

# BENIGN PAROXYSMAL POSITIONAL VERTIGO

GUEST EDITORS: STAVROS KORRES, LINDA LUXON, PAOLO VANNUCCHI, AND BILL GIBSON





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Guest Editors: Stavros Korres, Linda Luxon,  
Paolo Vannucchi, and Bill Gibson



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## Editorial

# Benign Paroxysmal Positional Vertigo

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Dizziness and vertigo are among the most frequently encountered symptoms in primary care, with benign paroxysmal positional vertigo (BPPV) being the commonest type of vertigo. Its clinical course may vary considerably from a self-treatable to a persisting and/or recurrent disabling problem, with as yet unidentified prognostic factors. Although it is named as such, there are a considerable number of patients who do not perceive it as a benign disease, but rather as an incapacitating condition that restricts their routine activities and has a significant impact on their quality of life [1, 2].

*Current Diagnosis and Management.* Until the theories of canalolithiasis and cupulolithiasis were reported, the treatment of BPPV had been based on either the avoidance of the provoking positions or habituation. The assumption that the dislodgment of otoconia toward the semicircular canals or the ampulla is the underlying pathophysiological mechanism has led to the development of canalith repositioning procedures (CRPs) [3–6]. Indeed, the successful results attributed to CRPs seem to have verified the respective theories. The careful observation, through the Frenzel glasses or videonystagmography, of the nystagmus provoked by simple changes in the position of the patient's head can usually provide the ability to localize the dislodged otoconia in the ampulla or the lumen of one or more of the six semicircular canals (SCCs) [7]. In some cases diagnosis can also go as far as detecting, for example, that the dislodged otoconia is located in the posterior arm of the horizontal SCC (canalolithiasis) if the nystagmus is geotropic in side positions, or in the anterior arm of the horizontal SCC, either free floating

(canalolithiasis) or attached to the cupula (cupulolithiasis), if the nystagmus is apogeotropic [8–10]. The details in the diagnosis that a specialized observer can reach through a noninvasive and simple-to-perform examination are indeed quite amazing, while the observation of nystagmus during CRPs allows speculations on the movement of debris and the appropriate treatment strategy. Finally, the simultaneous or successive insult of multiple canals might be a complex issue in the diagnosis and treatment of BPPV.

*Challenges in Diagnosis and Treatment.* Due to the existence of several and to a large extent unknown contributing factors, BPPV remains a challenging field that is constantly evolving in terms of pathophysiology, clinical manifestation, recovery, treatment, and recurrence. For example, clinicians sometimes encounter atypical and intractable BPPV patients who show frequent relapses or poor response to physical therapy. Anatomic variations, stenoses in the SCC lumen, or multiple clots of particles in the same SCC which cause unpredictable endolymphatic currents can account for some of the “difficult” cases. Another observation with intriguing underlying pathophysiology is the fact that the treatment of BPPV secondary to head trauma is less effective than that of idiopathic BPPV. Several other causes of intractability in BPPV have been reported including osteoporosis [11], trauma [12], position during bed rest [13], diabetes [14], and Ménière's disease [15]. Furthermore, there seems to exist an interesting but poorly understood relationship between migraine and BPPV [16, 17]. Finally the anterior SCC was considered in the past as being free from dislodged otoconia

due to its anatomical position, but recent observations have proved that such a BPPV variation, although rare, is indeed possible [18, 19].

The considerable variation in the vertical and torsional contributions to the nystagmus induced by the Dix Hallpike maneuver, especially when considering the anterior and posterior canals, constitutes another pathophysiologic and diagnostic question which cannot be explained solely by the canalolithiasis and cupulolithiasis theories [20]. The role of the interactions between the semicircular canals and the otolith organs in the clinical signs and symptoms, as well as the recovery from BPPV, is still under investigation [21, 22]. Potential involvement of the vestibular nuclei, ganglia, peripheral nerve fibres, and central nervous system vestibular centres is also being studied [23].

Recent high-resolution magnetic resonance imaging (MRI) seems to be able to identify an obliteration of the membranous labyrinth as filling defects of the inner ear fluid spaces, semicircular canal stenosis, and/or a plug of otoconial debris, and morphological abnormalities in the inner ear of patients with intractable BPPV such as fractures, stenoses, filling defects, and a fold of semicircular canals have been reported [24, 25].

BPPV is a frequent disease which is usually resolved spontaneously or by office-based canalith repositioning maneuvers. In some cases, however, it may also be manifested as a persisting and/or recurrent disabling problem. During the last decade, the treatment of these “difficult”, although rare, cases has rendered BPPV a constantly evolving and intriguing subject of investigation. Partly due to its numerous potential variations and partly due to its yet unknown pathophysiological mechanisms and predisposing and prognostic factors, BPPV remains a challenging and promising investigation field.

Stavros Korres  
Linda Luxon  
Paolo Vannucchi  
Bill Gibson

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## Clinical Study

# Why Treat Apogeotropic BPPVs of the Horizontal Canal? About 30 Observations

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Benign paroxysmal positional vertigo (BPPV), of the horizontal canal, in the apogeotropic form (AHBPPV) was described in 1995. Based on 30 observations of typical AHBPPVs of the horizontal canal, we endeavor to discuss the relevance of physiotherapy. *Material and Method.* Thirty observations of typical apogeotropic BPPVs of the horizontal canal treated with a 360° barbeque rotation on the BPPV side, reviewed in consultation at 1 and 3 weeks and reevaluated the following year. *Results.* Our cohort of 30 patients had an average age of 58.6 years. The apogeotropic BPPVs of the horizontal canal, which can be transformed into BPPVs of the posterior canal or into geotropic-type BPPVs of the horizontal canal do not recover more quickly. Patients who follow the positional advice do not recover more quickly than those who do not ( $P = 0.152$ ). The 15 patients treated on average 13.73 days after the onset of the disease did not recover more quickly after the start of therapeutic treatment than those treated later ( $P = 0.032$ ). *Conclusion.* Here, we demonstrate that the direction of rotation during the maneuvers is of no importance for the results. We show that transformability is not a guarantee of rapid recovery and that the therapist's effectiveness is limited when it comes to the short-term results.

## 1. Introduction

Benign paroxysmal positional vertigo (BPPV) of the horizontal canal was described in 1985 [1] as geotropic form (downbeating nystagmus, geotropic BPPV of the horizontal canal, GHBPPV). The apogeotropic form has been described more recently in 1995 [2]. This form, apogeotropic BPPV of the horizontal canal (AHBPPV), is characterized by an upbeat horizontal nystagmus provoked by a right or left lateral decubitus.

It accounts for between 16 and 26% of BPPVs of the horizontal canal [3], that is, approximately 2 to 4% of BPPVs where an impact on the posterior canal is dominant, an incidence which is slightly higher than that of the BPPVs of the anterior canal [4]. For 15 years now and since the first description of BALOH, therapeutic solutions have been discussed and must be validated.

Based on 30 observations of typical apogeotropic BPPVs of the horizontal canal, we endeavor here to discuss the relevance of physiotherapy and what it offers patients in terms of comfort and symptomatic relief.

## 2. Material and Method

*2.1. The Patients.* Between 2006 and 2007, 30 patients came to the clinic for exploration and vestibular rehabilitation for treatment of a typical AHBPPV, without central cause possible (MRI and neurologic examination normal).

### *2.2. The Method*

*2.2.1. The Treatment.* Each patient underwent a 360° barbeque rotation maneuver of the affected side.

To determine which side of the AHBPPV was affected we used the following arguments in order: (1) the side where the nystagmus was the weakest, (2) then the side in which vertigos were the weakest, (3) and lastly the way the nystagmus went to when the head was bent forward (bow and lean test).

The patients were reviewed during a systematic control at 1 and 3 weeks. A 360° barbeque rotation on the affected side was undertaken for the apogeotropic BPPVs of the horizontal canal.

When, during a session, we transformed the AHBPPV, we immediately performed the adapted maneuver: 270° LEMPERT's Barbeque roll maneuver on the side of the healthy ear on a GHBPPV and SEMONT's liberatory maneuver on a posterior canal BPPV (PBPPV).

Eight pieces of prophylactic advice were given after each maneuver.

In the case of an AHBPPV, these positional pieces of advice consisted of (1) sleep on the BPPV side, (2) put the night stand on the side of the BPPV, (3) do not make housework with the head tilted back, (4) or bent forward, (5) do not practice makeshift job with the head tilted back, (6) or bent forward, (7) do not work in garden with the head tilted back, (8) or bent forward.

*2.2.2. The Retrospective Information from the Clinical File.* Data were compiled from the clinical file on sex, age, time to therapeutic treatment, the side affected, transformability (into BPPVs of the posterior canal or geotropic BPPVs of the horizontal canal), the time to symptomatic recovery, and the number of maneuvers or consultations needed to obtain symptomatic recovery. The symptomatic or clinical recovery was obtained with disappearance of positional vertigo. The videoscopic recovery was obtained with disappearance of videoscopic positional nystagmus.

*2.2.3. Retrospective Information on the Control Consultation.* Thirty patients were reviewed in consultation in 2008 and we undertook the following.

- (1) An evaluation of the residual symptoms using the Vertigo Symptom Scale (VSS) [5] and the Dizziness Handicap Inventory (DHI) [6]. VSS and DHI are expressed as a total out of 100 points. The symptomatic score of our patients is expressed as a total out of 200 points (sum of VSS and DHI).
- (2) An evaluation lasting 10 seconds of residual nystagmus in videonystagmoscopy in the Head Shaking Test, in anteflexion, in right HALLPIKE, and left HALLPIKE, in right lateral decubitus and left lateral decubitus. The score was recorded as 1 if there was nystagmus, as 0 if there was none. The total score of each patient was evaluated in respect of 6 points.
- (3) An evaluation of the following prophylactic advice given at the end of each consultation. This following of the positional advice was evaluated for 8 items using the values 1 (always), 2 (almost always), 3 (almost never), 4 (never). The total number of points obtained for each of these items enabled us to calculate a positional risk score out of 32 points.

*2.3. Statistics.* We used the SPSS software. For the comparisons of quantitative values we drew on the Fischer test, and a  $P < 0.05$  was used as the test for a significant hypothesis. We then compared the averages between the two groups of 15 on both sides of the median. The correlations were evaluated using the Pearson score.

### 3. Results

Table 1 gives the diagnosis elements, the delays, the transformability and the evolution. This cohort of 30 patients had a gender ratio of 11 men to 19 women, 36.66%.

The average age was 58.6 (standard deviation 15.22, median 63, extremes 24 to 82 years of age). The positional vertigos on the day of the first consultation had been developing for 11.6 days on average (standard deviation 10.90, median 9, extremes 1 to 40 days). In 19 cases (63.33%), we determined the affected side thanks to the side in which the nystagmus in lateral decubitus (NLD) was the weakest. We used the side wherein vertigo was the weakest in 5 cases (16.66%, when right NLD = left NLD). Lastly, in 6 cases (20%) we delineated it thanks to the nystagmus caused by the head bent forward (bow and lean test, when right NLD = left NLD and right vertigo = left vertigo).

Fifteen patients had a right apogeotropic, 15 a left apogeotropic BPPV of the horizontal canal.

About the transformability at first consultation (C1 on Table 1), after the first maneuver (M1 on Table 1), 5 AHBPPV were transformed in GHBPPV. 3 of them remained on the second consultation, the 2 others became a PBPPV and a AHBPPV.

Regarding transformability and time to recovery at the second consultation (C2 on Table 1), we observed in our cohort during the first control consultation after 8.6 days on average: 15 patients who had recovered in terms of symptoms, but only 8 who had recovered from the angle of videoscropy (no positional nystagmus). In the case of the 22 others: 11 patients with a transformed BPPV (4 into PBPPV and 7 into GHBVVP) and 11 AHBPPVs of the horizontal canal.

Regarding transformability and time to recovery at the third consultation (C3 on Table 1, second control consultation) after 14.8 days on average we obtained for our 22 cases: 18 symptomatic recoveries and 14 videoscopic recoveries. For the 8 other patients with positive videoscropy we obtained 3 transformed BPPVs (1 BPPV of the posterior canal and 2 geotropic BPPVs of the horizontal canal) and 5 apogeotropic BPPVs of the horizontal canal.

Finally, the number of maneuvers and the number of consultations necessary to obtain videoscopic recovery are linked, but they also depart from each other. The average number of consultation is 2 and the average number of maneuvers is 2.2 for all our cases. If we only consider the 28 patients clinically cured on the third consultation, the average number of consultations is 1.92 and the average number of maneuvers is 2.10.

Table 2 summarizes our statistical study and the Fischer test. We examined the impact of the six factors on symptomatic and videoscopic recovery some time after the initial episode.

*3.1. Influence of the Factor "Age".* There is no significant difference regarding the distance in terms of time from the initial episode between the young and older groups of patients. Their symptomatic and videoscopic scores are similar and for these two elements the small  $P$  values are

TABLE 1: The elements which enabled the authors to undertake the diagnosis, the delays, the transformability and the evolution.

Case	Sex	Age	Duration before C1 (days)	Dominant side nystagmus	Dominant side vertigo	Bow test nystagmus	Side of BPPV	VNS type of BPPV at C1	VNS type of BPPV after M	Time between C1 and C2 (days)	Vertigo at C1	VNS type of BPPV at C2	VNS type of BPPV after M	Time between C2 and C3 (days)	Vertigo at C3	VNS type of BPPV at C3	VNS type of BPPV after M	Total of M	Total of C
1	M	46	13	R > L	R > L	0	L	APO	APO	20	No							1	1
2	M	47	5	L = R	L = R	R	L	APO	APO	6	No							1	1
3	M	67	5	R > L	R > L	R	L	APO	APO	5	No							1	1
4	F	54	1	L > R	L > R	L	R	APO	APO	3	No							1	1
5	F	53	21	L > R	L = R	L	R	APO	APO	19	No							1	1
6	F	24	1	L > R	L > R	L	R	APO	APO	5	No							1	1
7	M	29	28	L > R	L > R	L	R	APO	APO	20	No							1	1
8	M	54	2	R > L	R = L	R	L	APO	APO	4	No							1	1
9	F	64	2	L = R	L = R	R	L	APO	APO	7	No	APO	APO	37	No	No		2	2
10	F	66	8	L > R	L > R	0	R	APO	GEO	8	No	APO	APO	14	No	No		3	2
11	F	74	4	L > R	L > R	0	R	APO	APO	4	Yes	APO	GEO	10	No	No		3	2
12	F	49	10	R > L	R > L	R	L	APO	APO	10	Yes	APO	APO	31	No	No		2	2
13	F	64	12	L = R	R > L	R	L	APO	APO	4	Yes	APO	APO	8	No	No		2	2
14	F	80	2	L = R	R > L	R	L	APO	GEO	4	No	GEO	GEO	42	No	No		3	2
15	F	41	9	L > R	L = R	0	R	APO	APO	5	Yes	GEO	GEO	21	No	No		2	2
16	M	47	2	R > L	R > L	0	L	APO	APO	10	Yes	GEO	GEO	14	No	No		2	2
17	F	63	26	L > R	L = R	0	R	APO	APO	8	Yes	GEO	GEO	30	No	No		2	2
18	F	43	3	L = R	L = R	L	R	APO	APO	4	Yes	GEO	GEO	20	No	No		2	2
19	M	47	10	L > R	L > R	L	R	APO	GEO	5	No	POS	POS	21	No	No		3	2
20	F	80	40	L = R	L = R	R	L	APO	APO	8	No	POS	POS	15	No	No		2	2
21	F	82	15	L = R	L = R	L	R	APO	APO	6	No	POS	POS	15	No	No		2	2
22	F	50	13	L > R	L = R	L	R	APO	APO	6	No	POS	POS	12	No	No		2	2
23	F	77	11	R > L	R > L	R	L	APO	APO	17	Yes	APO	APO	47	No	No	APO	3	3
24	M	63	1	L = R	L > R	0	R	APO	APO	2	Yes	APO	APO	6	No	No	APO	3	3
25	F	75	8	R > L	R > L	R	L	APO	APO	16	Yes	APO	APO	30	No	No	APO	3	3
26	M	75	40	L = R	L > R	0	R	APO	APO	6	Yes	APO	APO	13	No	No	APO	3	3
27	F	64	26	L = R	R > L	R	L	APO	APO	20	Yes	APO	APO	49	No	No	APO	3	3
28	F	65	3	L = R	L = R	R	L	APO	GEO	9	No	GEO	GEO	44	No	No	GEO	4	3
29	M	72	15	R > L	L = R	R	L	APO	GEO	11	Yes	GEO	GEO	15	Yes	Yes	GEO	4	3
30	M	43	12	L > R	L > R	L	R	APO	APO	6	Yes	APO	APO	21	Yes	Yes	POS	3	3

TABLE 2: Summarizes our statistical study and the Fischer test.

	Average age	Average positional risk	Average scarring time	Average number of consultations before recovery	Average time to consultation	Average VSS and DHI scores	Average videoscopic score
Young subjects	46	15.08	10.27	1.6	10.4	42.67	3.6
Older subjects	71.2	16.02	11.27	2.4	12.8	43.87	4
Small <i>P</i>	<b>0</b>	0.307	0.76	<b>0.002</b>	<b>0.05</b>	0.918	0.892
Transformable AHBPPVs	56	15.42	8.25	1.88	9.81	40.13	3.52
Non-transformable AHBPPVs	61.2	15.68	13.29	2.12	13.39	46.41	4.09
Small <i>P</i>	0.375	0.307	0.101	0.333	0.346	0.563	0.547
Low positional risk	59.4	13.44	8.4	1.87	10.53	44.8	4.53
High positional risk	57.8	17.66	13.13	2.13	12.67	41.73	3.07
Small <i>P</i>	0.779	<b>0</b>	0.152	0.334	0.601	0.792	0.701
Short scarring time	56.2	14.8	3.87	1.93	11.3	38.67	4.26
Long scarring time	61	16.3	17.67	2.07	11.9	47.87	3.34
Small <i>P</i>	0.397	0.193	<b>0</b>	0.631	0.819	0.428	0.482
Low number of consultation	57.2	15	9.2	1.53	10.4	44.67	2.8
High number of consultation	60	16.1	12.33	2.47	12.8	41.87	4.8
Small <i>P</i>	0.623	0.307	0.348	<b>0</b>	0.556	0.81	<b>0.04</b>
Short time to consultation	57.6	15.73	11	1.57	3.73	39.73	4.27
Long time to consultation	59.6	15.37	10.53	2.43	19.47	46.8	3.33
Small <i>P</i>	0.326	0.636	0.89	<b>0.032</b>	<b>0</b>	0.543	0.603

not significant. However, we do note a significant difference between the number of consultations needed to relieve a young patient (1.6) and that needed to relieve older patients (2.4,  $P = 0.002$ ). This can perhaps be explained by the results of the following column since it seems that the young patients are treated more quickly (10.4 days) whereas the older patients are only treated after 12.8 days ( $P = 0.05$ ).

**3.2. Influence of the Factor “Transformability”.** We did not observe any significant differences immediately (number of consultations or acts) or at a later stage (VSS, DHI, VNS) between the group of apogeotropic BPPVs of the horizontal canal transformable (into BPPVs of the posterior canal or geotropic BPPVs of the horizontal canal) and the nontransformable group of apogeotropic BPPVs of the horizontal canal. Our two subgroups were similar and comparable in terms of age, time to consultation, and time to recovery. They had both followed the positional advice in an identical manner. All the small  $P$  values were greater than 0.1 for this criterion.

**3.3. Influence of the Factor “Positional Risk”.** Whether the patients completely followed the positional advice or not, their VSS, DHI, and VSS scores were unchanged some time after the initial episode. Our two sub-groups were comparable; all the small  $P$ -values were greater than 0.152 for this criterion.

**3.4. Influence of the Factor “Scarring Time”.** The time between the first maneuver and the control consultation at a later date does not influence the VSS, DHI, or VNS results either. The two sub-groups were comparable from the point

of view of the other criteria and the small  $P$ -values were all greater than 0.193.

**3.5. Influence of the Factor “Number of Consultations”.** The number of consultation is linked with the number of maneuvers. The study of this compounding factor reveals that the patients for whom clinical recovery was secured the most quickly are the ones who had the best prognosis of videoscopic recovery some time after the episode ( $P = 0.04$ ). The clinical or symptomatic recovery is link with the videoscopic recovery. But this does not have any impact on the symptomatic results or the patient’s impression 10.76 months after the episode ( $P = 0.81$ ). A videoscopic or clinical regaining of a patient immediately after the maneuvers does not mean that its symptomatic scores will be good.

**3.6. Influence of the Factor “Time to Treatment”.** The patients treated 3.73 days on average after the onset of the disease recovered more quickly than those treated 19.47 days after the onset of the disease ( $P = 0.032$ ). The number of consultations may be linked to this result. Nonetheless at a later stage, the two groups are doing equally well or poorly from the point of view of VSS, DHI, and VNS with small  $P$ -values greater than 0.5.

Our study concerning statistics and correlation with the Pearson test does not reveal any interesting results.

There is no correlation between the symptomatic results and the patients’ age ( $r = 0.129$ ), no correlation between the symptomatic results and the following of positional advice ( $r = -0.148$ ), and no correlation between the symptomatic results and videoscopic recovery ( $r = 0.110$ ).

Nor is there any correlation between the videoscopic results and the patients' age ( $r = 0.189$ ), the videoscopic results and the following of positional advice ( $r = -0.373$ ), and finally no correlation between age and the following of positional advice ( $r = -0.052$ ).

