

Anterior semicircular canal benign paroxysmal positional vertigo and positional downbeating nystagmus

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Abstract

Purpose: The aim of this study was to describe the clinical features and video-oculographic findings in patients with anterior semicircular canal benign paroxysmal positional vertigo (BPPV).

Materials and methods: *Study Design.* This is a prospective case series. *Setting.* The study was set at an outpatient clinic in a general hospital. *Patients.* Fourteen individuals with symptoms of BPPV and positional downbeating nystagmus (pDBN) were included in the study. The diagnosis was based on a history of brief episodes of vertigo and the presence of pDBN confirmed in the video-oculographic examination during Dix-Hallpike test (DH) or head-hanging maneuver. *Intervention.* Patients were treated by particle repositioning maneuver and the effectiveness was evaluated at 7, 30, and 180 days posttreatment. The treatment was repeated up to 4 times if pDBN was persistent. *Main Outcome Measures.* The main outcome measure is the number of patients without pDBN at 30 and 180 days.

Results: Video-oculography showed a predominant pDBN in response to DH. Of the 14 patients, 7 had arterial hypertension, and 5 of 14 cases presented abnormalities on the caloric test. Horizontal spontaneous nystagmus was found in 3 of 14 individuals. Positional nystagmus at different positional test was observed in 5 of 14 individuals, suggesting the involvement of several canals. Of the 14 patients, 10 (71%) did not present vertigo, and the positional tests were negative at 30 days. However, 3 cases presented a positive DH with persistence of BPPV episodes and pDBN at 30 days, and another developed a contralateral posterior canal affection. One of the patients maintained a persistent pDBN at 180 days despite the repeated maneuvers.

Conclusions: Video-oculography demonstrates that anterior canal BPPV is characterized by a predominant downbeating nystagmus in response to DH. These individuals may show alterations in the vestibular caloric, and they can have multicanal affection.

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1. Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular organ disorder that presents an easy and effective treatment [1,2]. Benign paroxysmal positional vertigo is defined by spells of vertigo and positioning nystagmus of short duration that are elicited by the turn of the head and produces the stimulation of the semicircular canal [3]. This situation leads to the production

of abnormal vestibulo-ocular reflexes, causing vertigo and nystagmus with distinctive characteristics depending on the canal affected. There are several clinical variants, posterior, horizontal, and anterior canal, the most frequent being the involvement of the posterior canal (PC) [4,5]. The diagnosis of PV BPPV is based on the observation of a characteristic positional nystagmus during the Dix-Hallpike test (DH) [6,7].

The affection of the horizontal or lateral semicircular canal is less common, and it represents between 5% and 16% of the cases of BPPV [3,8]. Different studies had described large series of cases [9–11] differentiating 2 mechanisms (canalithiasis or cupulolithiasis) that explain the features of the positional nystagmus (geotropic or apogeotropic, latency,

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and duration). The intraoperative demonstration of the presence of otoconia in the lumen of the semicircular canals has been accepted as the underlying mechanism to explain BPPV and the positional nystagmus [12,13]. Vestibular lithiasis can theoretically affect any of the 3 semicircular canals, and otoliths can be found free-floating in the canal (canalolithiasis) or found adhered to the cupula of the crista ampullaris (cupulolithiasis) [14].

The anterior canal variant of BPPV is considered a rare form, and it is characterized by a predominant downbeating nystagmus (pDBN) with a small torsional component in response to DH [4,15]. The anterior canal projects to the ipsilateral superior rectus muscle and to the contralateral inferior oblique muscle; the nystagmus is downbeating and torsional during the DH. If the downside ear is affected, the direction of the torsional component will be the same as in contralateral PC BPPV [15]. This variant has been described in occasional reports estimating a frequency between 1% and 11% [3,4,16]. A recent study that clarified the clinical significance of pDBN in cerebellar disorders reported a series of 12 patients with possible anterior semicircular canalolithiasis [17]. Moreover, the parameters of benign positional nystagmus, including 7 patients with anterior canal BPPV and pDBN, have been measured with 3-dimensional, dual-search scleral coils in a 2-axis, whole-body rotator [15].

In this report, a series of 14 cases of BPPV with pDBN that were compatible with the diagnosis of anterior canal BPPV is presented. Individuals were selected to define their clinical features and the video-oculographic (VOG) parameters of the nystagmus.

