Benign positional paroxysmal vertigo: videonystagmographic study using rotatory test

La vertigine parossistica periferica benigna: studio videonistagmografico mediante test rotatori

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Key words

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Parole chiave

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Summary

Benign Peripheral Paroxysmal Vertigo is a disease of the posterior labyrinth caused by endolymphatic debris, provoking vertigo with some movements of the head. Diagnosis is usually made by finding the positional nystagmus with appropriate manoeuvres. Spontaneous resolution is frequent and in these cases diagnosis is only probable and suspected from anamnesis. Aim of the present investigation was to establish more evaluation parameters in the study of Benign Peripheral Paroxysmal Vertigo. A series of 97 selected patients presenting Benign Peripheral Paroxysmal Vertigo, have been submitted to sinusoidal kinetic test. Patients have been studied during the acute phase of the condition and after recovery. Vestibulo-oculomotor reflex has been sought by stimulating the horizontal and vertical canals. Kinetic stimulus consisted in sinusoidal rotation at 0.12 Hz and 0.05 Hz. Evaluation parameters comprised preponderance, gain and phase of provoked nystagmus, recorded by means of an Ulmer videonystagmograph. Using this same technique of stimulation, 20 normal volunteers were studied in order to establish normal values for reference. Values obtained in the patient population of patients have been compared, by Student t test, with values obtained in the same cured patients and with those in normal subjects. In the patients with Benign Peripheral Paroxysmal Vertigo of the lateral canal a nystagmus preponderance toward the healthy side was observed, as well as an increase in the phase lead, also in the canals not affected by the condition. In cured patients, disappearance of the preponderance and persistence of the phase abnormalities are observed. These results suggest a multicanal pathogenesis of Benign Peripheral Paroxysmal Vertigo.

Riassunto

La Vertigine Parossistica Posizionale Benigna è una affezione del labirinto posteriore causata da detriti endolinfatici, che provoca vertigine in alcuni movimenti della testa nello spazio. La diagnosi è basata sul reperto di un nistagmo posizionale scatenato da apposite manovre. In molti pazienti la risoluzione spontanea è frequente: in tali casi la diagnosi è soltanto probabile e dedotta dall'anamnesi. Scopo del presente studio è quello di valutare la Vertigine Parossistica Posizionale Benigna anche con i tests cinetici. Pertanto abbiamo selezionato 97 pazienti affetti da Vertigine Parossistica Posizionale Benigna che sono stati sottoposti a test sinusoidali. Il riflesso vestibolo-oculomotorio è stato ricercato stimolando i canali orizzontali e le coppie dei canali verticali. Lo stimolo rotoacceleratorio è stato una rotazione sinusoidale alle frequenze di 0,12 e 0,05 Hz. I parametri di valutazione sono stati la preponderanza, il guadagno e la fase del nistagmo provocato e registrato per mezzo di un videonistagmografo di tipo Ulmer. Con la stessa tecnica abbiamo studiato 20 soggetti normali allo scopo di preparare valori normativi di riferimento. I valori ottenuti nel campione di pazienti sono stati comparati utilizzando il test t-Student con i valori misurati negli stessi soggetti dopo la guarigione e con i valori ottenuti nei soggetti normali. Nei soggetti affetti da Vertigine Parossistica Posizionale Benigna del canale laterale abbiamo notato una preponderanza verso il lato sano così come un incremento dell'anticipo di fase anche in canali non coinvolti dalla affezione. Nei soggetti guariti si è notata la scomparsa della preponderanza e la persistenza delle anomalie di fase. Questo risultato è compatibile con una genesi multicanalare della Vertigine Parossistica Posizionale Benigna.

Introduction

Benign Paroxysmal Positional Vertigo (BPPV) is a syndrome characterized by attacks of vertigo, triggered in certain head positions.
The pathogenesis of BPPV has long since been attributed to cupololithiasis, on the grounds of histological findings of otoconial deposits on the border of the cupola [1].