#### 4. Discussion

Etiologic diagnosis and lateralization of AHBPPV are difficult, still contentious, and disputed. The hypothesis of a cupulolithiasis [7] or a canalolithiasis located in the ductal side next to the cupule can be retained [8, 9]. According to some authors, sometimes a central etiology can be mentioned [10, 11]. In this study we only took in account the AHBPPV for which imaging and neurologic examination were normal. For an AHVPPB, the healthy side is the side where the apogeotropic nystagmus is the strongest [2, 9, 12], but it is also the side in which the vertigo felt by the patient is the most intense. It seems to these authors that the bow and lean test [13–15] is an important element but it needs a videoscopy. The nystagmus beats on the healthy side when the head is bent forward. It is reversed when the head is tilted back.

For this study we only selected AHVPPB for which arguments of lateralization matched. We aimed at corroborate or invalidate the efficiency of the assigned therapy. We preferred to exclude some AHVPPBs for which we were doubtful about the side affected. For that, we observed the diagnostic recommendations of the literature. Thus, when the apogeotropic nystagmus was too intense to enable a differentiation (11 cases), we asked the patient to tell us the side where vertigo was the strongest and in 5 cases this argument enabled us to lateralize the AHBPPV. In the 6 remaining cases we lateralized the BPPV thanks to the bow and lean test.

The treatment of geotropic BPPV of the horizontal canal was described before that of apogeotropic BPPV of the horizontal canal. The first articles dealing with this subject mention the possible effectiveness of therapeutic rotations in the horizontal canal plan [7, 16] for geotropic BPPVs of the horizontal canal. Unfortunately, these first references do not clearly indicate the rotation direction in these maneuvers. The concept of rotation in the opposite sense to the geotropic BPPV of the horizontal canal with at least  $270^\circ$  has become the accepted norm over the course of time [17] as well as postmaneuver recommendations along with, in some cases, extended decubitus on the side opposite the geotropic BPPV of the horizontal canal. The literature on this subject concerning apogeotropic BPPVs of the horizontal canal (scale of rotation, direction of rotation, and postmaneuver advice) is less clear and much debated. It is accepted that the transformation of an apogeotropic BPPV of the horizontal canal into a geotropic BPPV of the horizontal canal results from the displacement of the free-floating otoliths in the anterior section of the horizontal canal towards the posterior section [13]. Furthermore, this is theoretically, anatomically, and clinically conceivable. For some authors this transformation of an apogeotropic BPPV of the horizontal canal into a geotropic BPPV of the horizontal canal is a necessary

preliminary. They admit that, as geotropic BPPVs of the horizontal canal are easier to treat than apogeotropic BPPVs of the horizontal canal, transformable apogeotropic BPPVs of the horizontal canal will be easier to control [16, 18]. These different authors agree and suggest for apogeotropic BVVPs of the horizontal canal an approach maneuver that contradicts the one generally accepted as being effective in the treatment of geotropic BPPVs of the horizontal canal. Therapeutic rotation can be undertaken on the side opposite to the apogeotropic BPPV of the horizontal canal (anticlockwise/unaffected side for the ones on the right AHBPPV and clockwise/unaffected side for the ones on the left AHVPPB) with a scale of  $270^\circ$  at least and/or in some cases recommendations of extended sleep on the affected side. It is also possible to practice a repositioning maneuver to transform AHBPPV in GHBPPV [18, 19].

Our study has shown that the rotation direction in contradiction to the normal physiopathological direction and to the one recommended in these articles [7, 16] could be effective. Consequently, our study challenges the therapeutic rotation direction accepted for these apogeotropic BPPVs of the horizontal canal. It seems that this direction does not influence the results. In contrast to our expectations and the literature, we have shown that irrespective of whether a barbeque maneuver or a Guffoni maneuver is involved [18, 19], transformability did not in any way predict the sensitivity of apogeotropic BPPVs of the horizontal canal to these maneuvers, that this transformability did not permit more rapid control of apogeotropic BPPVs of the horizontal canal and that the videoscopic and symptomatic results of these apogeotropic BPPVs of the horizontal canal, whether transformable or not, were identical. Whether the AHBPPVs can be transformed into GHBPPVs or not, whether treated with an affected side barbeque maneuver or not, the results are similar, maybe with better results to the unaffected ear rotation. FIFE [7] with 6 AHBPPV, after one week and an affected ear barbeque  $360^\circ$  maneuver obtained 4 recoveries (66%). GUFONI [18] with 6 AHBPPV, after one week and an unaffected ear barbeque  $270^\circ$  maneuver obtain 6 recoveries (100%) and 4 transformations (66%). In our study with 30 AHBPPV after 4 weeks and an affected ear barbeque  $360^\circ$  maneuver, we obtained 22 recoveries (73%) and 13 transformations (43%).

Given the physiopathological hypotheses involving otolithic migration indicated in the apogeotropic BPPVs of the horizontal canal and given the accepted knowledge concerning BPPVs of the posterior canal and geotropic BPPVs of the horizontal canal, we rightly believe that a shift towards spontaneous healing of apogeotropic BPPVs of the horizontal canal is possible [8]. Furthermore, this has already been mentioned in conjunction with small series [17]. The simple fact of sleeping on the affected side over a prolonged period can lead to recovery [17]. We believe it would be interesting to conduct research within this framework into the potential noninfluence of the therapist if external factors could prove to be effective.

We have shown here that as the patient's age increases, the number of acts needed to achieve recovery rose significantly ( $P = 0.002$ ). We have shown also than an elevated number



of consultations (or acts or maneuvers) increase videoscopic score after 10.76 months. The more the AHBPPV needs maneuvers the more its videoscopy at a distance is distorted. We have demonstrated that neither the scarring time nor the following of positional advice modified the symptomatic results ( $P = 0.428$  and  $P = 0.792$ , resp.). Thus, in the case of apogeotropic BPPVs of the horizontal canal, rapid consultation (within a few days) involving videonystagmoscopy by a therapist significantly reduced the time required for short-term recovery particularly if the patient is young. Nonetheless, a few months later, the symptomatic and videoscopic status of all patients will be the same. It can, therefore, be stated that this treatment gives symptomatic comfort for a few days (3.73 days low average—19.47 days high average) to patients who will seemingly recover spontaneously.

In this study we also demonstrate that there is no correlation between the criteria for videoscopic and symptomatic recovery. This opens the door to a new concept for defining recovery from apogeotropic BPPVs of the horizontal canal. When it comes to assessing recovery, should we—as therapists—turn our attention to nystagmic criteria like we do for diagnosis [20]? Or should we focus on symptomatic criteria [5, 6], tools that have been validated in the past and seem to be more suited? We likewise demonstrate that there is no longer any correlation between the following of positional advice and symptomatic or videoscopic results. We could have imagined that age would influence these results but it does not.

## 5. Conclusion

Fifteen years after the first description of an apogeotropic BPPV of the horizontal canal, the therapeutic method is still a subject of debate. Based on 30 typical observations, we have demonstrated that the direction of rotation during the maneuvers is of no importance, that transformability was not a measure of positive results in the long term and that the effectiveness of the therapist regarding the short-term results was limited in our experience. Some external predetermined factors like age and time to consultation seem to be important. There does not seem to be any link between the symptomatic results, the videoscopic observations or the following of positional advice. Our next study will endeavor to compare our therapeutic results with complete abstention. The hypothesis of multiple etiologies, including some which fail to respond to physiotherapy in the treatment of apogeotropic BPPVs of the horizontal canal, should be mentioned.

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## Case Report

# About Nystagmus Transformation in a Case of Apogeotropic Lateral Semicircular Canal Benign Paroxysmal Positional Vertigo

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There are two forms of lateral semicircular canal benign paroxysmal positional vertigo: geotropic and apogeotropic. When the pathophysiological mechanism of the apogeotropic form is that of canalolithiasis, we can observe a transformation from an apogeotropic nystagmus into a geotropic one. Usually, this phenomenon happens simultaneously on both sides, thus enabling us to observe a right-beating paroxysmal positional nystagmus when the patient lies on the right side and a left-beating paroxysmal positional nystagmus on the left side. We describe a case in which the transformation occurred gradually, so that, after three head rotations from side to side in supine position, there was a right nystagmus beating toward the ground (geotropic) with the patient on the right side and a right nystagmus beating away from the ground (apogeotropic) on the left side. However, after further rotations we observed the nystagmus transformation also on the left side, with a geotropic nystagmus on both sides. The phenomenon of gradual transformation could happen because initially only a part of the debris moved from the anterior to the posterior aspect of the canal during head rotations.

## 1. Introduction

Benign Paroxysmal Positional Vertigo (BPPV) is the most frequently found type of vertigo in clinical practice. The posterior semicircular canal (PSC) is more frequently involved, the lateral semicircular canal (LSC) less frequently so, and the anterior semicircular canal (ASC) only rarely [1–3]. The LSC-BPPV is described in two forms [4, 5]: (a) the geotropic form, in which the direction of the fast phase of the nystagmus is right when the patient lies on the right side and left when the patient lies on the left side; (b) the apogeotropic form, with a right nystagmus when the patient lies on the left side and a left nystagmus when the patient lies on the right side. When the nystagmus is geotropic, the debris is located in the posterior aspect of the canal, and the pathophysiological mechanism is a canalolithiasis [5]. When the nystagmus is apogeotropic, the mechanism can be a canalolithiasis, with the debris within the anterior aspect of the canal, or a cupulolithiasis, with the debris attached to the cupula (on its canal or utricular wall) [6]. In some

cases, the apogeotropic form changes into the geotropic one. In this case the debris moves from the anterior aspect into the posterior aspect of the canal. Thus, in a patient who initially shows a left beating nystagmus when lies on the right side and a right beating nystagmus when lies on the left side, we later observe a right beating nystagmus when the patient lies on the right side and a left beating nystagmus when he/she lies on the left side. We have noted this transformation always at the same time on both sides; only in the patient described in this paper did we note the transformation earlier on one side and, after more head rotations in supine position, also on the other side. We will now describe the case and make a few conjectures as regards the pathophysiological mechanisms involved.

## 2. Material and Method

After collecting a detailed history the patient underwent a microscope otologic inspection and an audiometric and

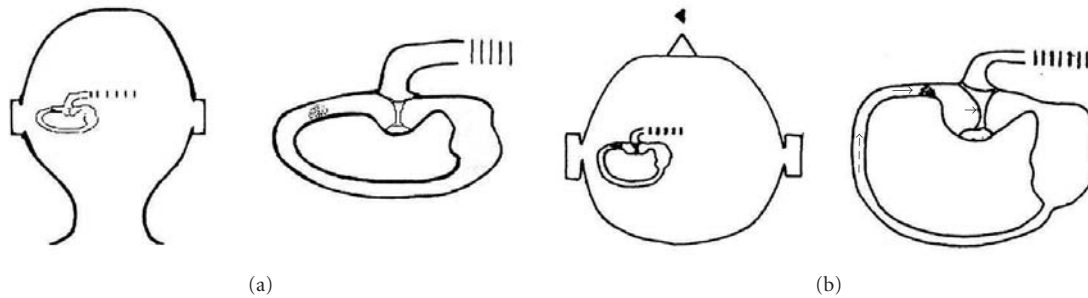


FIGURE 1: (a) Patient with a left LSC-BPPV of the apogeotropic form in sitting position (backward view): the debris is localized in the anterior arm of the canal and is free floating. (b) When the patient moves from the sitting to the supine position, the endolymphatic flow (dashed arrow) causes the debris to move towards the ampulla (thin arrow). Here, acting as a plunger, debris determines an ampullopetal deflection of the cupula (thick arrow), thus triggering a left-beating nystagmus (head arrow).

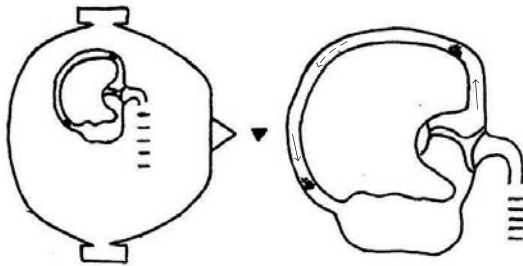


FIGURE 2: When the patient in supine position rolls his/her head by 180° from the left to the right side for the third time, pushed by the endolymphatic flow (dashed arrow), some of the debris moves into the posterior arm of the canal, while another part remains in the anterior arm, both moving in an ampulofugal way (thin arrows), thus triggering a right beating geotropic nystagmus (head arrow).

impedance testing; then we looked for spontaneous, gaze-evoked, rebound, and positional nystagmus, both with and without fixation (in the latter with infrared video cameras and nystagmus recording). We performed the Head Shaking Test (HST), the Head Impulse Test, and the caloric test (according to the Fitzgerald-Hallpike method) in order to study the canal paresis and directional preponderance. Lastly, we tested for the cervical Vestibular Evoked Myogenic Potentials (cVEMPs) and the Subjective Visual Vertical (SVV).

After the diagnosis, the therapy was a forced prolonged position in which the patient was asked to lie for eight–ten hours on the healthy side [7]. Informed consent was obtained from the patient.

### 3. Case Report

The patient was a 73-year-old male, who suffered from right Menière's disease for the past 10 years, with normal Computerized Tomography scan and Magnetic Resonance Imaging. In both 1996 and 2004, two infiltrations of transtympanic gentamicin were performed and a good control of the disease was obtained, so that the patient did not complain any more

of spontaneous vertigo lasting many hours, as typical of Menière's disease.

However, in December 2006 the patient reported positional and short-lasting vertigo triggered by moving his head from side to side in supine position. The audiogram showed a flat-type hearing loss with PTA to 80 dB on the right and a presbycusis with pure tone threshold, from 500 to 4000 Hz, of 15, 15, 30, 50, and 65 dB on the left. There was a caloric weakness in the right ear, with a canal paresis of about 50%, but both the Head Thrust Test and the HST were negative. The cVEMPs were normal on the left and absent on the right; the SVV was normal.

We studied the nystagmus with and without fixation using infrared video cameras. When the patient was moved from a sitting position to a supine one, a small, horizontal, left-beating nystagmus, lasting about two minutes, was observed. When the patient's head was rotated by 90° to the right, a more intense left-beating nystagmus (apogeotropic) appeared. When he moved his head by 180° to the left, a more intense right-beating (still apogeotropic) nystagmus was observed. In the following two rotations from side to side, we again observed an apogeotropic nystagmus on both sides albeit more intense with the head in the right-ear-down position.

In rotating the patient for the third time onto his right side, we noted a right-beating geotropic nystagmus thus obtaining a transformation from apogeotropic nystagmus into a geotropic one; but, in rotating the patient onto his left side, a right apogeotropic nystagmus was observed. The latter nystagmus was less intense than the previous one observed on the same (left) side. When we repeated the head rotations from side to side, we again observed a right beating geotropic nystagmus on the right side, and a right beating apogeotropic nystagmus on the left side, that became progressively less intense at each positioning. When moving the patient for the fifth time on his right side there was still a right nystagmus but rotating the patient on his left side, after a pause of three seconds, we observed a very violent left-beating geotropic nystagmus that was associated with intense vertigo and autonomic symptomatology. Now, because of a more intense geotropic nystagmus on the left side, we can

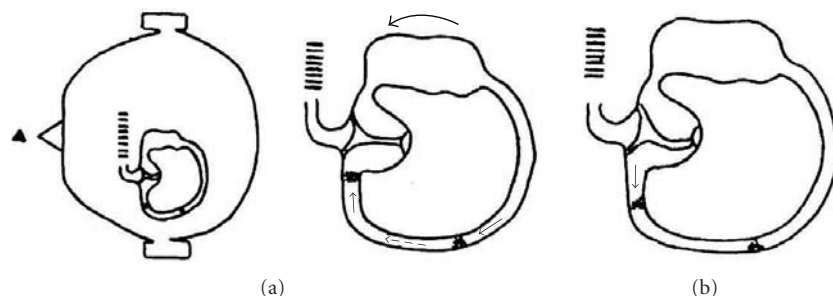


FIGURE 3: (a) When the patient in supine position rolls his/her head by 180° from the right side to the left side (outer arrow), the endolymphatic flow (dashed arrow) pushes the debris localized in the anterior arm of the canal towards the cupula and those in the posterior arm towards the lowermost part of the canal (thin arrows). (b) Therefore, when the head is still in this position, the debris in the anterior arm of the canal moves away from the ampulla due to gravity (thin arrow), provoking an ampullofugal flow and a right beating apogeotropic nystagmus (head arrow), while the debris in the posterior arm does not provoke any flow.

hypothesize a left LSC-BPPV, then on the opposite ear to that with Menière's disease.

Therefore, the transformation from the apogeotropic to the geotropic form happened gradually only after a lot of side-to-side head rotations and needed as long as forty minutes to occur.

The following night the patient stayed on his healthy (right) side for eight–ten hours (Forced Prolonged Position) [7], and on the next check-up LSC-BPPV was resolved because the nystagmus and symptomatology disappeared.

#### 4. Discussion

There are two types of LSC-BPPV: geotropic and apogeotropic [4–7]. Geotropic LSC-BPPV is more frequent, and is caused by a canalolithiasis [8]. Apogeotropic LSC-BPPV is less frequent and may be related to canalolithiasis, with debris in the anterior aspect of the lateral canal, or to cupulolithiasis [9] with debris attached to the cupula on the vestibular or canalar side. In both cases of apogeotropic LSC-BPPV (canalolithiasis or cupulolithiasis), an ampullofugal current happens when the patient lies on the affected side; when the patient lies on the healthy side, an ampullopetal current occurs.

How does one explain the transformation from apogeotropic to geotropic nystagmus in LSC-BPPV? In the canalolithiasis, if the debris moves from the anterior aspect to the posterior aspect of the canal, the apogeotropic form could become geotropic. In cupulolithiasis, transformation could be more difficult: for example, if the debris is on the canalar side of the cupula, it could become detached and, thus free to move in the canal, could transform the cupulolithiasis into canalolithiasis. If the debris is on the vestibular side of the ampullar receptor, the transformation could happen only if the debris detached from the cupula enters the vestibule and then goes into the posterior aspect of the LSC (it is very difficult that this phenomenon occurs: it is more probable that there is a resolution of the vertigo without any transformation). When the transformation happens, however, the apogeotropic nystagmus becomes

geotropic irrespective of patient's side positions (i.e. right or left side position).

Instead, in this particular case study, the transformation happened gradually, first on one side and then on the other.

We can conjecture that, in the beginning, the debris was inside the anterior aspect of the left lateral canal because of the left beating nystagmus that resulted from moving the patient from a seated to a supine position [10] (Figure 1), and because, by rotating the head by 180° from side to side, the nystagmus on the right side was stronger than that on the left side. Moreover, at the end of our study of this patient, we were to learn that the lateral canal involved really was the one on the left.

In rotating the patient's head in supine position from the left side to the right side for the third time, we observed a right beating geotropic nystagmus. This could be explained by the movement of the debris from the anterior aspect to the posterior aspect of the canal. But by moving the head to the left side, we did not observe a left beating geotropic nystagmus, but again a right beating nystagmus that was apogeotropic lying the patient on the left side. How do we explain this phenomenon? It is possible that during the rotation towards the right side only a part of the debris moved from the anterior to the posterior aspect of the canal, while another part remained in the anterior aspect (Figure 2). By moving the head towards the left side, the current pushed the debris localized in the anterior aspect towards the cupula and that in the posterior aspect towards the central part of the canal. Thus, with the patient remaining in this position, the debris in the anterior aspect fell towards the vestibule, provoking an ampullofugal current and an apogeotropic nystagmus, while the debris in the posterior aspect did not provoke any current (Figure 3). By moving the head again towards the right side, other debris moved from the anterior aspect to the posterior aspect, causing an ampullofugal current with a right beating geotropic nystagmus. In the end, all the debris was in the posterior part of the canal, so that, by moving the head towards the left side, the current was not able to move this heavier

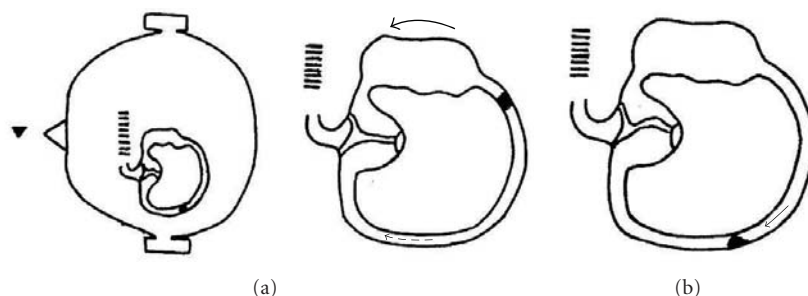


FIGURE 4: (a) In the end, when the patient in supine position moves his/her head from the right side to the left side (outer arrow), the endolymphatic flow (dashed arrow) is not able to move the debris in the posterior part of the canal towards the ampulla due to their weight. (b) It is only when the head is still that the debris suddenly falls, due to gravity, towards the cupula (thin arrow), provoking a violent left beating geotropic nystagmus (head arrow).

than endolymph material towards the ampulla. After a brief latency, the debris fell towards the cupula only when the head was motionless, due to gravity, provoking a very violent left geotropic nystagmus (Figure 4).

## 5. Conclusion

Some cases of apogeotropic LSC-BPPV are transformed into geotropic form, and others resolve directly. Moreover, usually the transformation is observed on both sides when the patient in supine position rotates his/her head from side to side. In this case study, the transformation occurred gradually: first on the right side we observed a right beating geotropic nystagmus but on the left side we again observed a right beating apogeotropic nystagmus; then, after other head rotations, we observed a geotropic nystagmus on both sides (with a left beating geotropic nystagmus even on the left side). In the end, the clinical picture was typical of geotropic form LSC-BPPV.

What can this case teach us? To explain the nystagmus that we observe during the examination of the vertiginous patients, it is necessary to consider the position of the patient, the plane and the direction of the head's movements, the current provoked by the movement, and the action of gravity on the debris when the head is still. We can try to explain the nystagmus observed only if we keep in mind all of these factors.