2. Patients and methods

2.1. Patients

Individuals were outpatients that reported a history of vertigo or dizziness. Eighty patients with BPPV and positional nystagmus were diagnosed during the years 2003 to 2004. Physical examination included otoscopy, Rinne test, Weber's test, pure tone audiometry, and a basic neurotologic examination (oculomotor, saccades, head-impulse test, cranial nerve examination, Romberg's test, Bárány's test, and Fukuda test). Clinical diagnosis was carried out by positional testing: standard DH maneuvers were used to stimulate vertical canals [18] and head-hanging maneuver was used for anterior canal, as previously described [16]; while patients were in a supine position, horizontal (yaw) rotations of the head were used to stimulate horizontal canals.

2.2. Inclusion and exclusion criteria

The inclusion criteria for diagnosis of anterior canal BPPV were (1) a positional test (DH to each side or the head-hanging maneuver) after a short latency period that produced vertigo that simulated the patient's symptoms and simulta-

neous downbeating nystagmus (DBN); (2) the nystagmus and vertigo habituate with the positional test; and (3) no evidence of central nervous system (CNS) disease.

Differential diagnosis between posterior and anterior canal involvement was based on the direction of the vertical component of the fast phase of the nystagmus response during DH, being upward in cases of PC involvement and downward in cases of anterior canal disease [16]. Patients with vertical nonpositional nystagmus (ie, spontaneous DBN) were excluded from this study. All the individuals were evaluated by a cranial magnetic resonance imaging with gadolinium to rule out a CNS disease.

Written information was facilitated to the patients, and an informed consent was obtained for all the individuals after explaining the treatment. The Ethical and Research Committee of the hospital approved this research study.

2.3. Video-oculographic examination

The VOG recording included spontaneous nystagmus, head-shaking nystagmus, positional testing (DH, horizontal rotations of the head in supine and head-hanging maneuver for the anterior canal) and water bithermal caloric testing. The camera was adapted in the contralateral eye to the side of testing to minimize the movement of the camera during DH. Caloric test by using a Variotherm Plus model water irrigator (Atmos, Berlin, Germany) was performed on each subject, with a water flow of 250 mL/20 s at 30°C and 44°C with an interval of 10 minutes between successive irrigations. All irrigations were performed with the eyes closed in the Hallpike position.

The recording was performed by a VOG 2-dimensional system with an infrared charge-coupled device camera connected to the software (SMI, Berlin, Germany) to analyze the eye response. Maximum slow-phase velocity (SPV) was determined by the software, and SPV values were compared with normal values of our laboratory. Eye position in the orbit was controlled during testing because a movement of the eyeball strongly influences the SPV and the direction of positioning nystagmus.

2.4. Treatment

All patients were initially treated by a particle repositioning maneuver (PRM) or Epley's modified maneuver without mastoid oscillator; individuals were recommended to avoid fast head turnings, and they were allowed to sleep in decubitus position. Patients were evaluated at 7 days posttreatment, and if they presented a positional nystagmus, PRM was repeated up to 4 times. Nonrespondent individuals were treated by a Semont maneuver and finally with Brandt-Daroff exercises.

3. Results

The mean age of the 14 individuals was 56 ± 17 (median, 53; range, 20–81) years, being 5 men and 9 women. Nine patients presented idiopathic BPPV, and 5 cases presented

after minor head trauma. Seven individuals (50%) had arterial hypertension, and 3 of 14 had a history of ischemic heart disease, one of them having received a coronary bypass. Five women had a history of depressive disorder, and 3 of them also presented migraine. The time course for BPPV was variable (range, 1–240 months). Of the 14 patients, 6 presented unilateral sensorineural hearing loss (from 35 to 85 dB), which was correlated with the affected side in 5 of 6 cases, and 1 woman (patient 13) had a bilateral symmetrical sensorineural hearing loss. None of the individuals was unemployed or retired because of BPPV. The clinical features of patients with BPPV and pDBN are shown in Table 1.

3.1. Video-oculographic examination

Vestibulo-ocular testing found a horizontal spontaneous nystagmus in 3 of 14 individuals. Caloric test showed an ipsilateral horizontal canal paresis in 4 of 14 cases (>25%) and a directional preponderance in 2 of 14 (>25%), so 5 of the 14 individuals presented an abnormal caloric test.

