared 73 patients without BPPV symptoms who were undergoing labyrinthine surgery (vestibular schwannoma excision or labyrinthectomy) and 26 patients with BPPV who were undergoing the posterior semicircular canal occlusion procedure. Additionally, they searched microscopically for the presence of particulate matter within the lumen of the membranous labyrinth of 70 archived temporal bones without a history of BPPV. They did not observe any particles intraoperatively in any of the 73 patients without a history of BPPV. Particulate matter was observed in only 8 of 26 patients at the time of the posterior semicircular canal occlusion procedure for intractable BPPV.

Of the 70 temporal bones examined, 31 did not show significant postmortem changes, nor did they demonstrate cupulolithiasis or canalithiasis. Particulate matter from the membranous posterior semicircular canal was removed from one patient at the time of posterior semicircular canal occlusion for intractable BPPV symptoms and was examined using scanning electron microscopy. The particulate matter appeared morphologically consistent with degenerating otoconia.

Kveton and Kaslegarian [11] found a posterior canal fenestration in 10 patients undergoing acoustic tumor removal via a translabyrinthine approach. Particles were identified in the membranous labyrinth in 9 patients. Only 1 of these patients described preoperative positional vertigo. Electron microscopy demonstrated within the membranous labyrinth particles that appeared to be of mixed proteinaceous and mineral content. These data suggest that further studies must be undertaken before the theory of endolymphatic particle migration can be confirmed as the etiology of positional vertigo.

According to these studies, it appears that the existence of canalolithiasis does not always produce BPPN. Why? That is the question; the answer awaits further investigations.

After they discovered BPPN in a patient with a cerebellar glioma, Riesco and McClure [12], attributed the nystagmus to a loss of inhibition of the vestibular system by the cerebellum. In experiments with a cat, Fernandez

[13] found BPPN with the ablation of the cerebellum nodulus. Initially, Jongkees [14] and, earlier, Cope and Ryan [15] in another publication hypothesized that cervical trauma or degenerative lesions, as spondylosis, disturbed proprioceptive information to the vestibular system and figured in the origin of BPPN.

Sangstrom [16] suggested that a compression of vertebral arteries by osteophytes produced cerebral ischemia and secondary BPPN. Miehlke [17] observed BPPN secondary to trauma of the inner ear, with blood in the inner ear. Stenger [18] found BPPN in one case of labyrinthic fistula. Citron and Hallpike [19] observed that the labyrinthic destruction suppressed the BPPN. Uemura and Cohen [20] demonstrated the inhibitory influence, on positional nystagmus, of a rostral lesion of the descending vestibular nucleus that receives information from otolithic organs.

Fluur [21] submitted the hypothesis that BPPN was produced by a perturbation of the integration of utriculoampullar information. Kornhuber [6] and, later, Fluur [22] demonstrated that the direct stimulation of otolithic organs did not produce BPPN. Mayne [23] and Oosterveld et al. [24] posited a causal origin in a disturbance of the integration system of the utricular and semicircular canal information. In a study published in 2006, Monzani et al. [25] provided clinical evidence of a potential role of emotional stress connected to adverse life events as a trigger of otoconial dysfunction.

At the Thirty-First Congress of the Neurootological and Equilibriometric Society in 2004, Barozzi and Cesarani [26] presented an interpretive model exemplifying the cybernetic theory. Korres [27] found frequent electronystagmography abnormalities in patients with BPPV, most commonly including canal paresis of the involved side. In 2003, Vibert [28] found a correlation between BPPV and osteopenia or osteoporosis in women with BPPV who were at least 50 years of age. In 2008, Anagnostou et al. [29] reported the case of one patient with acute-onset positional vertigo mimicking BPPV with a single enhancing lesion in the inner part of the superior cerebellar peduncle, disclosed only after thin-slice magnetic resonance imaging.

Several mechanisms probably are invoked, and probably interactions between various amounts of information issued from the semicircular canals and otoliths also occur. To study the utricular function, Anastasopoulos et al. [30] investigated linear vestibuloocular reflexes (LVORs) during lateral whole-body translation in 14 patients with unilateral BPPV. These authors noted the absence of significant changes of the LVOR, and they explained that fact either by minor utricular damage, which is functionally irrelevant, or by central compensation of a chronic unilateral deficit.