Other Authors [2-4] have postulated canalolithiasis in the pathogenesis of the syndrome: this mechanism would account for the characteristics of this dizziness: the deposits, but also accumulations of the endolymph [5] or inflammatory complexes [6], are joined in a semicircular canal, forming mobile particles that cause endolymphatic flow due to gravity inside the canal.
These materials typically provoke paroxysmal vertigo both with nystagmus with brief latency, duration ap-

proximately 30 seconds, fatigue (decreasing when some positions are repeated), changes in the direction of the nystagmus with changes in the position of the head.

Whilst BPPV has been described for the posterior canal (BPPV-PC), it also affects the horizontal (or lateral) canal [3 5 7] (BPPV-HC) and the anterior canal [8] even if with a lower frequency. Recently, free-floating particles have been detected during surgery [9]. The causes of BPPV are idiopathic; sometimes it may be observed following a trauma [6 10 11], stape surgery [12], viral labyrinthitis [13], or chronic otitis media [14], but, in most cases, it is considered idiopathic [15].

The visco-elastic properties of the endolymph and the tendency of debris to disperse result in the end of the crises and, in many cases, spontaneous recovery.

In those patients with previous dizziness and no positional nystagmus, the diagnosis is only suspected and may be deduced from the anamnesis; the percentage of such cases has been reported elsewhere as 8.7% of all equilibrium disorders [15].

If the attack of BPPV is due to a gravitational load, it is of clinical interest to determine whether:

1) abnormal values of vestibular stimulation tests in patients presenting BPPV are useful in the diagnosis of "probable" cases;
2) dysfunction of other canals in addition to the posterior canal initially involved.

Patients and methods

A total of 97 patients (56 female) suffering from BPPV, aged between 27 and 84 years (mean 54±13) have been studied. Of these, 37 were affected in the right posterior canal, 26 in the left posterior canal, 12 in the right lateral canal and 9 in the left lateral canal. Those patients with mixed lithiasis (lateral plus posterior canal) (13 cases) were not included in the study group.

Criteria for diagnosis of BPPV-PC were: the presence of positional vertigo and a vertical plus rotatory, transient, brief latency, fatigable nystagmus in the Cawthorne positions [16]. Classical Dix-Hallpike positional manoeuvres have not been employed due to difficulties in older patients and in patients with cervical disorders. BPPV-HC was diagnosed when positional vertigo and pure horizontal geotropic or apogeotropic nystagmus were observed in McClure-Pagnini positions [4 5].

Patients have been evaluated by means of kinetic tests of the sinusoidal type with stimuli of 0.12 Hz and maximum speed of 40°/sec and of 0.05 Hz and maximum speed of 60°/sec, using a Polman N/1 rotatory chair.

Horizontal vestibulo-ocular reflex (VOR) has been sought by stimulating the lateral canal (the patient's head hanged of 30° down). Vertical VOR has also been studied stimulating the couple right posterior canal – left anterior canal (the patient's head hanged of 90° to the right shoulder and down of approximately 45°, HH right) and the couple left posterior canal – right anterior canal (the patient's head hanged of 90° to the left shoulder and down of approximately 45°, HH left)

The following parameters have been evaluated:

a) nystagmus preponderance (with our instrumentation: positive sign if directed to left, and negative if directed to the right, for the horizontal canals; negative if directed superiorly and positive if directed down, for the vertical canals);
b) gain (ratio between maximal slow phase velocity and the speed of the chair);
c) nystagmus phase defined as difference in angular degrees between speed profile of the chair and that of the speed of the slow phases.

All the above-mentioned parameters were calculated for each of the canal couples.

Recording and measuring of nystagmus have been performed using an infrared CCD camera connected to a videonystagmograph (VNG) (Ulmer, Synapsis-Audiomedical, Marseille, France) and the computerized elaboration effected with the software prepared ad hoc. These devices proved to be without artifacts when performing manoeuvres.