The VOG recording demonstrated a pDBN during right DH in 5 of 14 cases, 3 cases being observed during left DH. Three cases presented pDBN during left and right DH and the head-hanging maneuver (patients 2, 4, and 13). The head-hanging maneuver produced pDBN in 5 cases with an increase of the maximum SPV in the response. Fig. 1 shows the eye movement recordings in patient 2 during the head-hanging maneuver. In these cases, the number of beats and

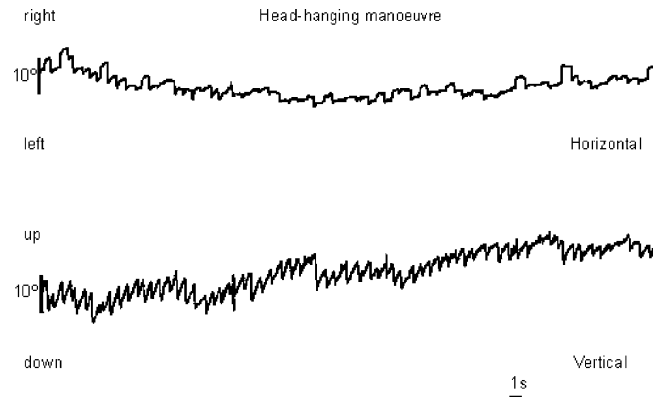


Fig. 1. Video-oculographic recording of pDBN in a patient with suspected anterior canal BPPV during the head-hanging maneuver. Note that main component is downbeating (55 beats per minute; SPV, 9.1°/s). This patient had a spontaneous left nystagmus that was also recorded during the positional test.

the maximum SPV of the pDBN were higher with the head-hanging maneuver than with the DH. The positional nystagmus suggesting a multicanal affection was observed in 5 of 14 of cases. Patient 3 presented a left lateral canal and right anterior canal BPPV, and patient 6 showed a right PC followed by pDBN. After 30 days of follow-up, this patient presented a left PC and right anterior canal during DH, with a good response to PRM.

Table 1
Clinical summary of patients with pDBN

No.	Sex	Age	Clinical history	Head trauma	Time course (mo)	Sensorineural hearing loss	Canal paresis ^a	Directional preponderance ^b	Diagnosis
1	M	68	HT, heart disease	No	6	L 50 dB	No	No	R AC BPPV
2	F	55	HT, depressive disorder, splenectomy	No	36	No	R (36%)	R (54%)	R + L AC BPPV
3	M	51	HT	No	2	No	No	No	L LC + R AC BPPV
4	F	65	HT, heart disease	No	60	R 50 dB	R (38%)	No	R AC BPPV
5	M	20		Yes	12	No	No	No	R + L AC BPPV
6	F	59	HT	No	240	No	No	No	R PC followed by pDBN, on follow-up R AC + L PC
7	F	52	Diabetes, oculomotor paresis right eye	Yes	1	No	R (26%)	No	L AC BPPV
8	M	81	HT, prostate cancer	No	12	R sudden 70 dB	No	L (37%)	R AC BPPV
9	F	38	Migraine, depressive disorder	No	3	No	No	No	R AC BPPV
10	M	54		No	150	No	No	No	L AC BPPV
11	F	43	Migraine, depressive disorder	Yes	13	L 45 dB	No	No	L AC BPPV
12	F	49	Migraine, depressive disorder	No	7	R 85 dB	L (32%)	No	R AC BPPV
13	F	74	Heart disease, depressive disorder	Yes	120	R + L 50 dB	No	No	L AC BPPV
14	F	80	HT	Yes	7	R 45 dB	No	No	R + L AC BPPV

M indicates male; HT, hypertension; L, left; R, right; AC, anterior canal; F, female; LC, lateral canal.

^a On caloric testing, more than 25%.

^b More than 30%.