Iida et al. [31] performed a pendular nondamped rotation test in a head-tilted position (60 degrees backward and then rotated 45 degrees to either the right or left) in 6 patients with BPPV. The excitability of the posterior canal in the affected ear was found to be lower than that of the anterior semicircular canal. In 2003, Gacek [32] suggested that the pathophysiological mechanism responsible for a position-induced vestibuloocular response in this disorder is neural rather than a mechanical stimulation of the sense organ. Loss of the inhibitory action of otolith organs on canal activation caused by degeneration of otolith neurons (saccular, utricular) is a possible explanation of the brief canal response induced by the positional stimulus.

According to the Gacek hypothesis [32]:

Features are not in relation with mechanical physiopathology:

1. The latency, limited duration, and fatigability of the rotatory nystagmus despite sustained provocation;
2. The long (sometimes years) periods of remission between episodes of BPPV activity;
3. The absence of nystagmus in the presence of subjective symptoms with provocation in some patients;
4. The absence of basophilic deposits in the cupula and membranous canal of the posterior canal sense organ in many of the donor temporal bones with a history of BPPV.

A qualitative and quantitative examination was performed on temporal bones from 5 donors with antemortem diagnoses of BPPV. These observations support a neural concept of BPPV rather than one based on a purely mechanical hypothesis.

The major pathological change founded in BPPV is degeneration of vestibular neurons, rather than an alteration in receptor sensitivity. Significant loss of superior vestibular ganglion and inferior vestibular ganglion cells was found in all temporal bones, whereas vestibular sense organs were normal. The assessment of vestibular ganglion cell loss revealed an approximately 50% loss in the superior vestibular ganglion of all 5 temporal bones and in the inferior vestibular ganglion of 3 temporal bones with BPPV.

The inferior vestibular ganglion of the remaining 2 temporal bones revealed a 30% loss of neurons but showed degenerative changes in saccular ganglion cells. The cause of this ganglionic degeneration is probably the reactivation of latent neurotropic viral infection [33].

Several additional observations are not explained on the basis of a change in the motion mechanics of cupular displacement in the positional test. The limited duration of nystagmus while provocation is maintained and the fatigability of this response cannot be explained by a change in the gravity sensitivity of the cupula. These features are more consistent with a refractory state of first-order vestibular neurons. The absence of nystagmus in patients with subjective

symptoms when provoked by the positional stimulus is also difficult to explain on the basis of gravity-sensitive deposits in the endolymph. Therefore, the concept of a gravity-sensitive change in semicircular canal physiology in BPPV is inadequate to explain most of the ocular response features of this disorder.

When the head is placed in the so-called Hallpike position, the hair cells in the superior part of the saccule and those in the posterior canal crista are depolarized, activating antagonistic extraocular muscles. However, if the saccular macula or its neural input is degenerated, the antagonistic effect on posterior canal input is lost, and the rotatory upbeat nystagmus associated with posterior canal receptor activation is released.

If the posterior canal neural input were also degenerated, nystagmus would not appear in the ear with a degenerated saccular nerve. A degenerated or atrophic singular nerve was never encountered in more than 250 surgical exposures of this nerve in patients with chronic BPPV. The nystagmus of posterior canal BPPV, therefore, may result from inadequate inhibition from the saccular macula, especially its superior part. In a similar way, lateral canal BPPV may represent decreased utricular inhibition of lateral canal activation.

The severity (nystagmus and nausea) of the provoked response may depend on the degree to which the saccular input is impaired. It is possible that some patients may have only nausea and imbalance without nystagmus, if the saccular deficit is minimal. If the saccular loss is almost total, the vertigo and nausea may be disabling. Most BPPV patients fall somewhere between these two extremes.