The same rotatory stimulation was used in the study of 20 normal subjects (12 female, mean age 46±9

Table I. VOR parameters in normal subjects: means and standard deviations ( ).

	Normal subjects		
	Prep.	Gain	Phase
0.12 Hz	–0.1 (0.9)	0.3 (0.11)	5.4 (3.6)
0.05 Hz	0.07 (0.86)	0.37 (0.13)	8.5 (5.2)
HH right	0 (0.8)	0.25 (0.14)	12.7 (7.4)
HH left	0.5 (0.79)	0.23 (0.08)	–168 (5.6)

Table II. VOR parameters in affected patients: means and standard deviations.

	BPPV right post. can.			BPPV left post. can.		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	–0.4 (2.2)	0.42 (0.23)	10.5 (7.6)	–0.1 (1.2)	0.39 (0.22)	11 (2)
0.05 Hz	–1 (3.2)	0.44 (0.18)	9.5 (7.9)	–1.3 (3.4)	0.44 (0.2)	12 (10)
HH right	0 (2.1)	0.28 (0.16)	15.5 (7)	0.4 (3.8)	0.27 (0.18)	17 (10)
HH left	1.9 (3.4)	0.3 (0.17)	–162 (10)	1 (3.2)	0.29 (0.16)	–162 (10)
	BPPV right lat. can.			BPPV left lat. can.		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	1.9 (1.4)	0.58 (0.22)	8.5 (4.2)	–1.8 (1.5)	0.39 (0.24)	8.6 (6.6)
0.05 Hz	1.7 (2.4)	0.56 (0.2)	7.7 (5.3)	–2.3 (4.8)	0.48 (0.12)	9.6 (5.7)
HH right	0.3 (2.2)	0.34 (0.14)	15 (6)	0.78 (2.1)	0.22 (0.08)	25 (24)
HH left	0.4 (2.4)	0.32 (0.1)	–169 (11)	1.5 (1)	0.26 (0.12)	–162 (7.6)

Table III. VOR parameters in cured patients: means and standard deviations.

	BPPV right post. can. (cured)			BPPV right lat. can. (cured)		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	0.6 (1.5)	0.31 (0.18)	9.3 (3)	–0.8 (1.5)	0.46 (0.11)	8.8 (3)
0.05 Hz	–0.3 (0.3)	0.42 (0.14)	8.2 (4.7)	0.5 (0.2)	0.45 (0.1)	10.0 (5)
HH right	–0.3 (1.5)	0.29 (0.14)	16.7 (10)	0.6 (0.2)	0.26 (0.1)	19.9 (9)
HH left	0.3 (1.4)	0.3 (0.14)	–162 (6.5)	0.8 (0.7)	0.3 (0.14)	–164 (2.8)
	BPPV left post. can. (cured)			BPPV left lat. can. (cured)		
	Prep.	Gain	Phase	Prep	Gain	Phase
0.12 Hz	0.3 (0.9)	0.39 (0.1)	8.7 (3)	0.6 (0.9)	0.38 (0.1)	9.4 (2.8)
0.05 Hz	0.4 (0.8)	0.51 (0.09)	9.1 (3.7)	0.3 (0.7)	0.42 (0.13)	7.9 (3.8)
HH right	–0.2 (1.9)	0.32 (0.17)	14.9 (8.2)	0.22 (0.3)	0.32 (0.09)	11.9 (5.4)
HH left	0.1 (0.2)	0.34 (0.2)	–160 (8.2)	–0.5 (0.32)	0.37 (0.2)	–161 (3.9)

Table IV. Student t test values, * p<0.05, ** p<0.01, normal subjects versus affected patients.