Table 2
Features of pDBN in patients with BPPV

No.	Position	Vertical component	Horizontal component	Latency (s), habit ^a	Duration (s)	No. of nystagmus	Vertical component velocity (°/s)	Horizontal component velocity (°/s)
1	R DH	Down	No	32, habit	22	8	8.2	0
2	R + L DH, head-hanging	Down	No	1, partial habit	18	10	13.7	0
3	R DH	Down	Right	1, habit	58	53	4.2	5.7
4	R DH	Down	Right	1, partial habit	56	53	11.6	5.7
5	R + L DH, head-hanging	Down	No	8, partial habit	7	8	15.1	0
6	R + L DH	Down	No	24, habit	28	42	7	0
7	L DH	Down	No	20	15	21	5.1	0
8	L DH	Down	No	4	7	8	9.5	0
9	R DH	Down	No	2	8	7	13.1	0
10	L DH	Down	No	3	50	18	23.7	0
11	L DH, head-hanging	Down	Left	7, habit	58	15	22.4	5.3
12	R DH, head-hanging	Down	No	6	54	45	17.3	0
13	R + L DH, head-hanging	Down	Left	10, habit	50	87	29.1	14.5
14	R + L DH, head-hanging	Down	Left	22, habit	30	26	21.1	14.4

^a Habituation, that is, attenuation of the nystagmus on repeated positional maneuvers.

Parameters of pDBN for each patient are shown in Table 2. The nystagmus had a latency of 20 to 32 seconds in 3 patients, a brief latency of 3 to 10 seconds in 6 cases, and appeared immediately in 4 cases. The nystagmus adapted (<1 minute) in all but one patient. All patients reported vertigo that habituated with repeated positional maneuvers.

3.2. Effect of repositioning in anterior canal BPPV

Of the 14 patients, 10 (71%) did not present vertigo, and the positional tests were negative at 30 days. However, 3 cases presented a positive DH with persistence of BPPV episodes and pDBN at 30 days, and another developed a contralateral PC affection. Although BPPV episodes were well tolerated, 1 of the 14 patients showed pDBN at 180 days despite the treatment with several PRM, Semont maneuver, and Brandt-Daroff exercises.

4. Discussion

This study presents a series of patients with BPPV and pDBN without evidence of a CNS abnormality, and provides new neuro-otologic information that can be useful to define anterior canal BPPV. Anterior canal BPPV is characterized by a predominant vertical DBN with only a small torsional component that can be either geotropic or apogeotropic in response to DH and/or the head-hanging maneuver [15,17].

The BPPV with pDBN is observed in CNS diseases, particularly in posterior fossa lesions [19,20]. No patient in our series had an Arnold-Chiari malformation, one of the most common causes of spontaneous DBN or multiple system atrophy. Multiple system atrophy has 3 main presentation forms, one with atypical parkinsonism (striatonigral type), one mostly cerebellar (olivopontocerebellar type), and one with mainly autonomic nervous system symptoms (Shy-Drager type) [21]. However, all individuals

in our series did not show any abnormalities in the cranial magnetic resonance imaging.

The VOG recording of the pDBN demonstrated a consistent down vertical component in all cases that were compatible with the diagnosis of anterior canal BPPV. The major limitation of this study is the lack of measurements of the torsional component of the pDBN. In a previous report with 4 patients, the fast component nystagmus was in-torsional and down in the ipsilateral eye, and was out-torsional and mainly down in the contralateral eye [4]. Unfortunately, our 2-dimensional VOG system only can measure horizontal and vertical components of nystagmuses.

Although there have been occasional reports of suspected anterior canal BPPV, these were not always concordant. During PRM of PC BPPV, transient pDBN has been found and attributed to invasion of the anterior canal by the canalicular debris [22]. Rahko [23] reported a series of 57 individuals with BPPV of the superior canal. They were all posterior and horizontal canal BPPV that had residual symptoms and dizziness after the treatment. A new test for diagnosis and a new maneuver were described for the anterior canal, but unfortunately, the eye movements during the procedures were not reported [22].

The introduction of infrared video goggles has provided a significant advancement for monitoring nystagmus in BPPV, and the pattern of positional nystagmus can localize the canal affected. Nakayama and Epley [24] has reported a series of 833 cases of BPPV with 62% PC, 9% horizontal canal, 20% multiple canal, and 2% anterior canal affected. A VOG nystagmus-based strategy was used for diagnosis and treatment by repositioning in this series. Three-dimensional VOG recording has shown a vertical DBN with a small ipsilateral torsional beating component, as expected for the anterior canal [17]. The explanation for this small torsional component is based on the smaller projection onto the roll plane of the anterior canal [25]. Because the torsional gain

of the human vestibular ocular reflex is about 0.75 in response to high-frequency (2 Hz) roll head impulses [26], the torsional component would be smaller than the horizontal and vertical components, and this can explain the clinically observed pDBN [15].