Alleviation of the response in these patients by posterior rehabilitation maneuvers (PRMs) may result from stimulation of remaining functional units in the saccule, which inhibits activation of the posterior canal crista. This form of treatment would succeed in cases in which there is sufficient residual saccular input.

von Brevern et al. [34] provide evidence that idiopathic BPPV is associated with utricular dysfunction. Manzari [35] suggested that recurrent BPPV is related with volumetric abnormalities of the vestibular aqueduct. It is also essential to remember that important metabolism dysfunction or abnormalities of the function of the central nervous system may be at the origin of liberation of deposits from the otoliths in the semicircular canals.

Kikuchi et al. [36] demonstrated the incidence of slow blood flow in the vertebrobasilar system by magnetic resonance imaging in cases of direction-changing positional nystagmus. *It is the demonstration that a complete otoneurological examination has to be reached to reveal the origin of the affection and to give a prognosis for the success of treatment*.

CLINICAL SYMPTOMATOLOGY

Vertical Benign Paroxysmal Positional Vertigo

Vertical benign paroxysmal positional vertigo (VBPPV) is revealed by the Hallpike maneuver that provoked the vertical benign paroxysmal positional nystagmus (VBPPN). Characteristics include a variable latency from 2–3 to 15 seconds, a vertigo or distress sensation, a rotatory nystagmus toward the downturned ear, in which intensity increases progressively and decreases to disappear in several seconds, an inversion of the sense of rotation of the nystagmus when a patient is re-placed in a sitting position, and the suppression of VBPPN after some maneuvers.

The usual consideration is that, if one of these characteristics is missing, the vertigo is not a VBPPN. However, a study published in 1982 [37] concerning 194 cases of VBPPN (138 unilateral and 56 bilateral) noted several particular cases, including those in which one of the characteristics may be absent without central disease and any other pathology. The author also found a syndrome in which the VBPPN exists for some days to some weeks, disappears, and reappears after a variable period. When the VBPPN is very important in the contralateral Hallpike position, a rotatory nystagmus toward this side can occur. Some subjects present a VBPPN that has existed for several years. In such cases, the characteristics vary from one examination to the other and, in some cases, the subjects present a vertical component only.

The VBPPN is suppressed when, before testing and with the patient in a sitting position, the contralateral ear is irrigated with cold or warm water [38]. Another research study [39] demonstrated the absence of correlation between the VBPPN and the existence of a directional preponderance syndrome of the nystagmus.

Horizontal Benign Paroxysmal Positional Vertigo

In 1985, Cipparone et al. [40] in Italy and McClure [41] in the United States observed horizontal benign paroxysmal positional vertigo (HBPPV). In 1989, Pagnini et al. [42] described the characteristics of this condition in a series of 15 patients: very short latency (<5 sec) in the lateral decubitus position; presence of geotropic or apogeotropic nystagmus (Fig. 3); progressive increase and decrease of the condition, often for more than 30 seconds, and occasional variation of its direction from geotropic to apogeotropic; an often horizontal orientation; in contralateral decubitus, the possibility of presentation in an opposite direction; and frequent inhibition by ocular fixation.

In 1996, Nuti et al. [43] reported data emerging from a study of 123 patients. In 2001, von Brevern et al. [44]

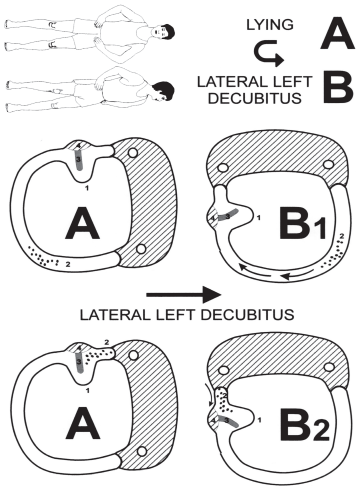

Figure 3. Horizontal canalolithiasis. *A*, Position of otoconias at rest. *B1*, Movement of otoconias induces ampullopetal deflexion of the ampulla → left horizontal nystagmus = geotropic nystagmus. *B2*, Movement of otoconias induces ampullofugal deflexion of the cupula → right horizontal nystagmus = apogeotropic nystagmus. *1*, semicircular canal ampulla; *2*, fragments of otoconias; *3*, cupula of the ampulla; and *4*, sensory cells.