	BPPV right post. can.			BPPV left post. can.		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	0.28	0.06	0.009**	0.49	0.08	0.22
0.05 Hz	0.07	0.08	0.30	0.08	0.11	0.13
HH right	0.25	0.25	0.49	0.27	0.35	0.042*
HH left	0.048*	0.10	0.03*	0.25	0.06	0.02*
	BPPV right lat. can.			BPPV left lat. can.		
	Prep.	Gain	Phase	Prep	Gain	Phase
0.12 Hz	0.0001 **	0.0004 **	0.002 **	0.03 *	0.11	0.04 *
0.05 Hz	0.01 *	0.0009 **	0.01 *	0.02 *	0.10	0.32
HH right	0.22	0.07	0.20	0.07	0.35	0.02 *
HH left	0.45	0.01 *	0.36	0.01 *	0.18	0.03 *

years) in order to define normal normative values for reference.
The study procedure has been planned performing kinetic stimulations after positional tests. Subsequently, patients were treated with liberatory Semont manoeuvre [17] for the posterior canal and with another method for the lateral canal [18].
Patients were evaluated twice in the first week: nystagmus positive cases were still submitted to a second manoeuvre, those negative and symptom free were observed on the 30th day.
The second battery of sinusoidal stimulations, identical to the first, was performed if the patients were still symptom-free and with no positional nystagmus. It should be pointed out that 2 patients with posterior, and 2 with lateral BPPV still complaining of vertigo and positional nystagmus were excluded from the study. All data related to stimulations have been compared using Student one-tailed t-test. Values of $p<0.05$ and $p<0.01$ were considered significant.
The following statistical comparisons have been analysed:
1) normal subjects versus patients presenting BPPV;
2) normal subjects versus cured patients;
3) affected patients versus cured patients.

The first comparison was made for each BPPV type (normal subjects vs right posterior canal, left posterior canal, normal subjects vs right lateral canal, left lateral canal). The second comparison has been made between normal subjects and pooled cured posterior or lateral canal. The third comparison has been made for each BPPV type between data of affected vs cured patients.

Results

The healthy volunteer subjects obviously showed no nystagmus preponderance (Table I). The apparently negative phase values obtained in left HH are due to vertical downward nystagmus evoked by clockwise rotation: the slow phase direction is measured in the opposite phase compared to the envelope of velocity of chair.
BPPV patients have been classified according to the side and type of canal involved; patients presenting right posterior BPPV showed an asymmetry with downward directional preponderance stimulating the left posterior canal (Table II).
Right lateral canal BPPV showed left nystagmic preponderance, while left lateral cases showed a right preponderance (Table II).
The same patients studied at recovery no longer showed the preponderances observed in the acute phase (Table III) or persistence of phase abnormalities.
Comparison between normal subjects and affected patients showed significant differences in preponderance and phase also stimulating canals not affected by BPPV as revealed by positional tests (Table IV); the gain differences observed in affected patients may be due to the high levels of arousal caused by vertigo experienced in the positional test. The comparison between normal subjects and cured patients does not underline any differences in preponderance but show that an increase in the phase lead is maintained in the cured patients (Table V).
The comparison between affected patients and cured patients failed to reveal any significant differences with the exception of high preponderance values in patients with BPPV of the right lateral canal (Table VI).

Discussion

Two mechanisms are hypothesised to explain vestibular symptoms in BPPV: cupololithiasis and canalolithiasis [19]; in both cases, the dynamic activation of posterior or lateral cupola outcomes abnormal responses.
The comparison between normal subjects and patients presenting lateral canal BPPV underlines a significant nystagmus preponderance towards the healthy side and an increase in phase lead. The preponderance towards the healthy side is in agreement with the findings of others of a caloric ipsilateral canal paresis [3 20-22] reversing with the resolution of the clinical symptoms: recovery of these parameters is related to the mechanisms of BPPV. It is feasible to suggest that a liberatory manoeuvre may be involved in the removal of high density floating debris from the canal or other clots adhered to the cupola.
Overall examination of the data and, particularly, the significant increase in phase lead between normal subjects and patient presenting posterior canal BPPV indicate a variation in the dynamic order of the couple cupola-endolymph. In patients presenting right posterior lithiasis, an increased phase lead, stimulating the posterior canal of the other side or the lateral canal is observed: similar findings are found in BPPV-HC in which also the stimulating vertical canals are impaired. These findings might be due to chance since these are not constant for all the canals and sides examined and also because of the variability in the significance levels, but may also suggest that otoconial deposits or district variations in endolymphatic density may also be localised in a sub-clinical form in canals other than those revealed by clinical signs. The increased phase lead is similar to that found after labyrinthectomy [23] or after canal plugging [24].
Corvera et al. [25] found smaller phase leads in the BPPV; however these data are not comparable with the present study due to the different range of stimulation frequencies employed (2-6 Hz).
The comparison between cured patients and normal subjects indicates the disappearance of preponder-