The abnormal caloric vestibular tests were found in some cases in our series (4 cases with unilateral canal paresis and 2 cases with increased directional preponderance). These findings in caloric testing are similar to those observed in patients with PC BPPV [3,27]. Moreover, horizontal spontaneous nystagmus was found in 3 of 14 individuals. These individuals may show abnormalities in the caloric test.

The natural history of BPPV is incompletely understood. Although spontaneous resolution of symptoms is observed in PC BPPV, it is also clear that patients reporting symptoms with a negative DH became positive DH after several months of follow-up, suggesting a relapsing course. It is not known whether repositioning can alter the natural history of recurrences of BPPV. Furthermore, the abnormalities on vestibular tests found in some patients with BPPV can explain the relapsing course of the disease because the causes that facilitate the shedding of otoliths from the utricular macula probably persist.

In our series, 7 of the 14 patients had arterial hypertension, suggesting that microvascular affectation could be associated with anterior canal BPPV. As it has been demonstrated, giant cell arteritis was associated with BPPV [28].

We support that BPPV is a complex disorder that can recur despite of short-term effectiveness of the PRM. In this series, the positional nystagmus indicating possible multi-canal affectation was observed in 5 of 14 cases, and those patients with recurrent episodes can sometimes demonstrate a new positional nystagmus, suggesting a change of the canal affected. Bilateral pDBN triggered by the DH was observed in 5 cases (2, 5, 6, 13, 14), being difficult to localize the affected side, so this confirms that the head rotation is unlikely to be critical for particle mobilization in anterior canal lithiasis [16]. The only difference in the left and right DH is that, during a contralateral DH, the head rotates in the plane of the affected anterior canal, whereas during the ipsilateral DH, the head rotates orthogonally to the plane of the canal. These bilateral cases could be suspected to be a bilateral PC BPPV, but here, the direction of the vertical component recorded is essential for the differential diagnosis.

Five individuals in our series had pDBN by the straight head-hanging maneuver (2, 5, 11, 12, 13). As discussed previously, the rotation in the canal plane is of less relevance than the final low head-down position [16]. During the DH, the head is rotated 45° in the horizontal plane previously, so the head cannot reach such low vertex position as with the head-hanging maneuver. This additional head angle may be crucial for provoking anterior canal BPPV as the ampullary segment will approach a vertical down-pointing position. Although cervical ankylosis or obesity can difficult the

head-hanging maneuver, we should perform it in patients with BPPV and a negative DH to rule out a pDBN.

The lower occurrence of lithiasis in the anterior canal is attributed to the anatomical features of the labyrinth. The debris within the anterior canal should be self-clearing, mainly because the posterior arm of the anterior canal descends directly into the common crus and the utricle. Persistence of pDBN may be explained in 2 ways. First, the canalith is incompletely returned into the utricle by a single PRM and some particles remain in the PC. Why this occurs is unknown, but it could be possible that the canalith may be too large, a failure of disaggregation or a stenotic common crus. This is consistent with the finding that some patients require a second or third PRM to become DH negative [29]. Second, another vestibular lesion (ie, ischemic, metabolic) that causes a continuous formation of debris in patients with persistent positional nystagmus may exist.

5. Conclusions

Video-oculography demonstrates that anterior canal BPPV is characterized by a predominant DBN in response to DH. These individuals may show alterations in the caloric response, and they can have multicanal involvement in same cases.