described the case of a patient with right HBPPV that showed a spontaneous nystagmus beating to the left, which disappeared after the rehabilitation technique and not after the PRM technique. In 2005, Vannucchi et al. [45] stressed the rules to identify the impaired side for lateral semicircular canal BPPV. These authors determined that the affected side of the *geotropic* forms is (1) the side on which the nystagmus is more intense, (2) the side on which spontaneous inversion occurs or is more evident, and (3) the side opposite the direction of the nystagmus when the patient is brought from the seated position to the supine position. In contrast, the affected side of the *apogeotropic* forms is (1) the side on which the nystagmus is less intense, (2) the side opposite that on which spontaneous inversion occurs, though this phenomenon is not frequent, and (3) the side to which nystagmus beats when the patient is brought from the seated position to the supine position.

In 2006, Choung et al. [46] developed a new test—the bow and lean test—to easily determine the ear affected by HBPPV and to evaluate its efficiency.

Anterior Canal Benign Paroxysmal Positional Vertigo

In 1994, Steddin and Brandt [47] reported characteristic clinical findings involving the anterior canal. In the Hallpike position, the nystagmus is down-beating, and its duration varied from 5 to 60 seconds, sometimes more. Its frequency ranges from 1.2% to 12% of the BPPV [48, 49]. In 2007, in a population of 260 patients with BPPV, Jackson et al. [50] described the existence of anterior canal benign paroxysmal positional vertigo (ABPPV) in 21.2% of study cases.

According to Lopez-Escamez et al. [51], these patients may show alteration in the vestibular calorics, and they can have multicanal effects. Some cases demonstrate a complex situation with a mixture of two VBPPVs [47] or a combination of HBPPV and VBPPV [52–54].

TREATMENT

After the completion of a full otoneurological examination and the exclusion of a central nervous disease, the best results are obtained by "liberatory maneuvers." Many maneuvers have been proposed by several authors.

Vertical Benign Paroxysmal Positional Vertigo

Maneuvers for VBPPV have been devised by Herdman and Tusa [52]; Brandt and Daroff [55]; Harvey et al. [56]; Semont et al. [57]; Toupet and Codognota [58]; Epley [59]; Li and Epley (the 360-degree maneuver) [60]; and Cohen [61]. The most frequently used are the Semont and Epley maneuvers.

Horizontal Benign Paroxysmal Positional Vertigo

Lempert and Tiel-Wilck [62] and their "barbecue rotation," Vannucchi et al. [63], de la Meilleure et al. [64], Crevits [65], and Gufoni and Mastrosimone [66] have all designed appropriate maneuvers for HBPPV. In 2004, Casani et al. [67] standardized the treatment protocol consisting of a barbecue maneuver followed by "forced prolonged position" in cases of geotropic nystagmus and a modified fourth step of the Semont maneuvers for apogeotropic nystagmus. In 2006, Asprella and Libonati [68] proposed the "strategy of the minimum stimulus" to treat semicircular canalolithiasis.

Anterior Canal Benign Paroxysmal Positional Vertigo

Until now, no consensus has been established for use in the ABPPV pathology. We prefer vestibular habituation training to the liberatory maneuvers in this pathology.

To learn the liberatory maneuvers, Beyea et al. [69] used a device (the DizzyFIX) designed to be a visual representation of the particle repositioned maneuver. This device, with a Web module consisting of a series of slides that outline the steps of the PRM, is particularly useful to teach the PRM and demonstrates its superiority over standard classroom instruction.

It is possible to use some mechanical devices to realize the liberatory maneuver [60,70]. Many authors have demonstrated the efficacy of this type of treatment. In 2007, Korm et al. [71] demonstrated in a group of 123 patients that repeated Epley maneuvers in fewer sessions rendered more positional nystagmus–free patients when compared to those submitted to more sessions of single maneuvers.