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## Review Article

# Diagnosis of Single- or Multiple-Canal Benign Paroxysmal Positional Vertigo according to the Type of Nystagmus

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Benign paroxysmal positional vertigo (BPPV) is a common peripheral vestibular disorder encountered in primary care and specialist otolaryngology and neurology clinics. It is associated with a characteristic paroxysmal positional nystagmus, which can be elicited with specific diagnostic positional maneuvers, such as the Dix-Hallpike test and the supine roll test. Current clinical research focused on diagnosing and treating various types of BPPV, according to the semicircular canal involved and according to the implicated pathogenetic mechanism. Cases of multiple-canal BPPV have been specifically investigated because until recently these were resistant to treatment with standard canalith repositioning procedures. Probably, the most significant factor in diagnosis of the type of BPPV is observation of the provoked nystagmus, during the diagnostic positional maneuvers. We describe in detail the various types of nystagmus, according to the canals involved, which are the keypoint to accurate diagnosis.

## 1. Introduction

Of all the inner ear disorders that can cause dizziness or vertigo, benign paroxysmal positional vertigo (BPPV) is by far the most common [1]. Additionally, it is a condition that in most instances may be easily diagnosed and treated, with a simple office-based procedure [2]. Since the initial description by Bárány in 1921 [3], there have been major advances in the understanding of this common condition. Recently, modern clinical research focused on diagnosing and treating various types of BPPV, according to the semicircular canal involved and according to the implicated pathogenetic mechanism. Multiple-canal BPPV has been specifically investigated, as the main source of various atypical forms of the disease, which until now were resistant to treatment with standard canalith repositioning procedures (CRPs) [4, 5]. The purpose of this paper is to present the data regarding the various types of nystagmus produced during the diagnostic maneuvers of BPPV, which in conjunction with the patient's history and symptoms, will help in obtaining accurate diagnosis and appropriate treatment. In all subsequent discussions,

the various types of nystagmus will be described according to their fast phase, relative to the patient's perspective (e.g., as horizontal nystagmus with a fast phase beating towards the patient's right ear is termed rightward horizontal nystagmus and a rightward torsional nystagmus, which is beating towards the patient's right ear, is a counterclockwise nystagmus, as seen by the observer).

## 2. Unilateral Posterior Canal BPPV

This is the most common type of BPPV, accounting for up to 90% of the patients [6]. The Dix-Hallpike provoking maneuver is used to diagnose the disease by moving the patient rapidly from a sitting position to a position of head hanging with each ear alternately undermost. Posterior semicircular canal involvement is proved from the type of the visually observed paroxysmal positioning nystagmus, which is beating towards the undermost and affected ear, with a torsional component clockwise when following leftward movement, or counterclockwise, when following rightward movement



[7]. Typically an upbeat nystagmus component is superimposed, resulting in a mixed torsional-vertical eye movement. Intense vertigo in conjunction with this pattern of nystagmus and the additional characteristics of a short latency, limited duration, intensity characterized by crescendo and decrescendo element, reversal on returning to the upright position, and fatigability on repetitive provocation may easily establish the diagnosis of posterior canal BPPV.

Canalolithiasis is the implicated pathogenetic mechanism for this disorder, characterized by the presence of free floating debris within the posterior semicircular canal, detached from the otoconial layer by degeneration or head trauma [8]. The otoconia gravitates into the posterior canal, where it forms a plug floating in its nonampullary branch. In the provoking Dix-Hallpike position the endolymph pulls on the cupula, because the free-floating otoconia falls under the influence of gravity. In the vertical canals, ampullofugal deflection produces an excitatory response. This would cause an abrupt onset of vertigo and the typical nystagmus described previously. Nystagmus latency is explained by inertia of the clot. The cupula deflection ends when the clot reaches its lowest position and accounts for the limited duration of the nystagmus. Fatigue is due to dispersion of the clot particles and reactivation after bedrest is caused by renewed clot formation.

An alternative pathogenetic theory, the cupulolithiasis of the posterior canal, may account for a small rate of cases with posterior canal BPPV [7, 8]. According to this, otoconia with a specific gravity greater than endolymph from a degenerating utricular macula settle on the cupula of the posterior canal, rendering it sensitive to gravity. Certain head movements may then produce inappropriate endolymph-cupula displacement, causing nystagmus and vertigo, which in this case is of longer duration. The latency before the onset of nystagmus reflects the inertia of the otoconial mass and the cupula, and the fatigability is presumably due to dispersal of the debris attached to the cupula or even to central vestibular adaptation.

The previously described profile of nystagmus correlates with the known neuromuscular pathways that arise from stimulation of the posterior canal ampullary nerves in animal models and humans [9, 10]. It should be noticed that the character of nystagmus changes with the direction of gaze, which is explained by contraction of the ipsilateral superior oblique and contralateral inferior rectus, following the stimulation of the posterior canal. When the patient lies in the lateral head hanging position, if he looks towards the uppermost unaffected ear, the axes of these two extraocular muscles nearly coincide, resulting in movement of the eyes in a vertical plane with predominance of the vertical component of the nystagmus. When looking towards the lowermost involved ear, the axes of these two muscles are nearly at right angles with the direction of the gaze, and their contraction results in apogeotropic rolling of the upper pole of the eye (slow phase) and predominance of the torsional component of the nystagmus with geotropic fast phase [11]. The Dix-Hallpike maneuver is usually positive only when performed with the involved ear undermost and negative on the contralateral side, permitting thus easy localization of the side of

the lesion (Figure 1). It should be also noticed, that posterior canal paroxysmal positional nystagmus is dissociated, with the torsional component being more evident in the ipsilateral eye, and the vertical upbeat component more evident in the contralateral eye, which can be explained by different angle of insertion of the oblique and rectus muscles [12, 13]. In Table 1, the various types of BPPV nystagmus are described, according to the involved semicircular canal and the side of involvement.

### 3. Unilateral Horizontal Canal BPPV

BPPV originating from stimulation of the horizontal semicircular canal is the second most common type of BPPV, accounting for approximately 5–15% of the patients [6, 14–16] but its frequency has been occasionally reported up to 30% [17]. The patient can get up or lie down, bend or straighten up with minimal complaints, but turning the head to either side in the supine position provokes intense vertigo, and a purely horizontal paroxysmal positioning nystagmus. Vertigo may be more intense than in posterior canal involvement and is usually associated with severe autonomic symptoms. Two major types of horizontal canal BPPV may be distinguished, according to the pathogenetic mechanism of canalolithiasis or cupulolithiasis. Canalolithiasis may manifest as BPPV with geotropic paroxysmal nystagmus, and less frequently with apogeotropic nystagmus, when the otoliths are located in the short arm of the horizontal semicircular canal, near the ampulla. Cupulolithiasis manifests as apogeotropic persistent nystagmus, commonly with absence of latency during the supine roll test.

**3.1. Geotropic Horizontal Canal BPPV.** This is the most common type of horizontal canal involvement, accounting for approximately 2/3 of the cases [2, 18]. The canalolithiasis theory can also explain this BPPV variant. Degenerative debris enter the nonampullary side of the pathological horizontal canal when the patient lies supine (Figure 2(a)). Diagnosis is made by the supine roll test, turning the head from the supine to either lateral position. When rotating the head to the pathological side (Figure 2(b)), gravity and the angular head acceleration make the mass descend in the canal towards the ampulla. The movement of the clot continues until the deepest position is reached and provokes an ampullopetal deviation of the cupula, resulting in a burst of nystagmus towards the ground. When maintaining the head rotation to the pathological side, a burst of nystagmus with opposite fast phase (away from the ground) can be seen. This may be attributed to short-term adaptation of the vestibulo-ocular reflex [19] or to an inversion of the direction of clot movement, due to a spontaneous reflux of endolymph between debris in the canal and membranous walls, facilitated by the elastic forces of the cupula. Another possibility is mixed canalolithiasis-cupulolithiasis, which may initially manifest as intense paroxysmal geotropical nystagmus owed to canalolithiasis, superimposed over the opposite nystagmus of cupulolithiasis, followed by the apogeotropical persistent nystagmus of cupulolithiasis. The same type of nystagmus

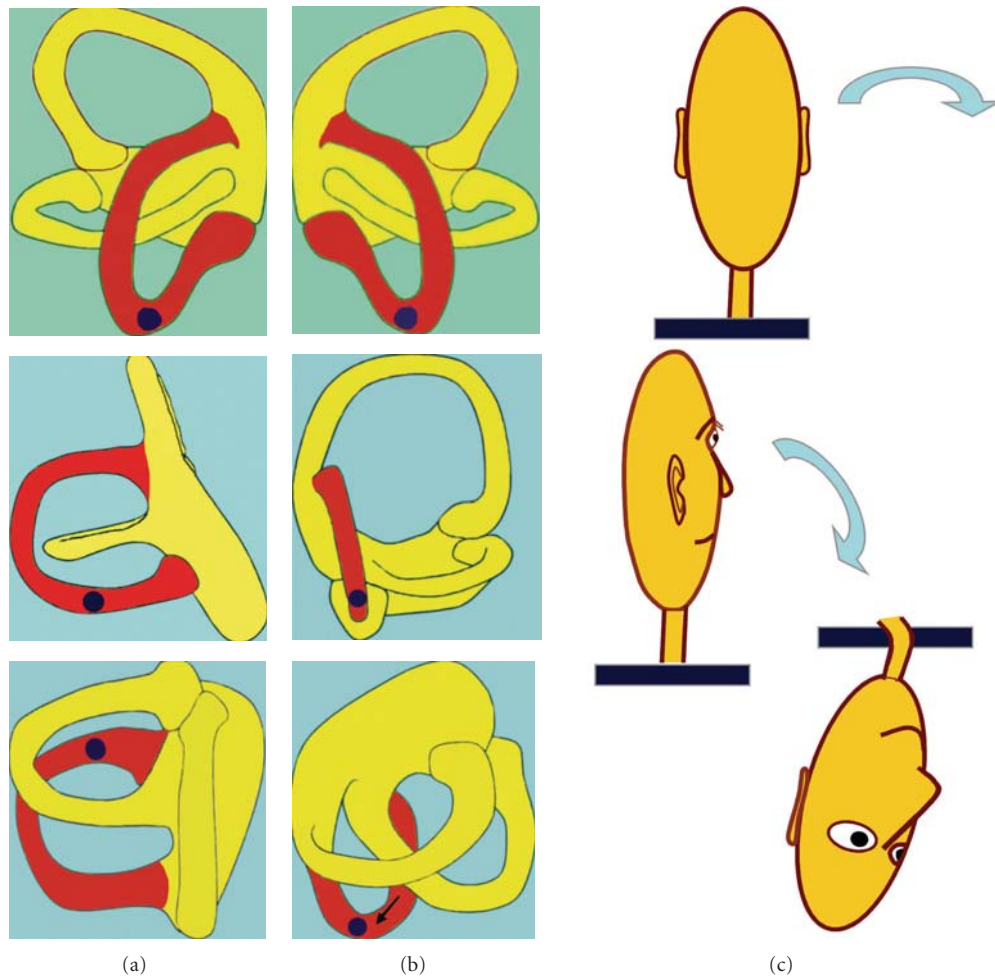


FIGURE 1: Stimulation of the posterior semicircular canal during the right Dix-Hallpike maneuver (c). On (a,b), the semicircular canals of the left and right ears, respectively, are shown. When otoconia is present in the ipsilateral (right) posterior canal (b), the maneuver causes its movement along the lumen of the canal, inducing BPPV. When otoconia is present in the contralateral (left) posterior canal (a), the maneuver does not cause any movement of the otoconia, because the head rotates orthogonally to the plane of the involved canal and the maneuver is negative.

can also be obtained by returning the head to the original position. When the head is rotated to the healthy side (Figure 2(c)), the mass is displaced further towards the nonampullary end of the canal with an ampullofugal displacement of the cupula, resulting in a nystagmus of lower intensity, beating towards the ground. Latency is usually shorter in horizontal canal BPPV. To summarize, horizontal canal BPPV owed to canalolithiasis manifests as bilateral geotropic horizontal nystagmus, which is more pronounced in the pathological side. This type of nystagmus is characterized by a short latency, a very sudden onset, and a longer duration as compared with the paroxysmal nystagmus of the posterior canal.

**3.2. Apogeotropic Horizontal Canal BPPV.** This BPPV variant may be caused by either cupulolithiasis, which manifests as apogeotropic persistent horizontal nystagmus [20], or, less frequently, by canalolithiasis, when the otoliths are located

in the short arm of the horizontal semicircular canal, near the ampulla [18]. Cupulolithiasis (Figure 3(a)) is thought to play a greater role in horizontal canal BPPV than in the posterior canal variant and accounts for approximately 1/3 of the cases [14]. As otoconia is directly adherent to the cupula, the vertigo is intense and persists while the head is in the provocative position. When the patient's head is turned toward the affected side (Figure 3(b)), the cupula will undergo an ampullofugal (inhibitory) deflection causing an apogeotropic nystagmus. Turning the head to the opposite side (Figure 3(c)) will result in ampullopetal (stimulatory) deflection, manifesting as a stronger apogeotropic nystagmus. To summarize, horizontal canal BPPV owed to cupulolithiasis manifests as bilateral apogeotropic horizontal nystagmus, which is more pronounced on the healthy side. This is explained by Ewald's second law [21], according to which excitation of the horizontal canal is a more potent stimulus than inhibition. In several cases, instead of cupulolithiasis, canalolithiasis of the short arm of the horizontal semicircular

TABLE 1: Diagnosis of the involved semicircular canal and the side of involvement, according to the appropriate diagnostic maneuver. SC: semicircular canal; R: right; L: left; P: posterior; A: anterior; H: horizontal; BPPV: benign paroxysmal positional vertigo.

(a) Vertical SC canals				
Involved SC canal		Diagnostic maneuver	Paroxysmal positioning nystagmus	
			Vertical	Torsional
P-BPPV R		Dix-Hallpike R (+)	Upbeating	Counterclockwise
		Dix-Hallpike L (−)	No nystagmus	
P-BPPV L		Dix-Hallpike R (−)	No nystagmus	Clockwise
		Dix-Hallpike L (+)	Upbeating	
A-BPPV R		Dix-Hallpike R (+)	Downbeating	Counterclockwise
		Dix-Hallpike L (+)	Downbeating	Counterclockwise
A-BPPV L		Dix-Hallpike R (+)	Downbeating	clockwise
		Dix-Hallpike L (+)	Downbeating	Clockwise
(b) Horizontal SC canals				
		Direction of nystagmus	Intensity of nystagmus	Pathogenetic mechanism
H-BPPV R	Supine roll test R (+)	Geotropic	More intense	Canalolithiasis
	Supine roll test L (+)	Geotropic	Less intense	
H-BPPV R	Supine roll test R (+)	Apogeotropic	Less intense	Cupulolithiasis or canalolithiasis of the short arm of the horizontal SC
	Supine roll test L (+)	Apogeotropic	More intense	
H-BPPV L	Supine roll test R (+)	Geotropic	Less intense	Canalolithiasis
	Supine roll test L (+)	Geotropic	More intense	
H-BPPV L	Supine roll test R (+)	Apogeotropic	More intense	Cupulolithiasis or canalolithiasis of the short arm of the horizontal
	Supine roll test L (+)	Apogeotropic	Less intense	

canal near the ampulla may occur [18], presenting with similar nystagmus (bilateral apogeotropic) as in the cupulolithiasis cases (Figure 4).

#### 4. Unilateral Anterior Canal BPPV

Anterior canal BPPV is quite rare and its incidence has been reported to range from 1-2% to 15% [6, 22]. It has been found that anterior canal BPPV produces bilaterally positive Dix-Hallpike maneuvers [23]. During a contralateral Dix-Hallpike maneuver (Figure 5), the head rotates in the plane of the affected anterior canal whereas during an ipsilesional Dix-Hallpike maneuver the head rotates orthogonally to the plane of the anterior canal (Figure 6). On both instances, the maneuver will be positive, due to the almost vertical orientation of the ampullary segment of the anterior canal. During the contralateral Dix-Hallpike test, the affected anterior canal is stimulated due to the movement of endolymph that takes place in its rotation plane. During the ipsilesional Dix-Hallpike test, the ampullary segment of the canal will also point downwards at about 40° off vertical. Consequently, displacement of otoconia in the involved anterior canal is induced and the test will be positive as well, although the provoked pressure against the cupula and the corresponding symptoms are expected to be less pronounced.

In anterior canal BPPV, the observed nystagmus when the patient is moved into the Dix-Hallpike position is mixed, with the direction of the fast phase being downbeating

and torsional [24]. When the direction of the torsional element of nystagmus is geotropic (superior pole of the eye moving toward the downside ear: counterclockwise on the right maneuver and clockwise on the left), excitation of the anterior canal of the downside ear may be inferred because debris in the ipsilateral affected anterior canal shifts to the most dependent position within the long arm of the canal, producing movement of the endolymph and excitation of the hair cells. This results in a torsional geotropic (right counterclockwise and left clockwise) and downbeating nystagmus. When the nystagmus is downbeating and torsional, but the fast phase of the torsional component is apogeotropic (superior pole of the eye beating toward the upside ear: clockwise on the right maneuver and counterclockwise on the left), it may be concluded that the affected anterior canal is in the upper ear. The differentiation between anterior and posterior BPPV should be based on the direction of the vertical component of the nystagmus. If the nystagmus is downbeating, the anterior canal of either ear may be affected, and conversely, if the nystagmus is upbeating, involvement of the posterior canal of the downside ear may be inferred.

Additionally, it should be noticed that the torsional nystagmic component is smaller for the anterior than the posterior canal nystagmus, because the anterior canals are placed nearer to the sagittal plane, in comparison with the posterior canals [23]. Accordingly, there is an upwards bias in vertical slow phase eye velocity, and more downbeat than torsional nystagmus is expected from anterior canal BPPV



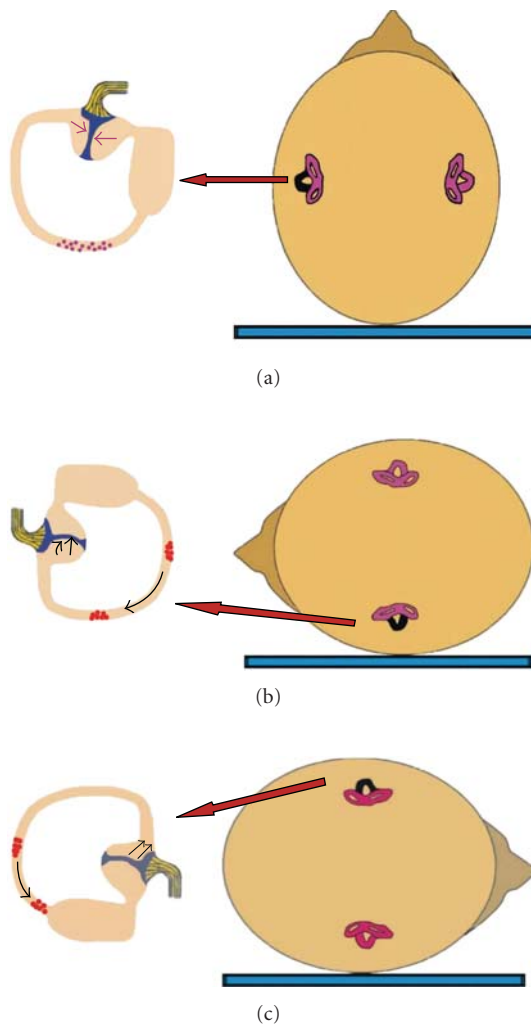


FIGURE 2: Mechanism of canalolithiasis of BPPV of the horizontal semicircular canal, when the left ear is affected (the involved left horizontal canal is colored black). (a) Patient in supine position with debris in the posterior part of the left horizontal canal. (b) When rotating the head towards the affected side, particles move towards the ampulla, producing an ampullopetal flow and triggering intense geotropic horizontal nystagmus. (c) When rotating the head towards the healthy side, particles fall in the opposite direction, causing an ampullofugal flow and triggering nystagmus beating again towards the ground, but less intense.

and more torsional than upbeat nystagmus in posterior canal BPPV. It has been observed in several instances, that the torsional component may be completely absent in anterior canal BPPV, and the disease may manifest as pure downbeating nystagmus, mimicking a central nervous system disorder. In this case, localization of the side of the lesion based on the produced nystagmus is not possible. Finally, it should be mentioned that occasionally, downbeating nystagmus may be seen during CRPs, caused by inappropriate (centripetal) movement of otoconia, indicating ineffective CRP, needing a repeat [18].