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References

- [1] Epley J. The canalith repositioning procedure for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1992;107:399-404.
- [2] Parnes L, Price-Jones R. Particle repositioning maneuver for benign paroxysmal positional vertigo. *Ann Otol Rhinol Laryngol* 1993; 102:325-31.
- [3] Brandt T. Positional and positioning vertigo and nystagmus. *J Neurol Sci* 1990;95:3-28.
- [4] Honrubia V, Baloh RW, Harris MR, et al. Paroxysmal positional vertigo syndrome. *Am J Otol* 1999;20:465-70.
- [5] Korres S, Balatsouras DG, Kaberos A, et al. Occurrence of semicircular canal involvement in benign paroxysmal positional vertigo. *Otol Neurotol* 2002;23:926-32.
- [6] Dix MT, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Ann Otol Rhinol Laryngol* 1952;61:987-1016.
- [7] Baloh RW, Sakala SM, Honrubia V. Benign paroxysmal positional nystagmus. *Am J Otolaryngol* 1979;1:1-6.
- [8] Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). *Can Med Assoc J* 2003;169:681-93.
- [9] McClure JA. Horizontal canal BPV. *J Otolaryngol* 1985;14:30-5.
- [10] Baloh RW, Jacobson K, Honrubia V. Horizontal semicircular canal variant of benign positional vertigo. *Neurology* 1993;43:2542-9.

- [11] Nuti D, Vannucchi P, Pagnini P. Benign paroxysmal positional vertigo of the horizontal canal: a form of canalolithiasis with variable clinical features. *J Vestib Res* 1996;6:173-84.
- [12] Parnes LS, McClure JA. Posterior semicircular canal occlusion in the normal hearing ear. *Otolaryngol Head Neck Surg* 1991;104:52-7.
- [13] Welling DB, Parnes LS, O'Brien B, et al. Particulate matter in the posterior semicircular canal. *Laryngoscope* 1997;107:90-4.
- [14] Brandt T, Steddin S. Current view of the mechanism of benign paroxysmal positional vertigo: cupulolithiasis or canalolithiasis? *J Vestib Res* 1993;3:373-82.
- [15] Aw ST, Todd MJ, Aw GE, et al. Benign positional nystagmus. A study of its three-dimensional spatio-temporal characteristics. *Neurology* 2005;64:1897-905.
- [16] Crevits L. Treatment of anterior canal benign paroxysmal positional vertigo by a prolonged forced position. *J Neurol Neurosurg Psychiatry* 2004;75:779-81.
- [17] Bertholon P, Bronstein AM, Davies RA, et al. Positional down beating nystagmus in 50 patients: cerebellar disorders and possible anterior semicircular canalolithiasis. *J Neurol Neurosurg Psychiatry* 2002;72:366-72.
- [18] Dix MR, Hallpike C. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc R Soc Med* 1952;45:341-54.
- [19] Barber HO. Positional nystagmus. *Otolaryngol Head Neck Surg* 1984;92:649-55.
- [20] Kattah JC, Kolsky MP, Luessenhop AJ. Positional vertigo and the cerebellar vermis. *Neurology* 1984;34:527-9.
- [21] Wenning GK, Ben Shlomo Y, Magalhes M, et al. Clinical features and natural history of multiple system atrophy: an analysis of 100 cases. *Brain* 1994;117:835-45.
- [22] Herdman SJ, Tusa RJ. Complications of the canalith repositioning procedure. *Arch Otolaryngol Head Neck Surg* 1996;122:281-6.
- [23] Rahko T. The test and treatment methods of benign paroxysmal positional vertigo and an addition to the management of vertigo due to the superior vestibular canal (BPPV-SC). *Clin Otolaryngol* 2002;27:392-5.
- [24] Nakayama M, Epley JM. BPPV and variants: improved treatment results with automated, nystagmus-based repositioning. *Otolaryngol Head Neck Surg* 2005;133:107-12.
- [25] Blanks RHI, Curthoys IS, Markham CH. Planar relationships of the semicircular canals in man. *Acta Otolaryngol* 1975;80:185-96.
- [26] Aw ST, Haslwanter T, Halmagyi GM, et al. Three-dimensional vector analysis of the human vestibuloocular reflex in response to high-acceleration head rotations. I. Responses in normal subjects. *J Neurophysiol* 1996;76:4009-20.
- [27] Baloh RW, Honrubia V, Jacobson K. Benign paroxysmal positioning vertigo: clinical and oculographic features in 240 cases. *Neurology* 1987;37:371-8.
- [28] Amor-Dorado JC, Llorca J, Costa-Ribas C, et al. Giant cell arteritis: a new association with benign paroxysmal positional vertigo. *Laryngoscope* 2004;114:1420-5.
- [29] Pollak L, Davies RA, Luxon LL. Effectiveness of the particle repositioning maneuver in benign paroxysmal positional vertigo with and without additional vestibular pathology. *Otol Neurotol* 2002;23:79-83.