Table V. Student t test values, normal subjects versus cured patients.

	BPPV right post. can. (cured)			BPPV right lat. can. (cured)		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	0.11	0.07	0.03*	0.09	0.01*	0.03*
0.05 Hz	0.27	0.12	0.42	0.23	0.12	0.24
HH right	0.3	0.14	0.04*	0.1	0.43	0.04*
HH left	0.37	0.02*	0.05	0.23	0.07	0.04*
	BPPV left post. can. (cured)			BPPV left lat. can. (cured)		
	Prep.	Gain	Phase	Prep	Gain	Phase
0.12 Hz	0.8	0.09	0.05	0.18	0.3	0.028*
0.05 Hz	0.22	0.3	0.38	0.32	0.12	0.28
HH right	0.19	0.17	0.04*	0.34	0.38	0.18
HH left	0.48	0.1	0.009**	0.28	0.19	0.03*

Table VI. Student t test, affected versus cured patients.

	BPPV right post. can.			BPPV left post can.		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	0.37	0.44	0.28	0.15	0.37	0.34
0.05 Hz	0.22	0.35	0.23	0.16	0.38	0.05
HH right	0.32	0.36	0.46	0.19	0.30	0.31
HH left	0.06	0.45	0.16	0.21	0.l40	0.10
	BPPV right lat. can.			BPPV left lat. can.		
	Prep.	Gain	Phase	Prep.	Gain	Phase
0.12 Hz	0.007 **	0.20	0.16	0.15	0.30	0.48
0.05 Hz	0.18	0.14	0.23	0.22	0.31	0.41
HH right	0.40	0.15	0.15	0.46	0.26	0.08
HH left	0.36	0.35	0.18	0.23	0.35	0.27

ances and maintenance of the increased phase lead: an element suggesting the persistence of the pathogenetic factors of BPPV, in the silent state, in previously affected patients. This persistence may explain the recurrence of vertigo, in many cases: long-term follow-up after liberatory manoeuvres reveals a recurrence rate of 33% in intervals up to 3 years [22]. Moreover, if abnormal values of phase were related only to the clinical appearance of BPPV, one might expect a difference between cured patients by manoeuvres with respect to the same cases observed during the acute stage. In our results, no such difference is present. This last finding suggests that BPPV mechanisms may be present in patients also in the absence of vertigo.

It is tempting to suggest that, in cupololithiasis, debris adhering to the cupola may persist or, in canalolithiasis, district density alterations of endolymph may be hypothesised.

Conclusion

Results emerging from the present investigation appear to indicate that otoconial deposits or district variations in endolymphatic density may be localised also in a sub-clinical fashion in canals other than those revealed by clinical signs; further studies or larger study populations are necessary to assign a possible role of the nystagmus phase in the diagnosis of "probable" BPPV.

References

[1] Schuknecht HF, Ruby RRF. *Cupulolithiasis*. Adv Oto-Rhino-Laryng 1973;20:434-43.

[2] Hall SF, Ruby RR, McClure J. *The mechanism of benign paroxysmal vertigo*. J Otolaryngol 1979;8:151-8.

[3] Pagnini P, Nuti D, Vannucchi P. *Benign paroxysmal positional vertigo of the horizontal canal*. ORL 1989;51:161-70.