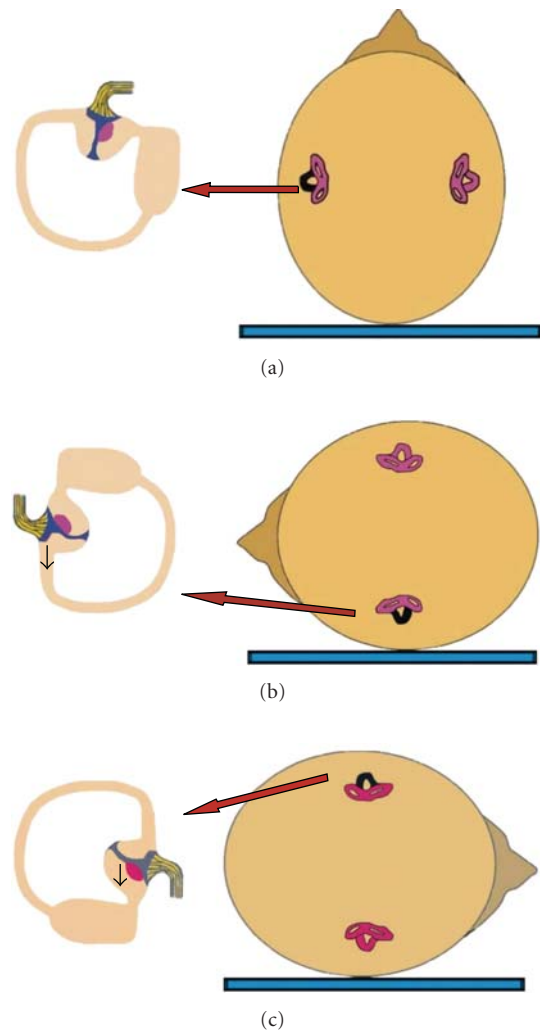


FIGURE 3: Mechanism of cupulolithiasis of BPPV of the horizontal semicircular canal, when the left ear is affected (the involved left horizontal canal is colored black). (a) Patient in supine position with debris adherent to the cupula of the left horizontal canal. (b) When rotating the head towards the affected side, the cupula will undergo an ampullofugal (inhibitory) deflection, triggering an apogeotropic horizontal nystagmus. (c) When rotating the head towards the healthy side, the cupula will undergo an ampullopetal (stimulatory) deflection, triggering a more intense apogeotropic nystagmus.

## 5. Multiple-Canal BPPV

Multiple-canal BPPV includes either involvement of the same canal on both sides or simultaneous involvement of different canals on the same or on both sides. It should be noticed that traumatic origin is quite common in multiple-canal BPPV, as previously reported [1, 4, 25, 26]. We should particularly think of and search for multiple-canal BPPV versus single canal BPPV when the patient has suffered head trauma. The specific types of multiple-canal involvement are further discussed.

## 6. Bilateral Posterior Canal BPPV

Bilateral posterior canal involvement is presumed when Dix-Hallpike maneuver is positive on both sides. However, care should be taken to avoid the erroneous diagnosis of pseudobilateral posterior canal BPPV as true bilateral BPPV [27]. The entity of unilateral mimicking bilateral BPPV was first described by Steddin and Brandt. According to these authors, inappropriate head positioning during testing of the unaffected ear causes displacement of the affected posterior canal from its perpendicular position. This makes the otolith debris move gravitationally towards the cupula, causing thus transient cupulolithiasis and evoking an inhibitory nystagmus. This nystagmus is directed towards the lower unaffected ear and this situation may be erroneously diagnosed as bilateral posterior canal BPPV. The inhibitory nystagmus usually has a lower amplitude and frequency than the excitatory nystagmus of the affected ear, and patients report less symptoms when the unaffected ear is tested. Additionally, the nystagmus during testing the noninvolved side may have a downbeating component and a longer duration [28].

Differential diagnosis between true bilateral and pseudobilateral posterior canal BPPV may be obtained based on the following [28, 29].

- (i) The presence of asymmetric nystagmus and symptoms of different intensity between right and left Dix-Hallpike maneuvers should arouse the suspicion of pseudobilateral posterior BPPV. The side with more intense nystagmus and symptoms may probably be the affected side.
- (ii) Performance of a head-down test, extending the head of the patient directly backward from the sitting to the supine straight head hanging position, might be helpful. During this test, both posterior canals get irritated, resulting in the appearance of nystagmus. This nystagmus has only a vertical upbeating component, because the torsional components, having opposite directions, are cancelled. True bilateral BPPV may be concluded in this case, whereas in case that the nystagmus retains its torsional component, pseudobilateral BPPV is probable. The true side of the disease may be found, observing the direction of this component, which beats clockwise on left posterior canal BPPV and Counterclockwise on right posterior canal involvement.
- (iii) The criterion of responsiveness to treatment is quite helpful. Successful treatment of the patient after performing the appropriate CRP on the side with more intense manifestations is proof of previously pseudobilateral BPPV [27]. When it is necessary to repeat the CRP contralaterally to obtain remission of the symptoms, this may be proof of bilateral posterior BPPV.

## 7. Bilateral Horizontal Canal BPPV

Bilateral horizontal canal BPPV is quite difficult to diagnose. The critical point in this case is that during the supine roll

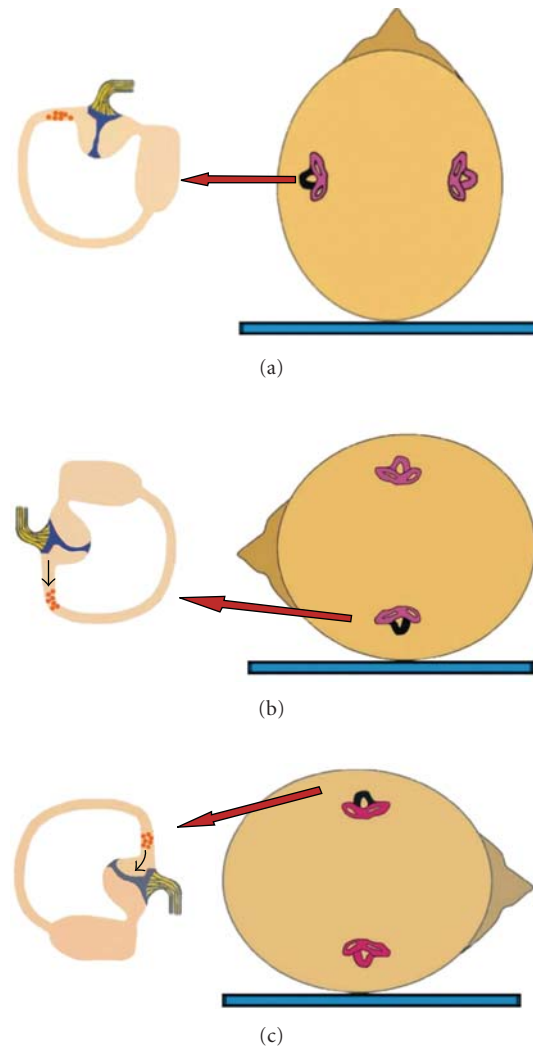


FIGURE 4: When canalolithiasis of the short arm of the horizontal semicircular canal near the ampulla occurs (left ear), a similar nystagmus (bilateral apogeotropic) as in the cupulolithiasis cases is observed during the supine roll test (the involved left horizontal canal is colored black). (a) Patient in supine position with debris in the short arm of the left horizontal canal. (b) When rotating the head towards the affected side, the particles move away from the cupula, which will undergo an ampullofugal (inhibitory) deflection, triggering an apogeotropic horizontal nystagmus. (c) When rotating the head towards the healthy side, the particles move towards the cupula, which will undergo an ampullopetal (stimulatory) deflection, triggering a more intense apogeotropic nystagmus.

test, unilateral horizontal canal BPPV elicits horizontal nystagmus on both sides, either geotropic (canalolithiasis mechanism) or apogeotropic (cupulolithiasis and canalolithiasis mechanism) [2].

**7.1. Geotropic Bilateral Horizontal Canal BPPV.** In a theoretical case of bilateral horizontal BPPV with geotropic nystagmus, supine roll test on either side would result in excitation of the horizontal canal of the lowermost ear, due to

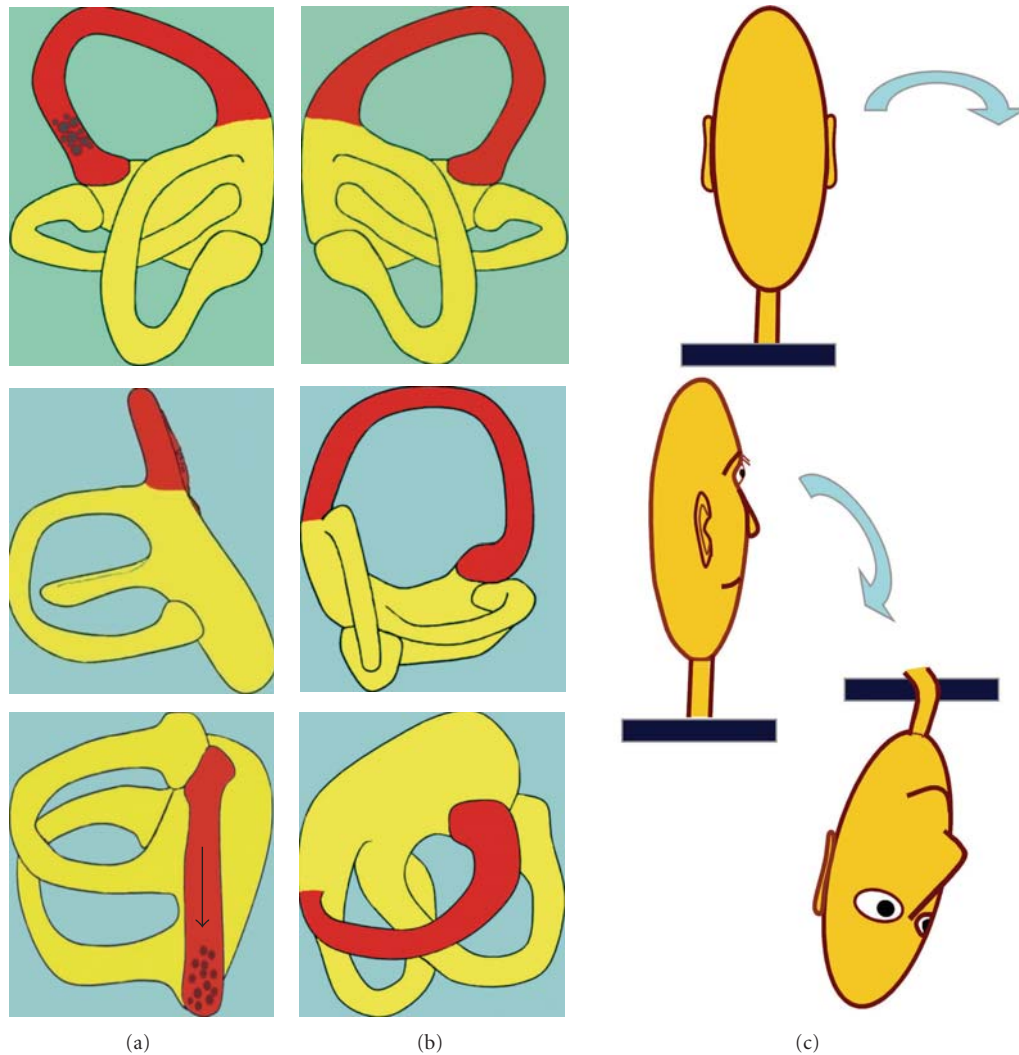


FIGURE 5: Stimulation of the left anterior semicircular canal during the right Dix-Hallpike maneuver (c). On (a,b), the semicircular canals of the left and right ears, respectively, are shown. When otoconia is present in the contralateral (left) anterior canal (a), the affected anterior canal is stimulated due to the movement of endolymph that takes place in its rotation plane, inducing BPPV.

ampullopetal endolymph flow and at the same time inhibition of the horizontal canal of the uppermost ear, due to ampulofugal endolymph flow (Figure 7). Vectorial summation would result in an intense, symmetric geotropic nystagmus.

**7.2. Apogeotropic Bilateral Horizontal Canal BPPV.** In a hypothetical case of bilateral horizontal BPPV with apogeotropic nystagmus, supine roll test on either side would result in inhibition of the horizontal canal of the lowermost ear, due to ampulofugal cupular movement, triggering an apogeotropic horizontal nystagmus. At the same time, excitation of the horizontal canal of the uppermost ear, due to ampullopetal movement of the cupula, would occur (Figure 8). Vectorial summation would result in an intense, more or less symmetric, apogeotropic nystagmus. It may be assumed that the nystagmus would be more intense in apogeotropic bilateral horizontal canal BPPV because of a dual pathogenetic mechanism: inhibition of the horizontal

canal of the lower ear and, concurrently, excitation of the horizontal canal of the upper ear. In comparison, in cases with unilateral involvement of the horizontal canal, only one mechanism contributes to the produced apogeotropic nystagmus: either inhibition of the affected ear on turning towards its direction or excitation of the affected ear on turning towards the healthy ear. The same mechanism is valid in cases with canalolithiasis of the ampullary arm of the horizontal canal.

It has been reported that 10% of the cases with unilateral horizontal BPPV may present with symmetrical nystagmus [30]. In this case it is difficult to detect the side of the lesion. To accomplish this, study of the pseudospontaneous nystagmus with the head pitch test has been proposed [31]. It has been reported that patients with horizontal BPPV may exhibit a spontaneous horizontal nystagmus while in the sitting position. This represents probably a pseudospontaneous nystagmus because it is strongly influenced by head

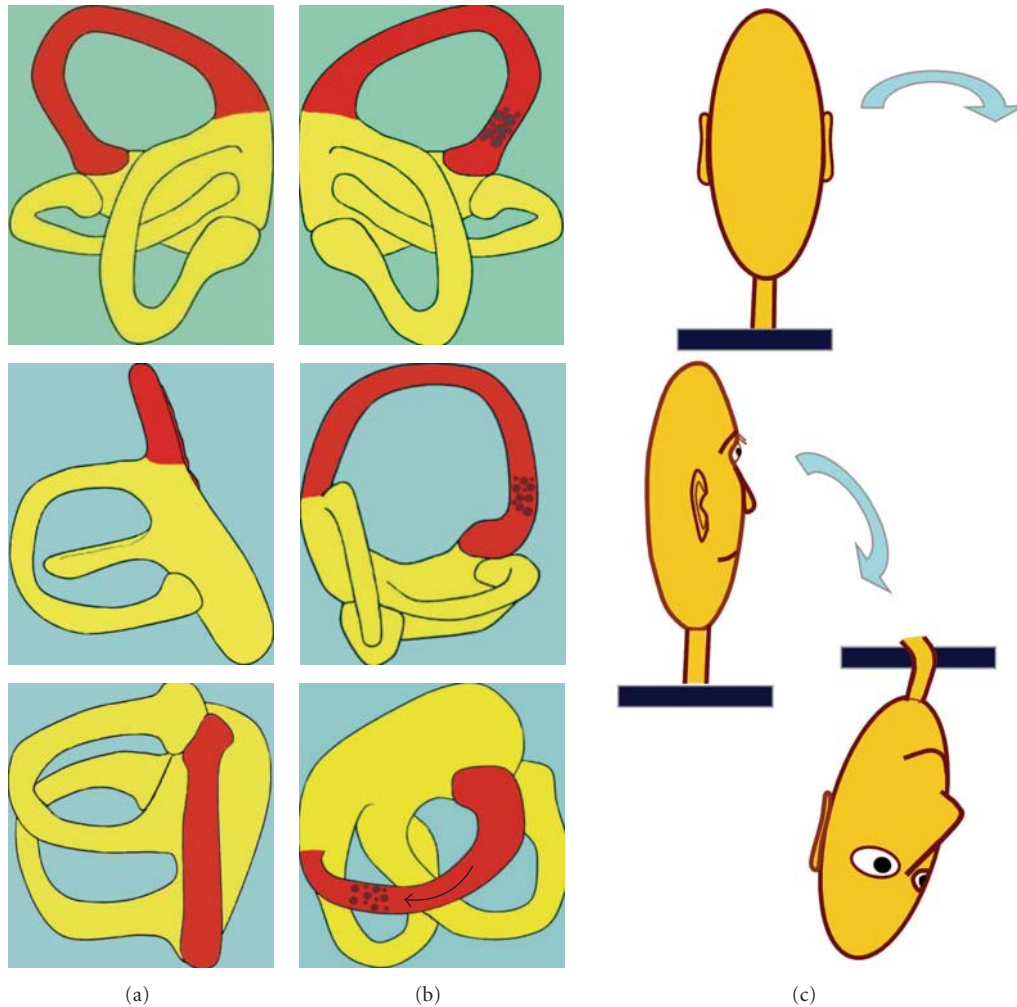


FIGURE 6: Stimulation of the right anterior semicircular canal during the right Dix-Hallpike maneuver (c). On (a,b), the semicircular canals of the left and right ears, respectively, are shown. When otoconia is present in the ipsilateral (right) anterior canal (b), the head rotates orthogonally to the plane of the affected anterior canal. However, due to the almost vertical orientation of the its ampullary segment, displacement of otoconia is induced as well and the test will be positive, although the provoked pressure against the cupula and the corresponding symptoms are expected to be less pronounced.

position and movements. When spontaneous nystagmus is absent, it is occasionally possible to evoke it with mild horizontal movements of the head. Pathogenesis of the pseudospontaneous nystagmus may be explained by the angle of  $30^\circ$  which exists between the horizontal plane and the horizontal semicircular canal, when the head is erect. Any head movements, even if minimal, would cause free debris floating inside the canal to move away from the ampulla, provoking a nystagmus with fast phase towards the unaffected ear. In case of cupulolithiasis, the attached otoconial mass would cause movement of the cupula in the opposite direction, triggering a reverse nystagmus (Figure 9(a)).

By bending the head  $30^\circ$  forward, spontaneous nystagmus should disappear because the horizontal canal assumes a true horizontal position, and either free debris or the heavy cupula is not further influenced by the gravity vector. Furthermore, by bending the head forward to about  $60^\circ$  (Figure 9(b)), gravity causes ampullopetal movement of the

debris, resulting in a nystagmus fast phase towards the affected ear, which is the opposite direction from that observed with the head erect. If the otoconia is attached to the cupula, the cupular deflection will be in the opposite direction, triggering an opposite nystagmus (towards the unaffected ear). Finally, backward bending of the head (Figure 9(c)) will cause an increase in spontaneous nystagmus, because the canal will be approximately in the vertical position, similar to the position at which we locate the patient to perform calorics. This type of testing is called "bow and lean test" [21], or more appropriately head pitch test [31], and has been used to determine the affected side, once we know from the previous maneuvers that the positional horizontal nystagmus is of the geotropic or the apogeotropic type.

Another method is to examine the appearance of positional nystagmus by performing the head down test [32, 33], quickly bringing the patient from the sitting to the supine position, in the sagittal plane (Figure 10). Frequently, a mild



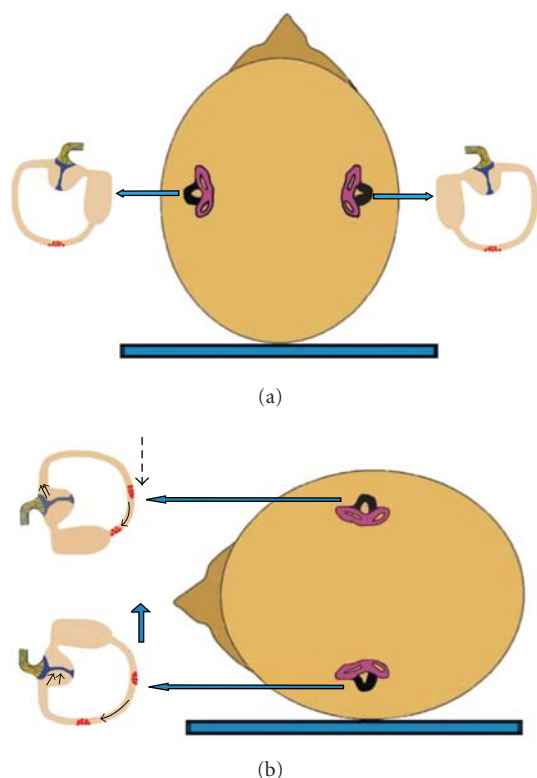


FIGURE 7: Mechanism of canalolithiasis of bilateral geotropic horizontal canal BPPV (the involved horizontal canal sare colored black). (a) Patient in supine position with debris in the posterior part of both horizontal canals. (b) Supine roll test on either side would result in excitation of the horizontal canal of the lowermost ear, due to ampullopetal endolymph flow and at the same time inhibition of the horizontal canal of the uppermost ear, due to ampullofugal endolymph flow. Vectorial summation would result in an intense, symmetric geotropic nystagmus.

horizontal nystagmus appears, attributed to the movement of debris in the horizontal canal, when canalolithiasis is the underlying pathology. This movement causes the debris to move ampullofugally, resulting in nystagmus towards the unaffected ear. In cases of cupulolithiasis, otoconial debris attached to the cupula causes ampullopetal movement, resulting in nystagmus directed towards the affected ear.

In cases of bilateral symmetrical nystagmus, owed to bilateral involvement, neither pseudospontaneous nystagmus nor nystagmus during the head down test should be present most of the time. However, if some type of nystagmus could be observed, its direction should not be stable, but changing according to the prevailing movement of otoconia in each horizontal canal. It should be further noticed that the criterion of symmetry of the nystagmus to diagnose bilateral horizontal canal BPPV is not a solid one, because asymmetric involvement of the two horizontal canals may occur as well. Combination of canalolithiasis-cupulolithiasis, either on the same or on different horizontal canals, would complicate the matter further. It has been reported though, that reversal of the geotropic nystagmus to apogeotropic, while the subject remains in the same lateral position during the supine roll

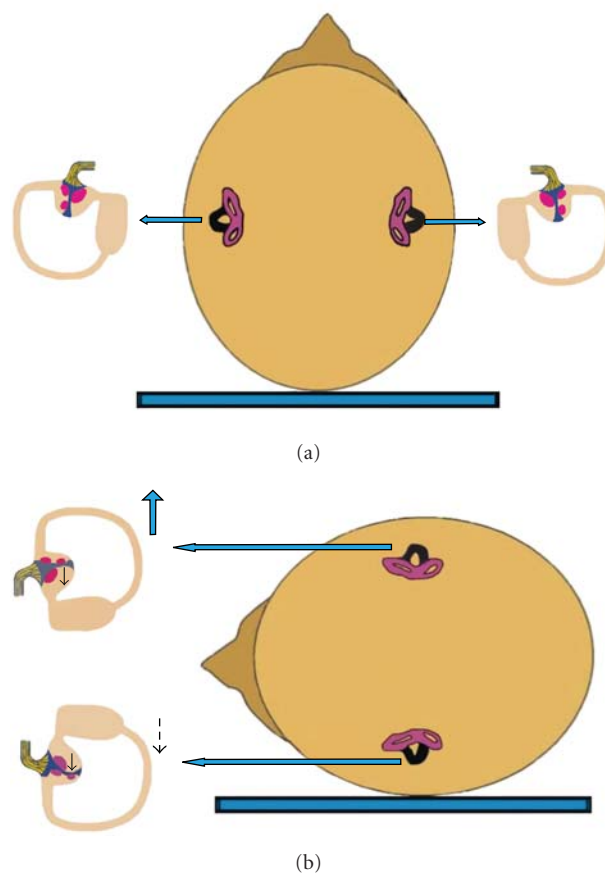


FIGURE 8: Mechanism of cupulolithiasis of bilateral apogeotropic horizontal canal BPPV (the involved horizontal canals are colored black). (a) Patient in supine position with debris adherent to the cupula of both horizontal canals. (b) Supine roll test on either side would result in inhibition of the horizontal canal of the lowermost ear, due to ampullofugal (inhibitory) deflection, triggering an apogeotropic horizontal nystagmus. At the same time, excitation of the horizontal canal of the uppermost ear would occur, due to ampullopetal (stimulatory) deflection of the cupula. Vectorial summation would result in an intense, more or less symmetric, apogeotropic nystagmus.

test, may be explained from concomitant canalolithiasis-cupulolithiasis in the horizontal canal of the lower ear [19].