Ganança et al. [72] objectively demonstrated the effectiveness of the Epley maneuver in BPPV associated with Ménière's disease. In a 2005 meta-analysis of nine controlled studies consisting of 505 patients, Withe et al. [73] suggested that canalith repositioning is a safe and effective treatment of BPPV. A single session successfully resolves positional nystagmus 72% of the time; symptoms spontaneously resolve at 3 weeks in one-third of patients.

In a 2004 meta-analysis, Woodworth et al. [74] demonstrated the efficacy of the Epley maneuver. In 2000, Macias et al. [75] studied variables affecting treatment of BPPV in 259 patients. The main variables include bilateral disease or location of disease other than in the posterior canal. Patient age, gender, method of diagnosis, and onset association with trauma had no statistically significant impact.

In cases of failure, the techniques of habituation, such as the vestibular habituation training of Norré and Beckers [76], or another training maneuver, such as that of Fujino et al. [77], are used. In 2004, Barozzi and Cesarani [26] proposed a new treatment with relaxation and vestibular electrical stimulation in patients resistant to two maneuvers or with recurrent vertigo. In a 2005 study involving 840 patients, Steenerson et al. [78] demonstrated that the treatment of BPPV can be effective using repositioning, liberatory, or log-roll maneuvers in combination with redistribution exercises. Steiner et al. [79] proposed a virtual reality approach for the treatment of BPPV.

In those rare cases in which the maneuvers or exercises fail, surgical treatment is possible, including semicircular posterior canal nerve section [80] or obliteration of the canal [81].

OUR EXPERIENCE

Reported here are some statistics collected by me on 190 cases of VBPPV, 30 cases of HBPPV, and 10 cases of ABPPV.

Vertical Benign Paroxysmal Positional Vertigo

The Semont maneuver employed in 190 cases of VBPPV resulted in disappearance of symptoms after only one maneuver in 170 cases. VBPPV disappeared after two maneuvers in 10 cases, and failure occurred in 10 cases. In the failed cases of vestibular habituation training, success was achieved in 9 cases in a maximum of 2 weeks, and failure in 1 case resulted in spontaneous remission after 6 weeks.

Horizontal Benign Paroxysmal Positional Vertigo

The Vannucchi maneuver achieved 89% success in 3 days for 20 of 30 cases of HBPPV. The Lempert maneuver in 10 cases brought 86% success after one maneuver. In the cases in which this maneuver failed, VHT always was successful.

Anterior Canal Benign Paroxysmal Positional Vertigo

In 10 cases of ABPPV, the VHT of M. Norré led to 100% success.

INVESTIGATING BPPV

The Royal Belgian Ear, Nose and Throat Society recently published guidelines on vertigo and dizziness [82]. In regard to the diagnostic progression in cases of BPPV, the suggested guidelines are as follows:

1. First episode of positioning vertigo
   - 1.1 If the history is evocative of BPPV,
     - perform otomicroscopy and a hearing test,
     - search for the pathological canal, and
     - execute the repositioning maneuver.
     - After one week, check the patient:
       - — If he or she is asymptomatic, the investigation is complete.
       - — If residual symptoms persist after two or three repositioning maneuvers, see the next section.
   - 1.2 If history and clinical presentation are "atypical"
     - Baseline explorations should include
       - — a complete clinical examination:
         - hearing tests,
         - brainstem evoked response audiometry,
         - videonystagmography or electronystagmography with rotatory and caloric proofs,
         - oculomotricity,
         - the subjective visual vertical perception test, and
         - vestibular evoked myogenic potentials.
         - As a function of these results, conduct
       - — a neurological examination and/or
       - — specific neurological imaging.

2. In case of relapse of positioning vertigo
   - Perform baseline explorations (see section 1.2).
   - Obtain a temporal bone scan if conductive hearing loss is present.

CONCLUSIONS

Liberatory maneuvers allowed a very quick disposition of all the symptoms of BPPV in the majority of cases. A recent study [83] confirms this opinion. It is absolutely necessary that these maneuvers be conducted by experienced practitioners. We propose to use the recommendations of the Royal Belgian Ear, Nose and Throat Society to diagnose and treat this type of vertigo.

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