[4] Epley JM. *The canalith repositioning procedures for treatment of benign paroxysmal positional vertigo*. Otolaryngol Head Neck Surg 1992;107:399-404.

[5] Mc Clure JA. *Horizontal canal BPV*. J Otolaryngol 1985;14:30-5.

[6] Parnes LS, Price-Jones RG. *Particle repositioning maneuver for benign paroxysmal positional vertigo*. Ann Otol Rhinol Laryngol 1993;102:325-31.

[7] Baloh RW, Jacobson K, Honrubia V. *Horizontal semicircular canal variant of benign positional vertigo*. Neurology 1993;43:2542-9.

[8] Kartsarkas A. *Nystagmus of paroxysmal positional vertigo: burdens new insights*. Ann Otol Rhinol Laryngol 1987;96:305-8.

[9] Parnes LS, McClure J. *Free-floating endolymph particles: to new operational findings during posterior semicircular canal occlusion*. Laryngoscope 1992;102:988-92.

[10] Barber H. *Positional nystagmus, especially after head injury*. Laryngoscope 1964;74:891-944.

[11] Barber H. *Head injury audiological and vestibular findings*. Ann Otol Rhinol Laryngol 1969;78:239-52.

[12] Spector M. *Positional vertigo after stapedectomy*. Ann Otol Rhinol Laryngol 1961;70:251-4.

[13] Mc Clure JA, Rounthwaine J. *Vestibular dysfunction associated with benign paroxysmal vertigo*. Laryngoscope 1977;87:1434-42.

[14] Stahle J, Terins J. *Paroxysmal positional nystagmus: electronystagmographic and clinical study*. Ann Otol Rhinol Laryngol 1965;74:69-83.

[15] Mosca F, Morano M. *Benign paroxysmal peripheral vertigo, incidence and treatment*. Ann Otolaryngol Chir Cervicofac 2001;118:95-101.

[16] Cawthorne T. *Positional nystagmus*. Ann Otol 1954;63:481-90.

[17] Semont A. *Les traitements des troubles vestibulo-oculaires.* These. Bibliotheque Nationale de Medicine. Paris 1984.

[18] Mosca F. *La vertigine parossistica da canale laterale, proposta di un metodo di trattamento*. Acta Otorhinol Ital 1999;19:144-9.

[19] Brandt T, Steddin S. *Current view of the mechanism of benign paroxysmal positioning vertigo: cupololithiasis or canalolithiasis?* J Vest Res 1993;3:373-82.

[20] Katsarkas A. *Electronystagmographic (ENG) findings in paroxysmal positional vertigo (PPV) as to sign of vestibular dysfunction*. Acta Otolaryngol 1991;111:193-200.

[21] Strupp M, Brandt T, Steddin S. *Horizontal canal benign paroxysmal positioning vertigo: reversible ipsilateral caloric hypoexcitability caused by canalolithiasis?* Neurology 1995;45:2072-6.

[22] Vannucchi P, Giannoni B, Pagnini P. *Treatment of semicircular canal benign paroxysmal positional vertigo*. J Vestibular Res 1997;7:1-6.

[23] Wesseinstein L, Ratnam , Anastasio TJ. *Vestibular compensation in the horizontal vestibulo-ocular reflex of the goldfish*. Behav Brain Res 1996;75:127-37.

[24] Angelaki DE, Hess BJ, Suzuki J. *Adaptation of primate vestibuloocular reflex to altered peripheral vestibular inputs. I. Frequency specific recovery of horizontal VOR after inactivation of the lateral semicircular canals*. J Neurophysiol 1996;76: 2941-53.

[25] Corvera B, Corvera J, Hernandez LM. *Benign positional vertigo produces specific pattern of abnormalities with high frequency vestibulo-ocular reflex testing*. Ear Nose Throat J 1994;73:768-71.

[26] Vrabec JT. *Benign paroxysmal positioning vertigo and otolith repositioning*. Arch Otolaryngol Head Neck Surg 1998;124:223-5.

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