It may be thus concluded that although patients with bilateral disease of the horizontal canal may exist, difficulty in diagnosis may explain why cases with this type of vertigo have been scarcely reported. Horii et al. [34] described such an interesting case of bilateral horizontal BPPV, treated successfully by canal plugging of the horizontal canal on one side and the Lempert maneuver on the other side.

## 8. Bilateral Anterior Canal BPPV

Bilateral anterior canal is also very difficult to diagnose. Theoretically, the Dix-Hallpike maneuver on the right side would cause paroxysmal nystagmus with a vertical downbeating component and a torsional component with the upper pole

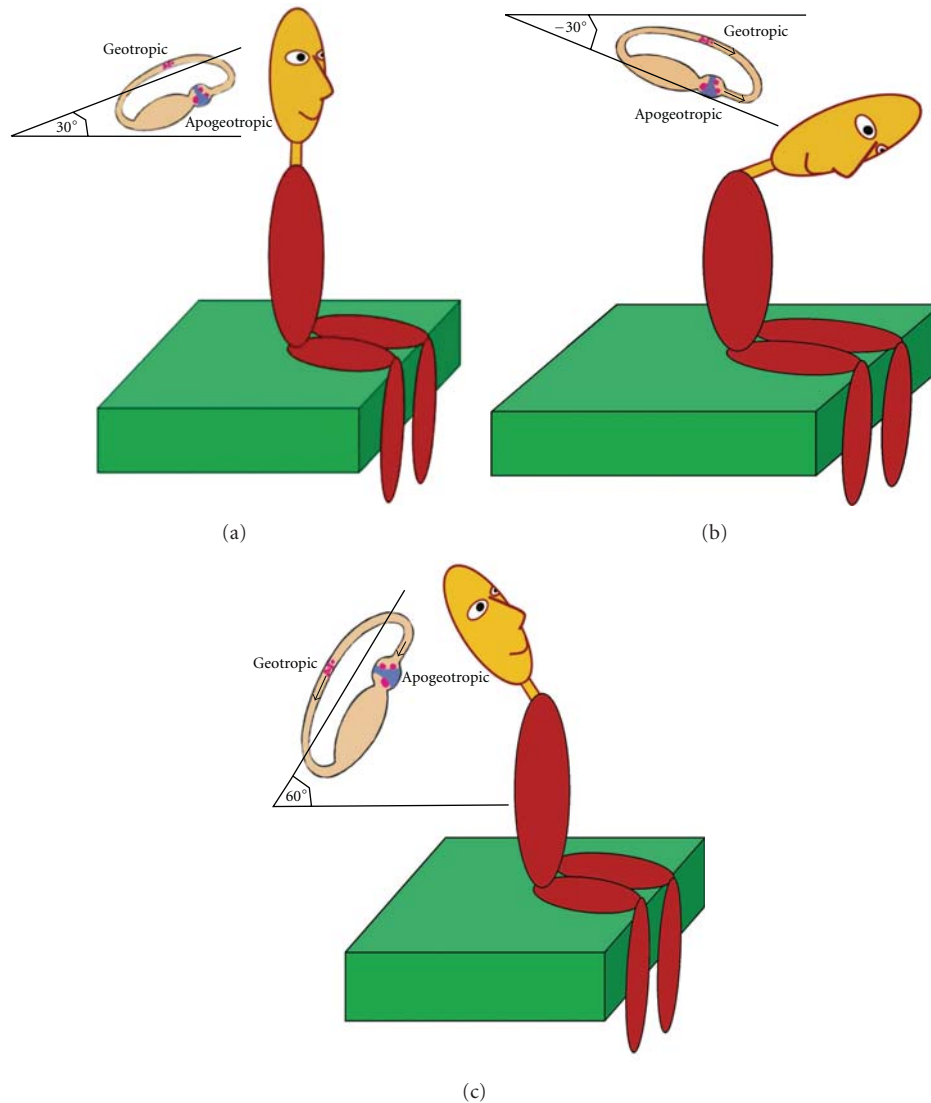


FIGURE 9: Pseudospontaneous nystagmus in horizontal canal BPPV. (a) When the head is erect, there is an angle of  $30^\circ$  between the horizontal plane and the horizontal semicircular canal, which may explain the pathogenesis of pseudospontaneous nystagmus. Any head movements would cause ampullofugal movement of otoconia, in case of canalolithiasis (geotropic type), provoking a nystagmus with fast phase towards the unaffected ear. In case of cupulolithiasis (apogeotropic type), the attached otoconial mass would cause deflection of the cupula in the opposite direction, triggering a reverse nystagmus. (b) When the head is bent  $60^\circ$  forward, pseudospontaneous nystagmus may change its direction, due to the inversion of the movement of otoconia (geotropic type) or of the deflection of the cupula (apogeotropic type). (c) When the head is bent backwards, pseudospontaneous nystagmus will increase, because the horizontal canal will approach the vertical position.

of the eye beating clockwise (at opposite direction of the posterior canal BPPV). This type of nystagmus is attributed to excitation of the contralateral anterior canal. However, as previously discussed, the ipsilateral anterior canal would be also excited, resulting in a downbeating vertical component, and a torsional in the opposite direction, but probably of a smaller intensity [35]. Vectorial summation of all the components would result in an intense vertical downbeating component and a weak torsional component towards the upper ear. In conclusion, the Dix-Hallpike maneuvers on both sides would produce a mixed nystagmus, with an intense vertical and a weak torsional component on both occasions opposite to those of posterior canal involvement.

Differential diagnosis would be difficult because in unilateral anterior BPPV, the torsional nystagmic vector may be quite often absent.

Furthermore, in a head down test, performed in a similar way as in the case of bilateral posterior canal BPPV, both anterior canals would get irritated again, resulting in the appearance of nystagmus. This nystagmus should be characterized by only an intense purely vertical downbeating component, because the torsional components would cancel each other, as having opposite directions. Differential diagnosis on these grounds would not be safe, because unilateral anterior canal involvement, as previously mentioned, may manifest as purely vertical as well. Finally, the criterion of

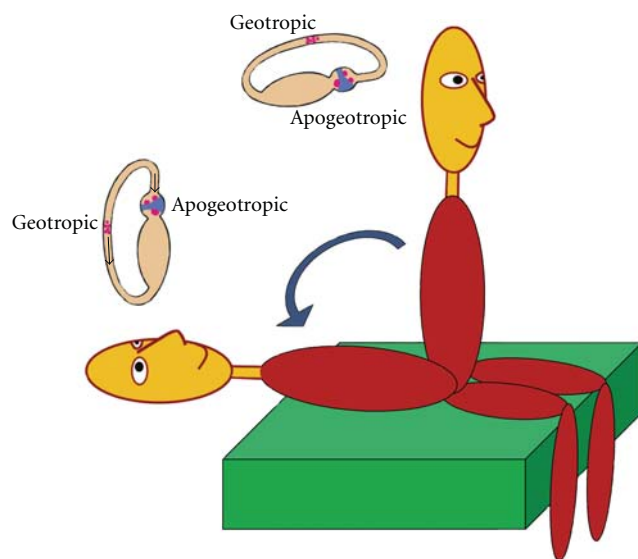


FIGURE 10: When the patient with horizontal canal BPPV is quickly brought from the sitting to the supine position, a mild horizontal nystagmus may appear, attributed either to ampullofugal movement of otoconia in the horizontal canal (geotropic type), triggering nystagmus toward the unaffected ear, or in cases of cupulolithiasis (apogeotropic type) to ampullopetal deflection of the cupula, resulting in nystagmus directed toward the affected ear.

responsiveness to treatment is not helpful in this case. There is not established therapeutic maneuver for treatment of the anterior canal BPPV, and both standard Epley and reverse Epley maneuvers have been used, as well as various specifically designed maneuvers [36, 37]. To conclude, although the entity of bilateral anterior BPPV has been previously reported [4, 22], any details concerning its diagnosis and treatment are missing. We believe that this diagnosis is presently highly hypothetical.

## 9. Horizontal and Posterior Canal BPPV

This is the most common case of mixed canal BPPV [37–39]. The involved canals may be either on the same side or on both sides. Diagnosis may be easily obtained, considering the features of the nystagmus on either maneuver. Mixed torsional geotropic-vertical upbeat nystagmus in Dix-Hallpike maneuvers reveals involvement of the posterior canal. Additionally, geotropic or apogeotropic horizontal nystagmus during the supine roll test will be evidence of horizontal canal BPPV. In most mixed cases, the horizontal nystagmus is geotropic due to canalolithiasis, although cupulolithiasis has been occasionally reported [40]. It should be noticed, however, that during the Dix-Hallpike tests, the horizontal canal is also, at least, partially stimulated, and a horizontal component of nystagmus may be evident in conjunction with the torsional-upbeat nystagmus of posterior canal origin [41]. Additionally, it has been shown that horizontal nystagmus, provoked during the supine roll test, exhibits also a vertical and a torsional component [42].

## 10. Horizontal and Anterior Canal BPPV

Occurrence of this combination is quite unusual, due to rare involvement of the anterior canal. Diagnosis of horizontal canal involvement is evident, according to previously described characteristics of the nystagmus. Anterior canal BPPV may be also diagnosed from the experienced clinician, as already described. The main feature for differential diagnosis from the posterior canal BPPV is the downbeating vertical component of the nystagmus. Additionally, the direction of the torsional component will show if either the ipsilateral or the contralateral canal is involved.

## 11. Posterior and Anterior Canal BPPV

This combination has also been reported [4, 22], but its diagnosis presents difficulties. Two categories of involvement should be distinguished, on the same side and on different sides.

- (i) If the involved posterior and anterior canals are on the same side, then Dix-Hallpike on this side would cause theoretically (1) a torsional component with the upper eye pole moving geotropically and a vertical upbeat component, due to posterior canal disease; (2) a torsional component with the same direction and a vertical downbeating component, due to anterior canal disease. The net result would be only a strong torsional component, because the two torsional components would be added and the two vertical components would be cancelled, as having opposite directions. Dix-Hallpike on the nonaffected ear would cause a torsional component with the upper eye pole moving apogeotropically and a vertical downbeating component, due to anterior canal disease, as previously discussed.
- (ii) If the involved posterior and anterior canals are on different sides, then Dix-Hallpike on the side where the posterior canal is involved, theoretically would cause (1) a torsional component with the upper eye pole moving geotropically and a vertical upbeat component, due to posterior canal disease; (2) a torsional component with opposite direction and a vertical downbeating component, due to anterior canal disease of the contralateral side. The net result will be absence of nystagmus, due to vectorial subtraction of the partial components. However, some asymmetry of canal involvement may manifest as mild nystagmus, either torsional, or vertical, or mixed, depending on the intensity of the partial components. Dix-Hallpike on the side of the involved anterior canal would cause a mixed nystagmus, with a torsional component with the upper eye pole moving geotropically and a vertical downbeating component, due to ipsilateral anterior canal disease.

From what has been mentioned above, it is understandable why mixed posterior-anterior canal is so difficult to be diagnosed with certainty. In case of suspicion, separate

CRPs for the posterior canal (mainly the Epley CRP) and a specific therapeutic procedure for the anterior canal [35] could support the diagnosis, if treatment could be obtained. However, it should be noticed that an Epley maneuver for posterior canal BPPV is also a reverse Epley (and probably therapeutical) for the contralateral anterior canal, thus complicating this issue further.

## 12. Conclusions

- (i) Typical posterior canal BPPV and horizontal canal BPPV are usually easy to diagnose, using the standard Dix-Hallpike and supine roll maneuvers, respectively.
- (ii) Anterior canal BPPV presents difficulties in diagnosis because it may demonstrate mixed vertical-torsional nystagmus on both right and left Dix-Hallpike maneuvers. Additionally, the torsional nystagmic component may be missing.
- (iii) BPPV involving both posterior canals may be easily detected, but it is almost impossible to diagnose in cases of bilateral horizontal or anterior canal involvement.
- (iv) BPPV involving two different canals, either on the same or on different sides, may be quite safely diagnosed in typical cases of posterior-horizontal and anterior-horizontal involvement. However, the combination of posterior-anterior canal involvement is more difficult to diagnose with certainty.

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## Review Article

# Inner Ear Disease and Benign Paroxysmal Positional Vertigo: A Critical Review of Incidence, Clinical Characteristics, and Management

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**Background.** This study is a review of the incidence, clinical characteristics, and management of secondary BPPV. The different subtypes of secondary BPPV are compared to each other, as well as idiopathic BPPV. Furthermore, the study highlights the coexistence of BPPV with other inner ear pathologies. **Methods.** A comprehensive search for articles including in the abstract information on incidence, clinical characteristics, and management of secondary BPPV was conducted within the PubMed library. **Results.** Different referral patterns, different diagnostic criteria used for inner ear diseases, and different patient populations have led to greatly variable incidence results. The differences regarding clinical characteristics and treatment outcomes may support the hypothesis that idiopathic BPPV and the various subtypes of secondary BPPV do not share the exact same pathophysiological mechanisms. **Conclusions.** Secondary BPPV is often under-diagnosed, because dizziness may be atypical and attributed to the primary inner ear pathology. Reversely, a limited number of BPPV patients may not be subjected to a full examination and characterized as idiopathic, while other inner ear diseases are underdiagnosed. A higher suspicion index for the coexistence of BPPV with other inner ear pathologies, may lead to a more integrated diagnosis and consequently to a more efficient treatment of these patients.

## 1. Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder in adults, with a lifetime prevalence of 2.4% [1]. Clinical and laboratory researches have revealed that BPPV is caused by vestibular lithiasis. Dense particles, most likely displaced otoliths, provoke an abnormal deflection to the cupula (a) most commonly when free-floating in the semicircular canals (SCCs) (canalithiasis), (b) when attached to, or impinging upon, a cupula (cupulolithiasis), or (c) most rarely when jammed in a canal or cupula (canalith jam). In any of these conditions, the abnormal deflection of the cupula induces vertigo, that can be severe and incapacitating, as well as nystagmus in the plane of the involved SCC [2]. The mechanism of detachment of the otoconia is not fully understood. It seems that any inner ear disease that detaches otoconia and yet does not totally

destroy SCC function can induce secondary BPPV. The most commonly recognised conditions associated with secondary BPPV are head trauma, vestibular neuritis, Ménière's disease, and postsurgical. Other lesions that affect the inner ear and have been implicated in the pathogenesis of secondary BPPV are sudden sensorineural hearing loss and migraine. Ideally, for a causative association to be strong, BPPV should be ipsilateral to the associated condition and symptoms should develop at the same time or after the development of the primary condition. In some cases, it is not clear whether there exists a true causative effect or there is a coincidental association. In most patients with BPPV, a direct association with an ipsilateral disease process affecting the labyrinth can not be identified and idiopathic BPPV remains the most common diagnosis.

To date, few studies have focused on secondary BPPV, which may often be an underdiagnosed entity. The aim of

this study was to review the incidence, clinical characteristics, and management of secondary BPPV. Despite the obvious similarities, differences between the clinical manifestations and the outcome of repositioning manoeuvres between the several types of secondary and idiopathic BPPV seem to dictate different diagnostic, counselling, treatment, and follow-up strategies. Another target of this study is to highlight the coexistence of BPPV with a number of pathologies that are also typically associated with dizziness. In these cases, BPPV is often underdiagnosed, because dizziness is attributed to the primary pathology. A number of BPPV patients may describe the resulting vertigo in a rather atypical way, while further testing might reveal typical BPPV [3, 4]. Although less frequently, the reverse may also be true. A significant number of BPPV patients may not be subjected to a full audiological and neurotological examinations and characterised as idiopathic, while other inner ear diseases are underdiagnosed. A higher suspicion index for the coexistence of BPPV with other inner ear pathologies, together with the appropriate examinations, may lead to a more integrated diagnosis and consequently to a more efficient treatment.

## 2. Methods

A comprehensive search for articles regarding BPPV was conducted within the PubMed library attributed 599 articles (search string: {benign paroxysmal positional vertigo OR positional vertigo OR benign paroxysmal vertigo} with Limits used in Title and English). Studies that did not include in the abstract information on incidence, clinical characteristics, and management of secondary BPPV were excluded from the study. A manual cross-reference search of the bibliographies of included papers was carried out to identify additional potentially relevant studies.

## 3. Results

Based on the inclusion in the abstract information on incidence, clinical characteristics, and management of secondary BPPV, 33 papers were initially included in this review. The manual cross-reference search of the bibliographies of included papers substantially increased the number of reviewed papers into 55. For reporting incidence, only large studies of >100 patients were included in this review [5–13]. Variation in the studies' methodology has in some cases complicated comparisons. For example, some studies do not state whether the inner ear disease predated BPPV or the coexisting pathology was only present and on which side. In some studies, head trauma is considered as an event in terms of idiopathic BPPV and therefore not a cause of secondary BPPV [9]. The prognostic role of the simultaneous diagnosis of BPPV and vestibular neuritis, idiopathic sensorineural hearing loss, or Ménière's disease has not been adequately addressed in the literature in terms of both vestibular rehabilitation and recovery from the primary inner ear disease. The same seems to apply for the therapeutical outcome of repositioning manoeuvres in postsurgical BPPV patients.

## 4. Discussion

**4.1. Incidence and Possible Pathogenetic Mechanisms.** A wide variation of incidence of secondary BPPV (3–66%) is observed across studies [5–13]. It is of note that in two large studies by Karlberg et al. (2847 subjects) [9] and Caldas et al. (1271 subjects) [11], the incidence of secondary BPPV varied considerably between 3% and 25.2%, respectively. This may reflect different referral patterns, different diagnostic criteria used for inner ear diseases, and different patient population. The commonest pathologies underlying the induction of secondary BPPV (as percentage of all cases of BPPV) included head trauma (8.5–27%), Ménière's disease (0.5–30%), vestibular neuritis (0.8–20%), and idiopathic sensorineural hearing loss (0.2–5%) [5–13].

The mechanical detachment of otoconia through *head trauma* is the most commonly associated condition, with the reported incidence of head trauma among BPPV patients ranging 8.5–27% [6–8, 12–14]. The nature and severity of the traumas causing trauma-BPPV are diverse, ranging from minor head injuries to more severe head and neck trauma with brief loss of consciousness. To reinforce the etiological relationship between head trauma and BPPV, we may note that the incidence of BPPV in a study of 150 consecutive severe head trauma patients has been reported to be significantly higher than in the general population (6.6%) [15]. Following the reported high incidence rates, secondary BPPV should be suspected in any case of head trauma accompanied with positional vertigo, and a Dix-Hallpike examination should be included in the diagnostic protocol of these patients, in some cases, despite the consequent patient discomfort.

The incidence of *Ménière's disease* (MD) among BPPV patients has been reported within the wide range of 0.5–30% [5–13]. Vice versa, based on a study of 500 patients with MD, it is estimated that approximately 65 to 70% of patients will experience BPPV between attacks of the disease [16]. Another interesting observation is that a significant percentage of MD patients (9/162 or 5.5%), mostly females, seem to develop intractable BPPV [17]. Therefore, the examination of MD patients should also involve the application of a Dix-Hallpike test for the exclusion of secondary BPPV. The importance of such a missed diagnosis lies, obviously, in the different therapeutical approaches, the immediacy of patient relief that may follow an appropriate repositioning manoeuvre, and the sustained efficacy of a long medical treatment for MD.

The underlying pathophysiological mechanism seems to regard an endolymphatic hydrops-induced destruction of the maculae of the utricle and saccule either through vascular compromise or through direct distortion of its surface, resulting in detachment of otoliths into the endolymph. Incidence rate increases as the MD course is prolonged [18]. This may be explained by the hypothesis that periodic hydropic distension, as seen in the natural course of MD, may enhance detachment of otoliths through macular fibrosis [17]. Temporal bone studies have verified the existence of free-floating deposits in at least one semicircular canal of subjects with BPPV and MD, as well as significant differences

in the incidence of cupular and free-floating deposits in the posterior and lateral semicircular canals between subjects with MD and healthy controls. The findings have been associated with the duration of disease rather than with aging [19]. Therefore, an exclusion of secondary BPPV should be incorporated in the clinical examination of patients with MD, especially those with a long history of the disease.

The incidence of *vestibular neuritis* among BPPV patients has been reported within the wide range of 0.8–24.1% [9–11]. Vice versa, in patients with vestibular neuritis the incidence of BPPV appears to be more frequent (9.8–20%) than in the general population [20–22]. These percentages seem to justify the application of a Dix-Hallpike examination to patients with vestibular neuritis, as well as the performance of a detailed clinical and laboratory neurotological testing in patients with BPPV. In fact, the application of nystagmography in idiopathic BPPV patients has been reported to reveal ipsilateral canal paresis at a percentage of 13–47% [7, 14, 23]. Furthermore, the percentage of abnormal vestibular-evoked myogenic potentials (VEMPs) in BPPV patients has been reported to be statistically higher than in control ears ( $P < 0.005$ ) [24]. Although these findings have been hypothesized to correspond to a more extensive inner ear lesion, their diagnostic and/or prognostic value remains unclear.

The pathogenetic mechanism underlying secondary BPPV in patients with vestibular neuritis seems to derive from the distribution of the vestibular nerve in the inner ear. The superior vestibular nerve innervates the cristae of the anterior and lateral SCCs and the macula of the utricle. A lesion of the lateral semicircular canal and the superior vestibular nerve is associated with abnormal nystagmographic findings. The typical superior vestibular nerve lesion sparing the inferior division of the nerve seems to be the main pathogenetic mechanism underlying BPPV in vestibular neuritis [25]. Damage to the utricle may detach the otoconia. More extended utricle damage is possibly expected to be more likely to induce the detachment of otoconia. However, the prognostic role of BPPV in patients with vestibular neuritis has not been investigated yet. After otoconia is detached from the utricle, it could enter the posterior SCC duct. The clinical signs and symptoms of posterior SCC BPPV will be presented, because this SCC is innervated from the inferior vestibular nerve. Damage to the superior vestibular nerve innervating the anterior and lateral SCCs may abolish the vestibuloocular reflex pathway from these SCCs. Therefore, posterior SCC-BPPV nystagmus is, as expected, the typical finding in BPPV patients with vestibular neuritis. This implies that at least some function in the inferior vestibular nerve remains, as it is also supported by the preserved vestibular-evoked myogenic potentials in postneurolabyrinthitis patients [26]. These potentials are most likely of saccular origin and both the macula of the saccule and the crista of the posterior canal are innervated by the inferior vestibular nerve.

*Postsurgical BPPV* seems to be another underdiagnosed entity. Surgeries involving drilling and especially maxillofacial and dental surgery including placement of dental implants [27, 28] and cochlear implantation [29, 30], have been associated with secondary BPPV. Incidence has been

reported at 3% and 0–28%, respectively [28–32]. The incidence of secondary BPPV in otosclerotic patients ranges between 6.3 and 8.5%, it is developed between the 5th and 21st days after surgery and attributed to utricular trauma [33, 34]. Surgeons should probably not omit the exclusion of BPPV in patients complaining of Postsurgical dizziness by the use of a simple clinical examination, before subjecting them to imaging examinations and before a final diagnosis involving other Postsurgical complications is reached.

According to the hypothesized mechanism, drilling might detach utricular otoconia mechanically, in a similar way as head trauma does. The hypothesized mechanism underlying secondary BPPV after cochlear implantation presents some additional interesting aspects. Regarding this type of Postsurgical BPPV, a considerably heterogeneous and long time interval has been reported between surgery and initiation of BPPV symptoms (28–165 days in the study of Viccaro et al. and 1–880 days in the study of Limb et al.) [29, 30]. For the patients with delayed development of BPPV, direct falling of bone dust particles into the cochlea during cochleostomy, as well as dislodging of otoliths through, electrical stimulation, has been hypothesized to account for BPPV [29]. After falling into the cochlea, bone dust particles might, through a microrupture of the basilar membrane, travel into the endolymphatic compartment of the scala media and into the lumen of the posterior semicircular canal, thereby producing canalolithiasis and subsequent delayed-onset BPPV [30]. The hypothesis of the dislodging of otoconia because of electrical stimulation seems to be less possible, when taken into consideration that approximately 1/3 of these patients have been reported to experience symptoms before implant activation [27].

Although in most studies there is no distinction between sudden deafness and *sudden sensorineural hearing loss* (SSNHL), or whether the hearing loss was the sole symptom or part of a coexisting pathology, there seems to be an association between idiopathic sudden sensorineural hearing loss and BPPV. This pathology is encountered in 0.2–5% of BPPV patients. Vice versa, the diagnosis of BPPV has been reported for 12.7% of patients with SSNHL [35]. Therefore, patients with idiopathic sudden hearing loss and dizziness should be subjected to clinical examination for the diagnosis of BPPV, even though typical BPPV symptoms may not be described by the patient [3, 4]. Regarding the underlying pathogenetic mechanism, it is logical to hypothesize that otoconia is in these cases detached due to vascular compromise or viral lesions of the macula; however, the underlying mechanism remains actually unknown.

The incidence of BPPV is also known to be higher in patients who suffer from *migraine*. Lempert et al. [36] found that the prevalence of migraine in patients with BPPV was twice as high as that in age- and sex-matched controls. The relationship between migraine and BPPV is poorly understood. It has been suggested that migraine causes vasospasm of the labyrinthine arteries, hence inducing local ischemia which facilitates otoconia detachment from the utricular macula [37].

Possibly through the same mechanism of vascular depletion of the inner ear, BPPV has been reported to occur in



association with giant-cell arteritis, diabetes, osteopenia/osteoporosis and hyperuricemia [38–41]. As far as osteopenia/osteoporosis is concerned, disturbed internal structure of the otoconia or their interconnection and attachment to the gelatinous matrix and reduced capacity to dissolve the dislodged otoconia owing to increased concentration of free calcium in the endolymph have also been proposed as possible underlying mechanisms [41, 42].

**4.2. Clinical Characteristics.** BPPV secondary to mild head trauma has been reported to affect younger populations with more even age and gender distribution in comparison to the idiopathic form. An important difference noted by several authors is the higher incidence of bilaterality [12, 13, 43]. Most bilateral cases in both idiopathic and secondary BPPV groups seem to apply to the PSC. Some authors report no differences in the semicircular canals involved, while others note a consistently higher prevalence of the posterior (PSC) than the horizontal semicircular canal BPPV (HSC) in both groups [10, 12, 43]. Association with chronic dizziness has been reported to be similar in the two groups [43].

Bilateral involvement is also a significant characteristic of BPPV secondary to *Ménière's disease*. In the 41 patients with unilateral *Ménière's disease* reported by Gross et al. [17], 18 had bilateral BPPV, 16 had BPPV of the same ear, and 7 had only contralateral BPPV. The horizontal semicircular canal has been reported as the most commonly affected. Onset is usually noted within one week following an attack in the majority of patients (60%), whereas simultaneous onset is uncommon (10%) [10]. This clinical characteristic possibly implies the need for more than one diagnostic session, before BPPV is safely excluded for a MD patient. Female predominance in secondary BPPV seems to follow the current epidemiology of *Ménière's disease* [17, 18]. There is a literature discrepancy regarding the most commonly affected canal. The posterior as well as the lateral semicircular canal have both been reported as the most frequently involved by different authors [10, 18].

BPPV secondary to *vestibular neuritis* is expected on average as late as 18 days after the onset of the primary disease [10]. The late emergence of BPPV after vestibular neuritis may highlight the necessity for the repurchase of the Dix-Hallpike examination at the follow-up sessions, especially in patients who present a slow recovery. BPPV seems to be in these cases a negative prognostic factor, since it has predominantly been diagnosed in patients who did not fully recover from the disease [20]. As it has been analysed in terms of the possible underlying pathogenetic mechanism, BPPV seems to consistently affect the posterior canal of the ipsilateral ear.

On the contrary, more than half of the patients with secondary BPPV due to *idiopathic sudden sensorineural hearing loss* develop it relatively early, within 24 hours after the onset of deafness [35]. Information on the most commonly affected SCC(s) is not clearly stated in the relevant reports.

Finally, *postsurgical* BPPV secondary to middle ear surgery, cochlear implantation, dental and maxillofacial surgery also affects predominantly the posterior semicircular canal possibly because the posterior SCC is situated lower than

the vestibule in the supine position [27, 29, 30]. The mean onset time for BPPV after maxillofacial and dental surgery has been reported at 4.1 days after the exclusion of patients who developed BPPV seven days or later after surgery [27]. Regarding cochlear implantation and due to the specific, other than drilling, pathogenetic mechanisms that have implicated in the development of this type of secondary BPPV, authors have adopted less strict time interval criteria, by reporting on patients who developed BPPV up to 165 or 880 days after surgery [29, 30]. Bilateral involvement in these surgeries seems to be rare, in contrast to what might have been expected through the pathogenetic mechanism of the transmission of mechanical energy through the bones and perilymphatic fluids toward the maculi [29, 44]. Patient age, implant side, device type, and aetiology of hearing loss were randomly distributed with respect to likelihood of postoperative BPPV [30]. Interestingly, no postcochlear implantation BPPV cases have been reported even in large pediatric populations [45].

**4.3. Management.** Patients with idiopathic BPPV tend to present significantly higher rates of symptoms' resolution with canalith repositioning procedures (CRP) than those with secondary BPPV due to head trauma, vestibular neuritis or *Ménière's disease* [13, 21, 46–48]. The mean durations of treatment until complete resolution of signs and symptoms have been reported at 2.28 for idiopathic BPPV and at 4.87 days for secondary BPPV. Such differences seem to apply also among the various types of secondary BPPV. In a retrospective study of 69 patients with secondary BPPV, the mean duration of treatment has been reported to be 6.28 days for idiopathic sudden sensorineural hearing loss with BPPV, 5.07 days for BPPV with vestibular neuritis and 2.28 days for BPPV with *Ménière's disease* [10]. Significant differences were noted between patients with posttraumatic BPPV and patients with idiopathic BPPV regarding both complete resolution rates after a single CRP and recurrent attacks during the 6- to 42-month followup [13]. In cases, however, where a single CRP was not enough to achieve complete resolution of symptoms and signs, the number of multiple CRPs required does not seem to reach statistical significance between the secondary and idiopathic BPPV groups [13, 46]. The aforementioned differences in the management and prognosis of idiopathic and secondary BPPV may lead to the hypothesis that they may result from quantitatively or qualitatively different lesions [47]. Moreover, the diverse clinical courses in the various subtypes of secondary BPPV may be explained by the different pathophysiologies associated with variant inner ear diseases [10]. The worse prognosis of BPPV with unilateral vestibulopathy has not been verified by other authors who, in a small group of 35 patients, reported that acute vestibular neuritis patients seem to have a tendency for a better outcome than secondary BPPV patients with any other etiology [46]. The same authors have reported that resolution rates of paroxysmal positional nystagmus after CRP seem to be similar in idiopathic and secondary BPPV patients. Despite these contradicting comparisons, the authors admit that secondary BPPV is still more difficult to

treat than idiopathic cases because in a considerable number (42%) of patients with secondary BPPV, persistence of positional vertigo after CRP is noted after the disappearance of nystagmus on the Dix-Hallpike manoeuvre [46].

Detailed neurotological studies of patients with secondary BPPV have reached the hypothesis that an additional vestibular lesion, also causing vertigo with positional triggers, may coexist with BPPV and preserve the symptoms of vertigo in some patients [46]. BPPV patients have been reported to reveal ipsilateral canal paresis at a percentage of 13–47% [7, 14, 23]. In as much as one-third of these patients, especially patients with head trauma and vestibular neuritis, the side of the paresis has been reported to be the side opposite to the ear treated for BPPV. Although the incidence of additional vestibular pathology in the contralateral side may indeed be just an incidental unrelated finding, a contrecoup lesion in the cases of head injury cannot be excluded [46]. Directional preponderance in the absence of canal paresis has been found in 22% of secondary BPPV patients, and in the absence of central vestibular abnormalities, this may also be considered as a sign of peripheral vestibular dysfunction. Therefore, the difficult management of secondary BPPV may in many cases be attributed to additional inner ear lesions. At least patients who remain symptomatic after CRP should be subjected to a more comprehensive testing of the SCCs and otolithic system. Patients with evidence of concomitant vestibular pathology would be expected to require further vestibular rehabilitation in the form of systematic or customized exercises.

Regarding the recurrence rates of BPPV secondary to *head trauma*, there is no agreement in the literature [12, 13, 49]. Some authors report that secondary BPPV has a greater tendency to recur [12, 13], while others fail to find any differences in recurrence after repositioning manoeuvres or associated chronic dizziness [43, 49].

Recurrence has been consistently reported to be significantly more common in BPPV secondary to *Ménière's disease* than in idiopathic cases [18, 50]. Endolymphatic hydrops seems to be associated with higher BPPV recurrence rates [50, 51]. The higher recurrence rates may be explained by the periodic hydropic distension, which is seen in the natural course of *Ménière's disease* and may result in repeated release of otoconia [17]. The difficulties in the treatment of BPPV in these patients may also be attributed to the repeated hydropic distension. This may reduce the elasticity of the membranous labyrinth and result in partial collapse or adhesions of the semicircular canal membranous labyrinth which therefore exhibits partial obstruction(s) [18]. A dilated saccule, as well as adhesion of otoliths to the membranous labyrinth has also been postulated as a possible mechanism of partial obstruction [17, 52, 53]. In all the above mentioned hypotheses, partial obstruction can persist independently of *Ménière's disease* recurrences. With partial obstruction, canalith repositioning is impeded, although still feasible, and this may provide some explanation for the persistent BPPV and the lower responses of these patients to canalith repositioning manoeuvres [17]. There is no data neither on the possible prognostic role of BPPV in the course of MD nor on the effect of BPPV on the rehabilitation of these patients.

Although several studies have highlighted the negative prognostic value of vertigo in *idiopathic sudden sensorineural hearing loss*, the majority of cases do not regard BPPV [54, 55]. The unfavourable prognosis of patients with idiopathic sudden sensorineural hearing loss and BPPV reported by Lee and Ban [35] may need to be verified by additional studies. Such studies on the possible prognostic role of BPPV in the course of vestibular neuritis and on the effect of BPPV on the rehabilitation of these patients are also currently lacking.

Information on the management of *postsurgical* BPPV is also scarce. Most authors note that patients were successfully treated with CRP, without further commenting on the number of repositioning manoeuvres needed to resolve BPPV symptoms. In a cohort of 8 patients after cochlear implantation, Viccaro et al. [29] have reported the case of one patient with persistent BPPV that did not respond to repositioning manoeuvres and continued to have symptoms when turning the head toward the implanted side. BPPV does not seem to affect cochlear implant performance [30].

## 5. Conclusions

Secondary BPPV seems to be an underdiagnosed entity, especially among patients with known causes of vertigo such as *Ménière's disease*, vestibular neuritis, idiopathic sudden sensorineural hearing loss, and postsurgical patients. A higher suspicion index and the incorporation of the Dix-Hallpike test in the examination battery of all patients with vertigo, regardless of a known primary inner ear disease, may lead to the diagnosis of underlying secondary BPPV and offer those patients an optimal and efficient treatment. Reversely, BPPV seems to be associated with inner ear disease in more cases than it has been generally believed. In many patients, the initial finding of BPPV is assumed to be the final diagnosis and other neurotological tests are neglected. It is however recommended to complete the neurotological examination even if BPPV has been diagnosed with the clinical provocative tests. Especially in BPPV patients with persisting symptoms, a comprehensive audiological and neurotological evaluation should probably be performed in order to recognise any associated inner ear pathology.

Unfortunately, the clinical differences between idiopathic and secondary BPPV demonstrated by large studies are indefinite and of limited clinical value. Most studies agree that secondary BPPV is more difficult to treat than idiopathic and patients require longer time intervals before becoming free from clinical symptoms. The clinical manifestation of an additional inner ear lesion may be a possible explanation. Therefore, a detailed medical history, and clinical, laboratory, and follow-up examination seem to be of outmost importance for the diagnosis and successful management of secondary BPPV. Further clinical studies are needed in order to investigate the possible prognostic role of secondary BPPV in various inner ear diseases as well as any differences in the time course and efficiency of rehabilitation in these patients.

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## Review Article

# Benign Paroxysmal Positional Vertigo (BPPV): History, Pathophysiology, Office Treatment and Future Directions

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BPPV is the most common cause of vertigo. It most often occurs spontaneously in the 50 to 70 year age group. In younger individuals it is the commonest cause of vertigo following head injury. There is a wide spectrum of severity from inconsistent positional vertigo to continuous vertigo provoked by any head movement. It is likely to be a cause of falls and other morbidity in the elderly. Misdiagnosis can result in unnecessary tests. The cardinal features and a diagnostic test were clarified in 1952 by Dix and Hallpike. Subsequently, it has been established that the symptoms are attributable to detached otoconia in any of the semicircular canals. BPPV symptoms can resolve spontaneously but can last for days, weeks, months, and years. Unusual patterns of nystagmus and nonresponse to treatment may suggest central pathology. Diagnostic strategies and the simplest “office” treatment techniques are described. Future directions for research are discussed.

## 1. History and Pathophysiology

Benign paroxysmal positional vertigo (BPPV) is the most common vertiginous disorder in the community. The cardinal symptom is sudden vertigo induced by a change in head position: turning over in bed, lying down in bed (or at the dentist or hairdresser), looking up, stooping, or any sudden change in head position. There is a wide spectrum of severity. Mild symptoms are inconsistent positional vertigo. Moderate symptoms are frequent positional attacks with disequilibrium between. When severe, vertigo is provoked by most head movements, giving an impression of continuous vertigo. The symptoms can last for days, weeks, months, or years, or be recurrent over many years.

The earliest reference to it may have been by Shakespeare in “Romeo and Juliet” [1]. In Act I, Scene II [enter Romeo and Bevolio] Bevolio says “Tut man, one fire burns out another’s burning. One pain is lessen’d by another’s anguish; turn giddy, and be help by backwards turning...” In the medical literature the first descriptions of positionally induced vertigo are attributed to Adler [2] and later Barany [3], who believed it was a disorder of the otolith organs. Barany elicited vertigo in a 27-year-old woman by turning her head from side to side in a supine position and noted

“...there appeared a strong rotatory nystagmus to the right with a vertical component upwards, which when looking to the right was purely rotatory, and when looking to the left was purely vertical.” In 1952 Margaret Dix (1911–1981) and Charles Hallpike (1900–1979) [4] at Queen Square Hospital, based on 100 patients, presented a symptomatological definition and a provocative positional test for what they called “positional nystagmus of the benign positional type.” For symptoms they note: “The story given by the patient is characteristically that the giddiness comes on when he lies down in bed or when he turns over in bed, or when such a position is taken up during the day; for instance lying down beneath a car or in throwing the head backward to paint a ceiling.” Their diagnostic test: “...the patient is first seated upon the couch with the head turned to one side and the gaze fixed firmly on the examiner’s forehead. The examiner then grasps the patient’s forehead firmly between his hands and briskly pushes the patient back into the critical position [30 degrees below the level of the couch and turned some 30 to 45 degrees to one side]. The reaction which results calls for some detailed description.” As did Barany they noted a torsional nystagmus with the upper pole of the eye beating (fast phase) toward the ground and that it “fatigued” on retesting. Additionally, they observed a

response latency of approximately 5 seconds, a crescendo and decline of nystagmus, and a reversal of the nystagmus as the patient sits up. To eliminate the possibility that the response could be induced by vascular occlusion from rotation of the neck they tested patients on an apparatus which avoided it. The same response occurred. In Britain Hallpike was a pioneer of temporal bone histology. The right temporal bone of 40-year-old woman with “positional nystagmus of the benign positional type...to the right with the right ear undermost” was examined. In the macula of the utricle, the otolithic membrane was absent. They concluded: “The general picture is one of chronic tissue changes resulting either from infection or trauma...” and “We are thus directed to the conclusion that the lesion is a peripheral one and in the labyrinth towards which, when undermost, the nystagmus is directed”. Hallpike provided further evidence for a peripheral cause by abolishing symptoms in two patients with a chemical labyrinthectomy of an acoustically dead ear [5] and in one patient by an eighth nerve section [6]. Both Barany and Dix and Hallpike concluded that “positional nystagmus of the benign positional type” was caused by disorder of the utricular macula.

By the early 19th century the bony and some membranous structure of the inner ear were anatomically well described but their functions unproven. Common notions were the following. The cochlea was responsible for mediating the nature and pitch of sound; the saccule and utricle were for perception of loudness, and the semicircular canals for transmission of bone-conducted sound and perception of sound direction [7]. Marie-Jean Flourens (1794–1867) was a professor of comparative anatomy in Paris, and in 1824 he published his experimental results on pigeon semicircular canals [8]: “If the membranous ducts are injured, a painful sensitivity to tones is observed, accompanied by abrupt and violent movements of the head... If the horizontal canals are severed, the animal turns on its vertical axis; if the posterior vertical canal is severed the animal rolls over backward, and if the anterior vertical canal is severed the animal falls forward...” Flourens concluded that the semicircular canals inhibited motion (“forces moderatrices”) and influenced direction of motion, rather than having a role in balance. Flourens’ work had been largely ignored but was known to Prosper Meniere and acknowledged in his final paper in 1861 [9]. According to Adam Politzer (1835–1920) in his “History of Otolology” [10] “the realization that the vestibular and semicircular canal structures are not organs of sound perception, that sound perception is transmitted solely through the cochlea, is the single most important result of Flourens’ experiments”. However it was another sixty years until a more sophisticated understanding of semicircular canal functions and their generated nystagmus was achieved by Julius Ewald (1855–1921) who was later Professor of Physiology at the University of Strassburg (now Strasbourg). In pigeons, he cannulated each semicircular canal and applied negative and positive pressures and observed the directions and intensity of the induced nystagmus [11]. The two major findings have become known as Ewald’s Laws: (1) the direction of the induced nystagmus is in the plane of the canal being stimulated, and (2) in the horizontal

canal an ampullopetal (towards the vestibule) movement of endolymph causes the greatest response where as in the posterior and superior canals an ampulofugal (away from the vestibule) endolymph movement causes the greatest response. At the time the differences were perplexing, as expressed by a writer in 1920 [12]: “It is, however, difficult to imagine how the same endolymph current can be stimulating for the one endorgan and hindering for the other”.

Thirty years later the advent of the electron microscope allowed a more detailed view of inner ear ultrastructure. In 1954 Wersall [13] showed that each vestibular sensory cell has one kinocilium and many stereocilia. The finding of morphological polarization of kinocilia on vestibular sensor cells [14, 15] explained Ewald’s paradox. In horizontal canal cristae the kinocilium is on the vestibule side of the stereocilia; in the posterior and superior canals the kinocilium is on the canal side of the stereocilia (Figure 1). In the 1960s, experiments in cats [16] clarified the relationship between canal receptors and extraocular muscles. Each receptor is connected to one ipsilateral and one contralateral muscle. The second order neurones are either excitatory (to the agonist muscles) or inhibitory (to the antagonist muscles) (Figures 2, 3, 4, and 5).

In 1962 Harold Schuknecht (1917–1996) at Harvard University in Boston [17] proposed that BPPV “might be caused by detached utricular otoconia, acting upon the cupula of the posterior semicircular canal. Although at that time there were no confirming human pathological studies, the concept seemed plausible from a purely theoretical point of view.” In 1969 Schuknecht [18, 19] confirmed finding basophilic staining masses attached to the posterior canal cupula in patients who had had BPPV symptoms. He called this cupulolithiasis (heavy cupula) and assumed the masses were detached utricular otoliths which were removed by decalcification in preparation. This was supported by Gacek’s report of five patients where the selective resection of the posterior ampullary nerve abolished BPPV symptoms [20]. Cupulolithiasis became the dominant theory for nearly thirty years, although it did not explain the variable and often long latency and fatigability of the nystagmus. It was the impetus for two early specific treatments. Previously “treatment” had been by Cawthorne’s exercises in which the patient was instructed to repeat continually any movement which caused the vertigo until it ceased, on the assumption that central adaption was occurring [21]. Based on the cupulolithiasis theory Brandt and Daroff [22] devised an inpatient treatment where subjects lay down to the provocative side, sat up for thirty seconds, and then lay to the other side every three hours. After seven to ten days, 61 of 67 subjects were free of symptoms. The assumed aim was detachment of the particle from the posterior canal cupula. In France Semont (a physiotherapist) and Sterkers [23, 24] modified this to a logical physician-controlled treatment they called the Liberatory maneuver, now known as the Semont maneuver (Figure 6). The patient is lain down to the side of the symptomatic ear, facing down. When the nystagmus ceases, the patient is moved rapidly through 90 degrees to the opposite side (where the symptomatic ear becomes uppermost). Either immediately or up to 15 seconds later

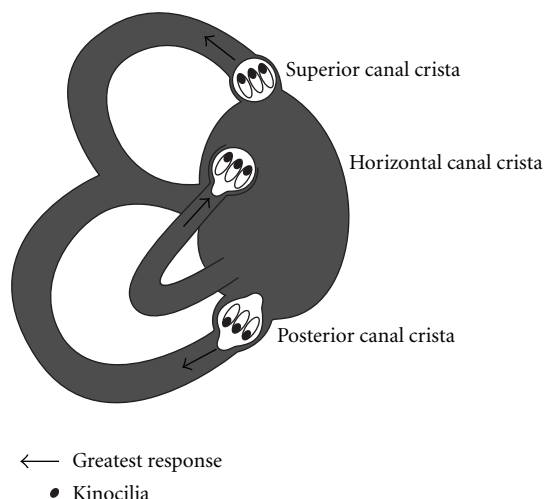


FIGURE 1: Orientation of kinocilia in the semicircular canal cristae. In the horizontal canal kinocilia are on the vestibule side. In the posterior and superior canals kinocilia are on the canal side.

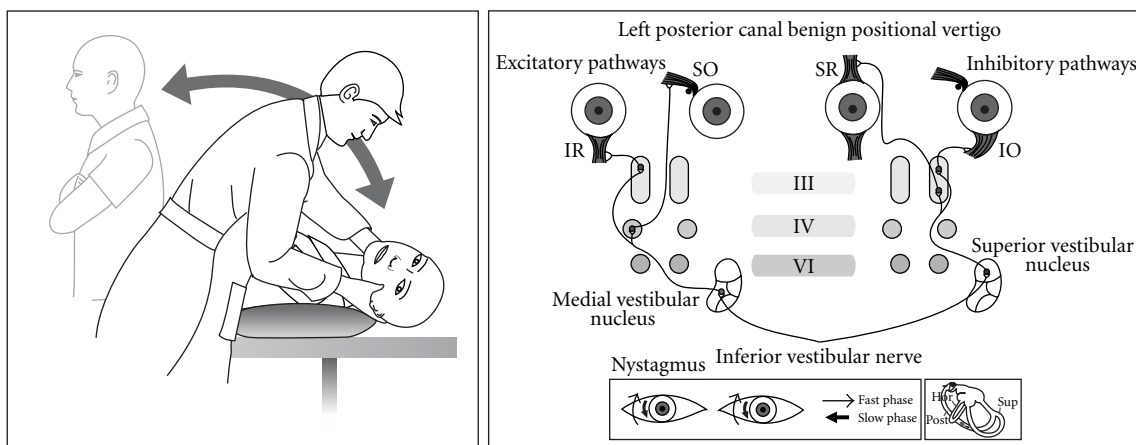


FIGURE 2: Posterior canal BPPV in a left ear showing Dix Hallpike test, inner ear, and receptor connections to the extraocular muscles.

the patient experiences vertigo and has nystagmus identical to the symptomatic side. The technique was little known outside France.

In attempting to explain the latency and fatiguability of BPPV nystagmus, Hall et al. [25] (at the University of London, Ontario) and later Epley [26] (a solo private practice otologist in Portland, Oregon) made models of the semicircular canals and proposed that they were better explained by free-floating particles in the posterior canal, which Epley called canalithiasis. Also at the University of London, Ontario, Parnes and McClure, in attempting a surgical posterior canal occlusion, observed and photographed free otoconia in the endolymphatic compartment [27]. Based on his models Epley proposed a controlled set of head movements he called the canalith repositioning procedure (CRP) [28] (Figure 7). Epley had presented this as an instruction course at the American Academy of Otolaryngology, Head and Neck Surgery meetings since 1980 and endured considerable derision because he used a heavy massage vibrator over the mastoid process [29].

After seeing canaliths at operation, Parnes [30] described an almost identical particle repositioning maneuver (PRM) (often known as the Modified Epley maneuver) whose main difference is its slower pace.

BBPV (85% posterior canal) is now recognized as the most common cause of vertigo in adults. It is estimated that 2.4% of people experience at least episode in their life [31]. 9% of residents in a home for the elderly were found to have BBPV [32]. The onset is most commonly between the fifth and seventh decades. It is the most common cause of vertigo after a head injury [33, 34]. An episode of vestibular neuritis [35] and a period of bed rest [36] are common antecedents. Omission of a simple clinical test can result in patients undergoing unnecessary, expensive investigations [37].

Previously “nontypical” forms of positionally induced nystagmus were assumed to always have a central cause. While performing CRPS, Epley observed a sudden change of “typical” torsional posterior canal nystagmus to horizontal direction-changing nystagmus and deduced the nystagmus

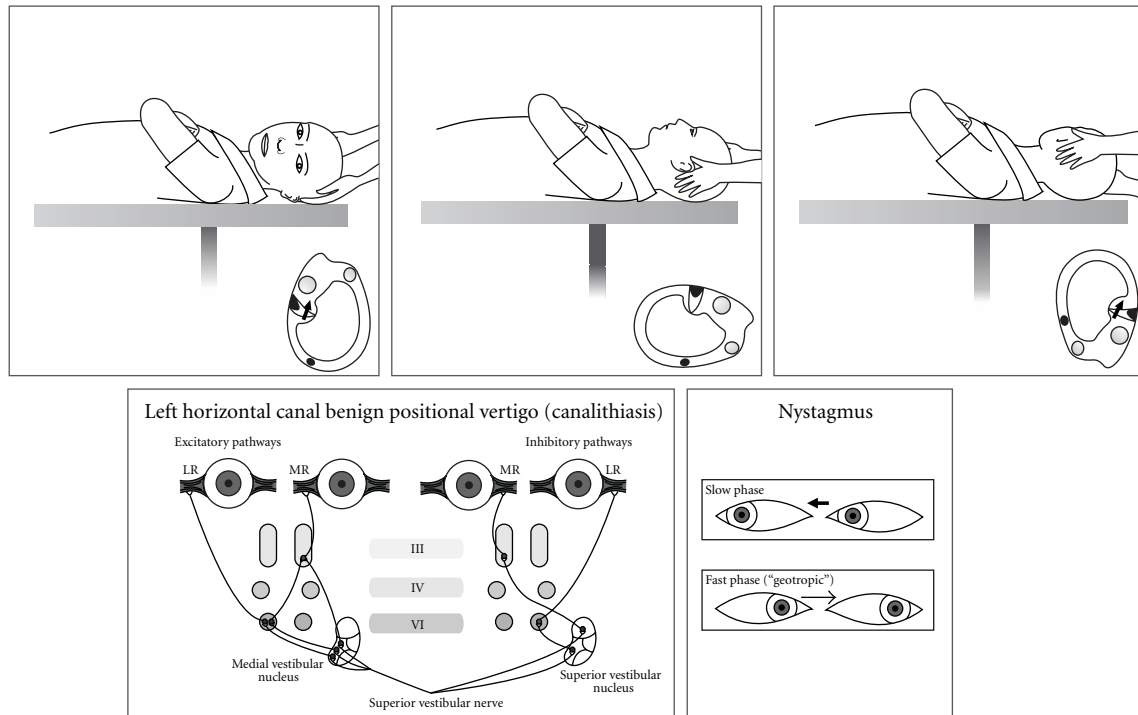


FIGURE 3: Horizontal canal BPPV (canalithiasis) in a left ear showing Head Roll test, inner ear, and receptor connections to the extraocular muscles.

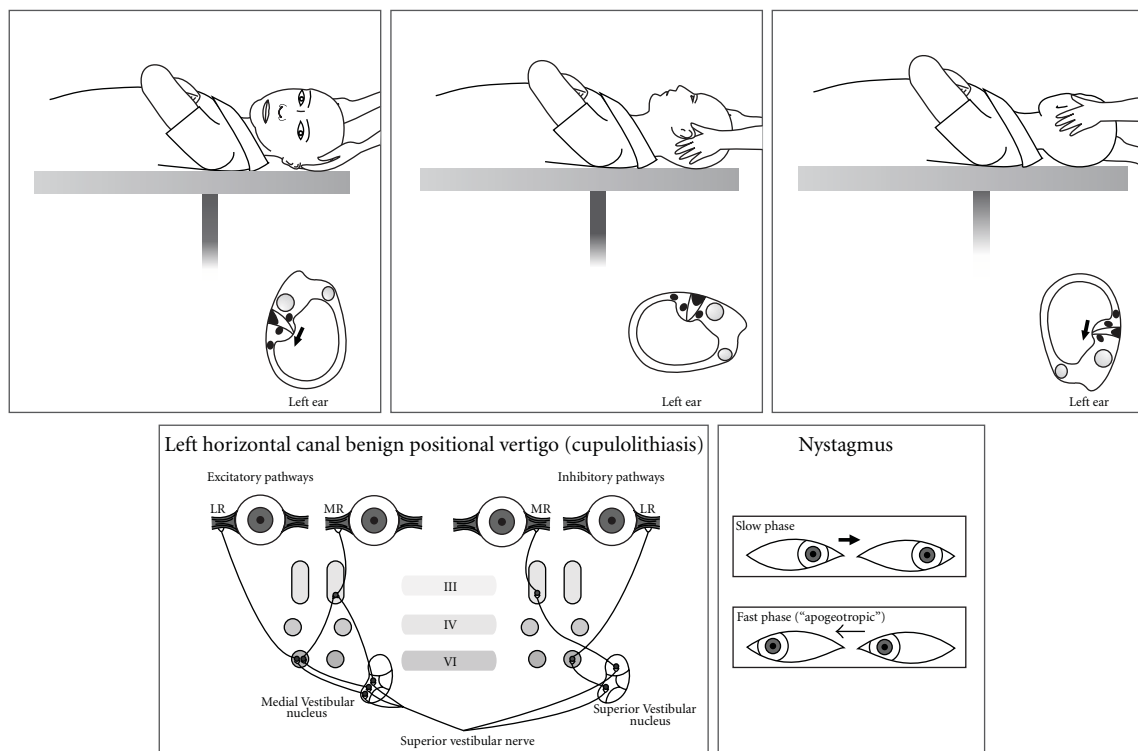


FIGURE 4: Horizontal canal BPPV (cupulolithiasis) in a left ear showing Head Roll test, inner ear, and receptor connections to the extraocular muscles.

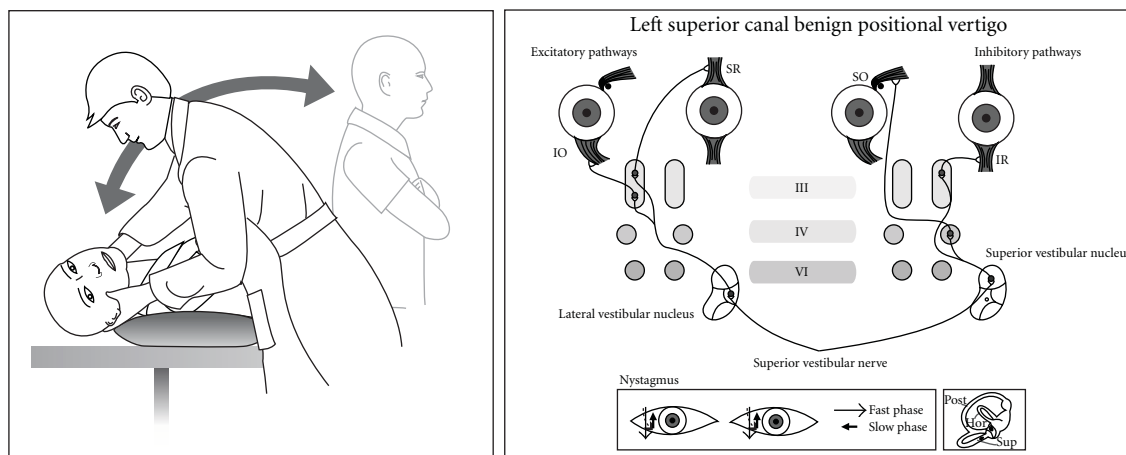


FIGURE 5: Superior canal BPPV in a left ear showing Dix Hallpike test, inner ear, and receptor connections to the extraocular muscles.

that would be caused by otoconia in the horizontal and even the superior canal [38]. Without clinical proof Epley predicted the logical treatment for horizontal canal BPPV would be a 360 degree horizontal plane rotation away from the symptomatic ear. In 1985 McClure [39] had published the electronystagmographic (ENG) traces of seven subjects who had intense positional vertigo and direction-changing horizontal nystagmus when supine. The fast phase was towards the undermost ear (geotropic). McClure suspected a “viscous plug” in the horizontal canal which was causing a piston effect on the horizontal canal receptor. As discovered by Ewald, an ampullopetal (towards the vestibule) cupula deflection is known to cause the most intense nystagmus and vertigo. Horizontal canal BPPV was then reported by others [40–43] and its particularly intense vertigo confirmed. Early repositioning attempts failed [41]. A 270 degree “barbecue” rotation was trialled [44].

These simple horizontal repositioning techniques remain the usual way of treating the horizontal variant of BPPV (Figure 10). Occasionally the most intense nystagmus is away (apogeotropic) from the undermost ear, implying a particle or particles attached to the cupula, or close to it, on its canal or utricular side [43, 45–47]. The cupula becomes “heavy” and is ampullopetal when the symptomatic ear is undermost and ampullopetal when it is uppermost. It can be difficult to ascertain which is the symptomatic ear, but it is likely to be the *undermost ear* which initiates the *least nystagmus* (Figure 4). Horizontal canal BPPV comprises approximately 15% in most series. As for posterior canal it can occur *de novo*, after mild head injury or by “canal conversion” during posterior canal repositioning [45, 46]. It is likely that patients with horizontal canal BPPV inadvertently treat themselves by rolling over in their sleep, if it is in the desirable direction. It they turn in the “wrong” direction they trigger and awake with vertigo.

Although Brandt et al. [48] in 1994 had alluded to “the rare anterior [superior] canal BPPV, the spontaneous symptoms occur when the affected ear is uppermost”, the first detailed description of superior canal BPPV is usually attributed to Herdman and Tusa [49] who documented

two patients whose positionally induced nystagmus was accompanied by downbeat and torsional nystagmus likely to be caused by a superior canal receptor and which ceased after repositioning treatment, implying it was rare form of BPPV. Subsequently superior canal BPPV was recognized and reported by others [50–56] in whose series it accounts for approximately 1% of all BPPV diagnoses. In a review [52] of 50 consecutive patients with positionally induced nystagmus, 75% had a central cause: multiple system atrophy, cerebellar degeneration, and other miscellaneous causes with immediate onset of downbeat nystagmus on a Dix Hallpike test. In 25% (“idiopathic”) a Dix Hallpike test or a head-hanging test elicited downbeat nystagmus with a short latency. In half the subjects a torsional nystagmus could be seen through Frenzel glasses, but in one it was only discernible by video imaging. Aw et al. [54] studied forty-four patients whose BPPV had not responded to conventional repositioning, using 3-dimensional research coils and a 2-axis whole-body rotator. Seven had downbeat nystagmus with a small torsional component, and all responded to a “head-over-heels” forward rotation in the plane of the superior canal. Differences in the ampullary segments of the posterior and superior canals most likely explain why superior canal BPPV downbeat nystagmus can be triggered by a Dix Hallpike test to either side and for its small (or absent) torsional component. In most cases the symptomatic ear is the *uppermost ear* (Figure 5).

## 2. Office Management

The author uses the simplest repositioning techniques.

**2.1. Posterior Canal BPPV.** Diagnosis is by the Dix Hallpike test. Older patients with neck, back, and hip problems require special care, and the test can be more simply done over a pillow (Figures 2 and 5). The patient **MUST** experience vertigo. Occasionally an initial negative test may become strongly positive after the patient does vigorous headshaking.



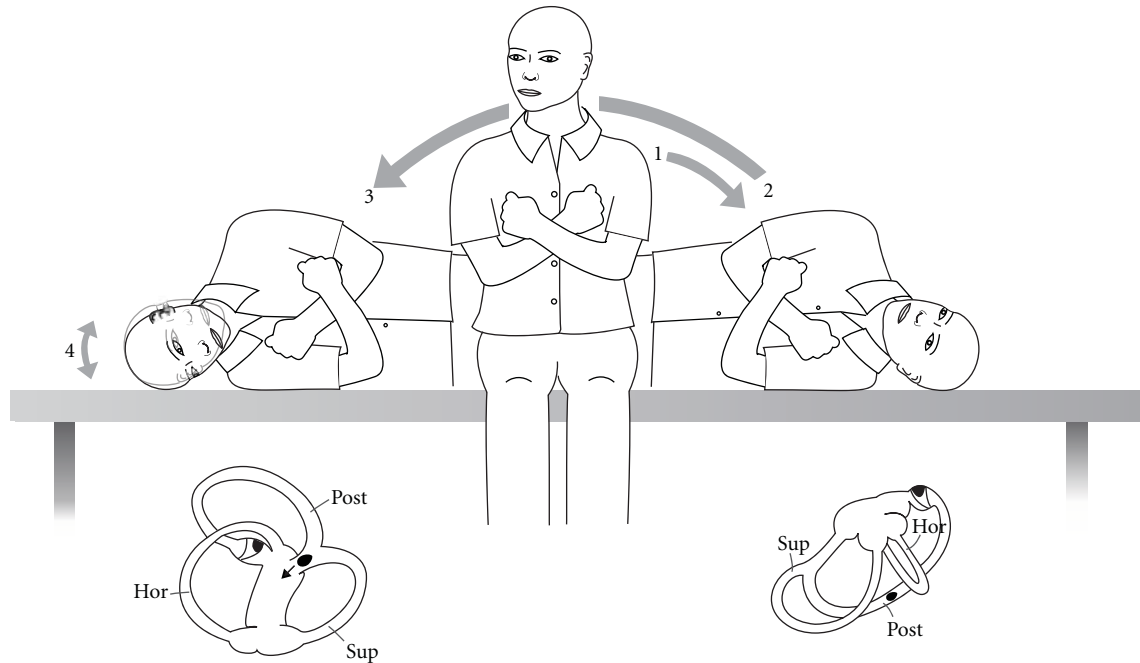


FIGURE 6: Semont maneuver for posterior canal BPPV in a left ear.

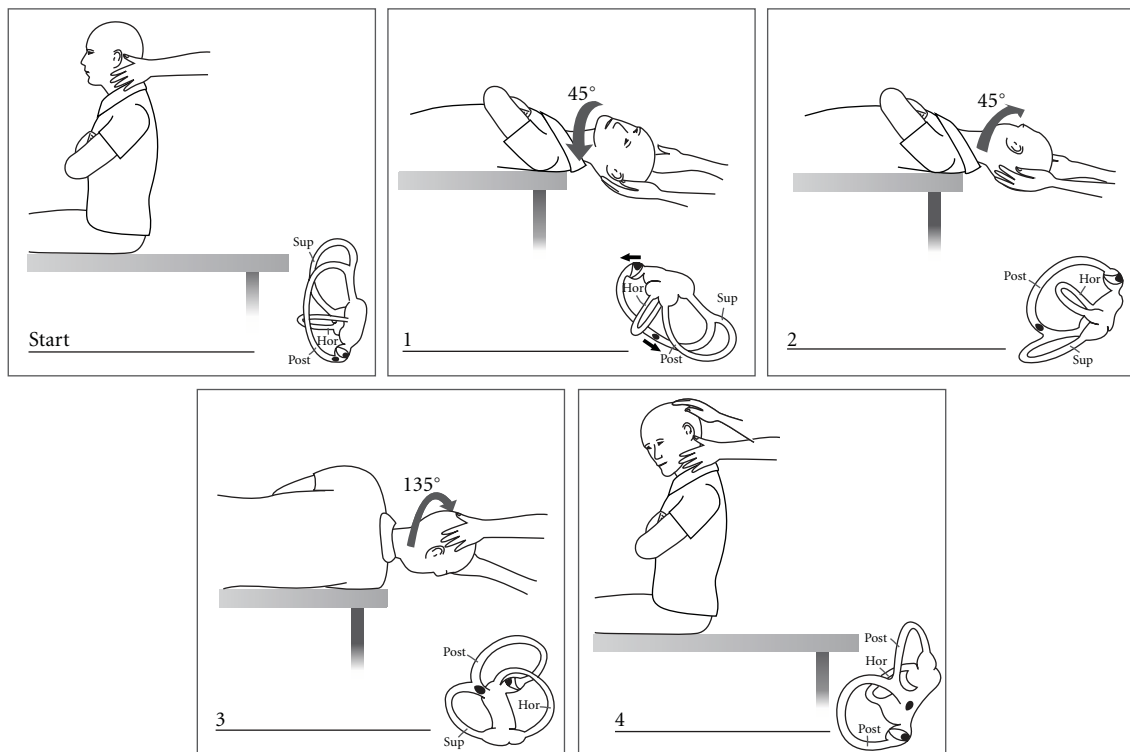


FIGURE 7: Epley canalith repositioning procedure (CRP).

The most common repositioning treatment is Epley's CRP as modified by Parnes with one-minute pauses between head positions (Figure 7). Following the CRP a repeat Dix Hallpike test is done. If positive, the CRP is repeated with mastoid vibration (Hitachi Magic Wand 250 Hz massage

vibrator). If the test is negative, no further repositioning is done. However it is NOT confirmation of treatment success, and retesting should be done. There is no widely accepted interval. One week is a reasonable goal. Younger, agile patients can be shown how to conduct their own

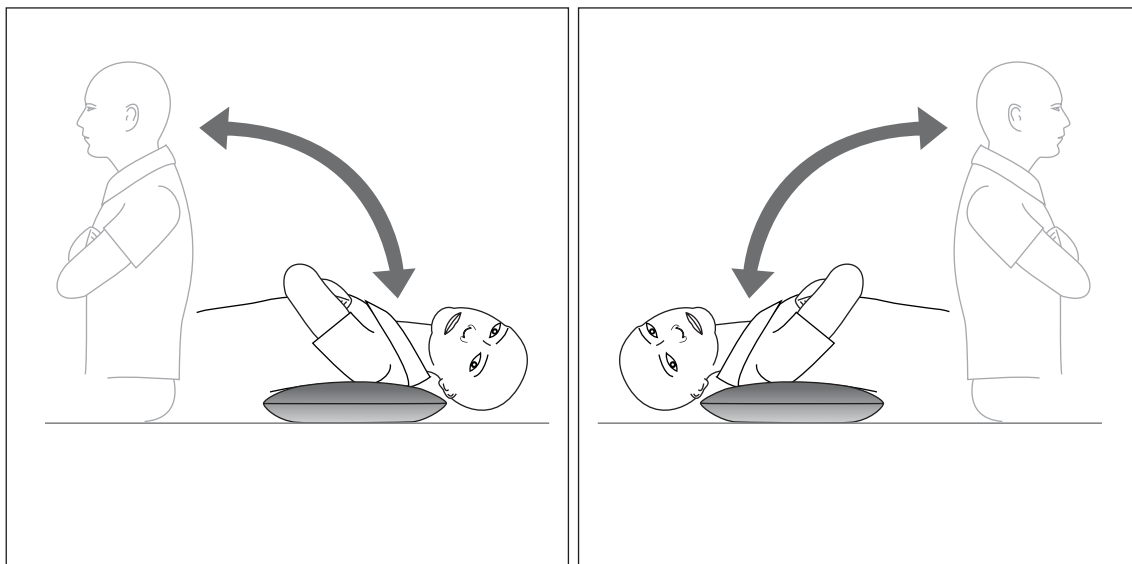


FIGURE 8: Self-testing at home by lying down over a cushion.

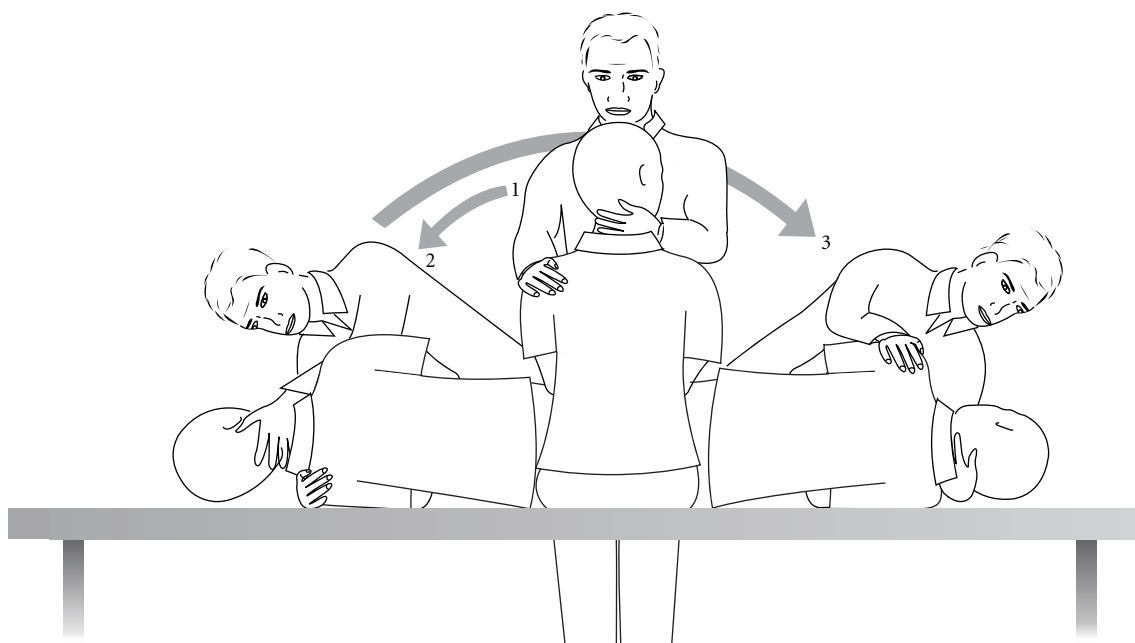


FIGURE 9: Safer technique for performing the Semont maneuver, with the patient moved by a hand under the shoulder and the other hand supporting the neck.

follow-up test at home by lying down over a cushion on the floor (Figure 8). However, older patients, who often report success by avoiding provocative positions, **MUST** be seen and formally retested.

If at followup the Dix Hallpike test is positive, repeat treatment can be by a further CRP with mastoid vibration or by the Semont maneuver. Descriptions of the Semont maneuver typically show the patient held and moved by two hands around the neck. This is extremely inappropriate for many individuals who are larger, obese, or who have

neck problems. A safer technique is for the physician to rapidly move the patient from side to side by a hand under the downmost shoulder (symptomatic ear) and the other supporting the neck (Figure 9).

A Cochrane collaboration review of the Epley CRP and 5 subsequent random controlled trials found a significant success compared with nontreated controls [57, 58]. Comparison of trials is confounded by variation in the number of CRP cycles used per treatment, clinician experience, and the treatment setting. There have been relatively few trials [59, 60]

confirming the efficacy of the Semont maneuver compared with sham treatment. The Semont maneuver is the most logical first treatment for a patient with posterior canal cupulithiasis (immediate nystagmus onset) with attached otoconia more likely to be dislodged by a centrifugal force.

Epley initially recommended that after a CRP the patient should sleep propped up for two nights to prevent repositioned particles from returning [28]. However, numerous studies have not shown any advantage from posttreatment restrictions [61, 62]. The use of adjunctive mastoid vibration has remained contentious, probably because of the power range of the devices used [63].

The vast majority of BPPV treatment studies have been performed in specialist practice settings. While very few patients can or even wish to administer self-treatment, it is an understandable goal. Self-administered CRP at home after initial office CRP achieved a slightly greater improvement [63]. As an adjunct to self-treatment the newly released “DizzyFix” dynamic visual device (Clearwater Clinical) [64] significantly improved the performance of volunteers learning Parnes’ modification of the CRP (Figure 10). It is a useful teaching tool on correct CRP technique for patients and health professionals.

**2.2. Horizontal Canal BPPV.** Horizontal canal BPV is most likely to be discovered as the patient is undergoing a Dix Hallpike provocative test [46, 47]. Occasionally it suddenly becomes apparent (“canal Conversion”) after a CRP, with brisk horizontal-rotatory nystagmus. If there is pillow under the shoulders, it is removed and the patient is moved down the examination couch so that the head is midline and in the horizontal plane. Then the head is gently turned to one side and then the other (“head roll” test). Usually there is a clear, repeatable pattern of brisk nystagmus towards one undermost ear (maximum geotropic) and then weaker nystagmus (apogeotropic) when the opposite ear is down. If a “canal conversion” has occurred, the symptomatic ear is already known. The vertigo tends to be more intense than for posterior canal BPPV, and some patients become nauseated and require an antiemetic. Once the symptomatic ear has been identified, the mechanism and its different repositioning in the horizontal plane (“barbecue” repositioning) are explained to the patient (Figure 11). With the examiner seated at the head of the examination couch the patient is asked to rotate 360 degrees in four stages, a minute apart. At the third position the patient should be resting on the elbows with the neck flexed, so that the horizontal canal is vertical, which is where the particle will exit the canal if it has been successfully moved. The head roll test is repeated and, if negative, treatment ceases.

If on the head roll test the nystagmus is apogeotropic, the likely mechanism is cupulolithiasis in the undermost ear with the *least* nystagmus. The particle(s) could be on either side of the cupula. On the presumption it is on the canal side the standard direction rotation is carried out. If unsuccessful (likely vestibule side) rotation is done in the opposite direction. Additional headshaking or mastoid vibration can be used. Sometimes apogeotropic nystagmus



FIGURE 10: “DizzyFix” dynamic visual device for teaching the modified Epley CRP to patients and health professionals. It is not a model of the semicircular canals but a representation assisting accurate head positioning and appropriate timing for a posterior canal particle to be successfully expelled.

reverses to geotropic, implying that an attached particle(s) has become free. If there is not a clear pattern, or if the patient becomes very nauseated, it is advisable to retest on another day. Central pathology must be kept in mind.

**2.3. Superior Canal BPPV.** On a Dix Hallpike test if there is downbeat nystagmus superior canal, BPPV is a possibility. If the cause is central, the nystagmus onset is immediate, and the patient does NOT experience vertigo. If it is BPPV, there will be a latency of onset and the patient MUST experience vertigo. Any torsional component may be imperceptible to the naked eye. Repeating the test with the head lower (“head-hanging” test) than usual may intensify the response. The particle may be in either ear but most likely in the uppermost ear. On these assumptions there are two simple “office” treatments.

The first is the Epley CRP performed with “head hanging” and commencing with the suspected ear *uppermost*. The second is the Li maneuver [65] where the patient is moved rapidly from a supine (midline) head-hanging position to a face-down position at the opposite end of the couch (Figure 12).

**2.4. Recurrences, Failed Office Treatment, and Complications.** Reported rates of spontaneous complete resolution of BPPV at a month range from 20% to 80%. The American Academy of Otolaryngology, Head and Neck Surgery Clinical Practice Guideline, Benign Paroxysmal Positional Vertigo recommends that physician retesting at one month after repositioning treatment should be the standard interval after treatment [58].

Patients treated for BPPV should be told that there is a likelihood of recurrences. Most trials involve a short follow-up period. In trials with longer followup the recurrence rate at one year is estimated at 15% [66] and 37%–50% at 5 years on the Kaplan-Meier curve [66, 67]. Posttraumatic BPPV

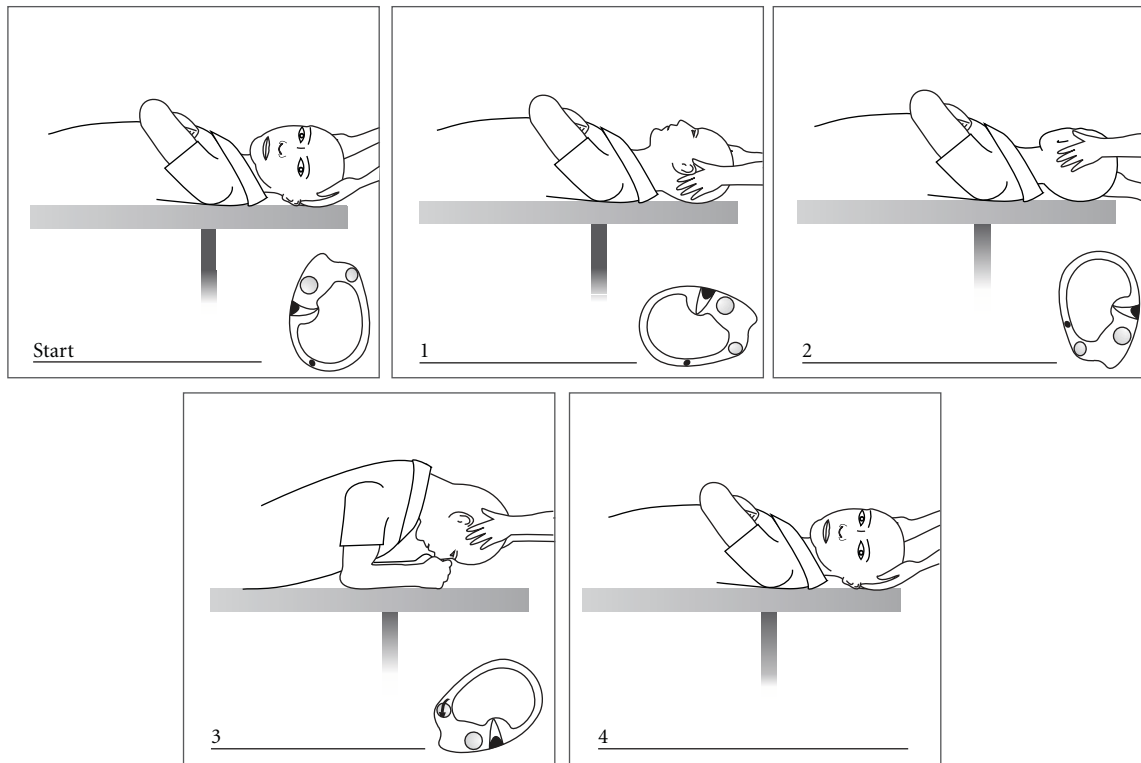


FIGURE 11: “Barbecue” repositioning for horizontal canal BPPV in a left ear.

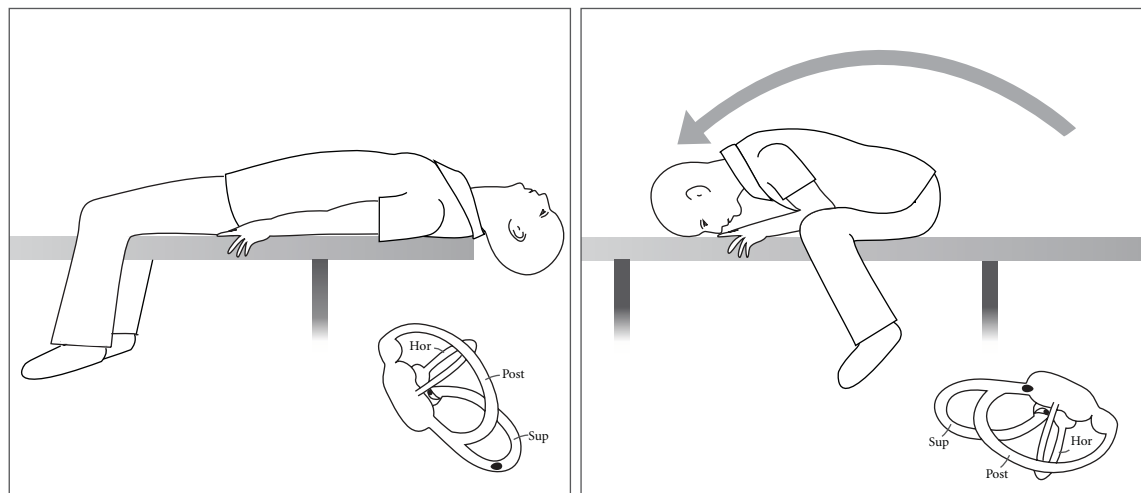


FIGURE 12: The Li manoeuvre for superior canal BPV in either ear (left ear).

may have a higher recurrence rate than spontaneous BPPV [68].

The most common “complication” of BPPV repositioning treatment is canal conversion. Considering the population age in which it is usually performed there is a surprising sparsity of literature on cervical spine and neurological complications [69].

**2.5. Central Pathology.** Central nervous system disorders can masquerade as BPPV, in particular intracranial tumours

[52, 70, 71] and migraine. Nystagmus features which strongly suggest a neurological cause are downbeat nystagmus, direction-changing nystagmus without a change in head position, nausea with up or downbeating nystagmus, and preexisting and continuing nystagmus. The cardinal distinguishing feature from BPPV nystagmus is that the patient does NOT experience brief rotational vertigo. Therefore patients with such nystagmus or symptoms of BPPV not showing resolution after repositioning require neurological examination and MRI scanning of the brain and posterior fossa.

Migraine is now a well-recognised cause of recurrent vertigo [72]. During an episode positional testing can elicit upbeat torsional or horizontal nystagmus similar to BPPV [73, 74]. Features supporting migraine as a cause are headache, absence of brief acute vertigo induced by the Dix Hallpike test, disappearance of all nystagmus within days, and a recurrent pattern.

### 3. Future Directions

Currently Epley's canalith theory explains most of the features of BPPV: the latency and type of nystagmus, according to the involved canal, and the logic and efficacy of repositioning treatments. However, as for many other inner ear disorders, certain details of its pathophysiology, in particular spontaneous recovery, remain elusive, largely due to the inability to internally image the inner ear in enough detail and the prior reliance on histological techniques. Increased knowledge of human otoconial physiology and pathology will be important.

As episodes of BPPV recover without treatment, it is reasonable to assume that otoconia exit a canal during normal head movement, particularly in horizontal canal and superior canal BPPV. It has been demonstrated that frog otoconia rapidly dissolve in endolymph with physiologic calcium levels, but more slowly if the calcium level is raised [75]. Therefore a major reason for spontaneous recovery is the ability of normal endolymph to dissolve otoconia if they do not return to the utricle. In mammals otoconia are calcite crystals of calcium carbonate. In rats scanning electron microscopy (EM) shows a progressive degeneration of otoconial structure in the oldest rats [76], a phenomenon consistent with the common age range in BPPV. In female rats made artificially oostepenic/oosteporotic, scanning EM shows ultrastructural changes in otoliths [77] suggesting that there could be relationship between bone biochemistry and recurrent BPPV in older women. Seventy-five percent of thirty-two women aged between 50 and 85 years who had BPPV were shown to have osteopenia or osteoporosis compared with eighty-three healthy controls [78].

Measurement of vestibular-evoked myogenic potentials (VEMPs) is new electro-physiological technique for measuring saccule and otolith function. In some patients with vestibular neuritis absence of the cervical VEMPs (c-VEMP) on the side of the labyrinthitis predicted the nondevelopment of BPPV which only occurred if the cVEMP was present. More recent studies on the "health" of the otolith organs using c-VEMPs [79, 80] have shown significantly large number of prolonged responses in patients with BPPV compared with healthy controls, suggesting neuronal degeneration of the saccule. In contrast newer ocular VEMPs (o-VEMP) also measure utricular function and are likely to be more relevant in establishing otolith dysfunction and recovery in BPPV. Distortion of the visual horizontal due to ocular torsion occurs in utricular dysfunction, as in vestibular neuritis. Measurement of the visual horizontal in BPPV patients was abnormal in 42% before-treatment, 15% after repositioning and in 8% two weeks later suggesting initial

utricular dysfunction and its possible restoration from the return of otoliths [81]. Encouraging advances in imaging may eventually enable *in vivo* correlation. Three-dimensional T2-weighted 3-dimensional fast MRI imaging with steady-state acquisition sequences can now show some reliable detail of semicircular structure such as narrowed areas and "filling defects" [82]. In patients with "intractable" BPPV 89% had abnormal canals compared with healthy controls, but there was no correlation between the affected canal and nystagmus type. Subtle variations in canal diameter, length, and width may correlate with a predisposition to BPPV and to treatment failures.

Finally, the American Academy of Otolaryngology, Head and Neck Surgery Clinical Practice Guideline on BPPV [58] has recommended sixteen aspects meriting further research, including the true prevalence and burden of untreated BPPV in older adults, the natural history of untreated BPPV, agreed endpoints for clinical trials, and importantly the functional impact of BPPV on work safety and the rates of falls it may account for in the elderly.

### Conflict of Interests

The author declares that there is no conflict of interests.

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