Eur Arch Otorhinolaryngol (2000) 257:372–375

OTOLOGY

Stefano Di Girolamo · Fabrizio Ottaviani
Emanuele Scarano · Pasqualina Picciotti
Walter Di Nardo

Postural control in horizontal benign paroxysmal positional vertigo

Received: 2 December 1999 / Accepted: 17 February 2000

Abstract Sixteen patients affected by benign paroxysmal positional vertigo of the horizontal semicircular canal (BPPV-HSC) were investigated by means of dynamic posturography (DP) and during bithermal caloric stimulation. Data were compared to data from 40 patients with benign paroxysmal positional vertigo of the posterior semicircular canal (BPPV-PSC) and 20 healthy controls. No postural deficit was observed before or after a liberative Lempert's manoeuvre when patients were compared to control subjects. BPPV-PSC postural scores were significantly impaired compared to scores from the BPPV-HSC group. A residual significant postural impairment was also observed after a successful liberative manoeuvre in the BPPV-PSC group. Electronystagmographic recordings before recovery revealed significant hypoexcitability of the affected ear in 8/16 patients of the BPPV-HSC group. After the liberative manoeuvre, a symmetric bilateral response to caloric stimulation was recorded in all patients. Three main conclusions can be drawn from the present data. First, disorders of the horizontal semicircular canal do not change postural control. Second, dynamic posturography can detect the postural imbalance due to posterior semicircular canal dysfunction even after resolution of paroxysmal vertigo attacks. Third, utricular dysfunction can be ruled out as a cause of the residual postural deficit observed in BPPV-PSC patients. Therefore the recovery delay observed even 1 month after the liberative manoeuvre in the BPPV-PSC-group might be due to the persistence of small amounts of residual debris in the canal, to paralysis of ampullar receptors, or to the time needed for central vestibular re-adaptation.

S. Di Girolamo (✉) · F. Ottaviani · E. Scarano · P. Picciotti
W. Di Nardo
Istituto di Clinica Otorinolaringoiatrica,
Policlinico Agostino Gemelli, Università Cattolica del Sacro Cuore,
Largo Agostino Gemelli 8, 00168 Rome, Italy
e-mail: sdigirolamo@rm.unicatt.it
Tel.: +39-06-30154439, Fax: +39-06-3051343

Key words Benign paroxysmal positional vertigo (BPPV) · Horizontal semicircular canal · Postural balance · Utricular function

Introduction

Benign paroxysmal positional vertigo (BPPV) is a common peripheral vestibular disorder that is clinically characterized by positional paroxysmal nystagmus. A deficit of postural control is usually present and has been documented by many different tools [2, 7, 8, 14, 15, 25].

A liberative manoeuvre significantly relieves paroxysmal symptoms of BPPV-posterior semicircular canal (BPPV-PSC) [20] and significantly improves postural control, as demonstrated by means of static [3] and dynamic posturography [6]. However, incomplete postural recovery was recorded in a previous paper and we suggested that it could be due to altered otolithic function [12, 23]. This deficit, as hypothesised for otolithic post-traumatic vertigo [4], is probably caused by the unequal loads of the utricular macula beds resulting from detachment of the clot floating in the semicircular canal.

If this hypothesis were true, the same postural deficit should be present in patients affected by BPPV-horizontal semicircular canal (BPPV-HSC) but, as far as we know, no paper in the literature deals with this topic. In order to evaluate postural control in BPPV-HSC and to verify the presence of the utricular dysfunction hypothesized in BPPV-PSC we have studied postural control in BPPV-HSC and the vestibular labyrinthine function.

Subjects and methods

Sixteen patients (nine women, mean age 48.7 years, and seven men, mean age 58.5) affected by BPPV-HSC underwent complete clinical neuro-otologic evaluation, including pure-tone audiometry, tympanometry, dynamic posturography, bithermal caloric test and auditory evoked potentials. Spontaneous, positional and positioning nystagmus using Frenzel's glasses and video-oculography were investigated.

The diagnosis of BPPV-HSC was based on the following features: history of brief episodes of vertigo induced by rolling the

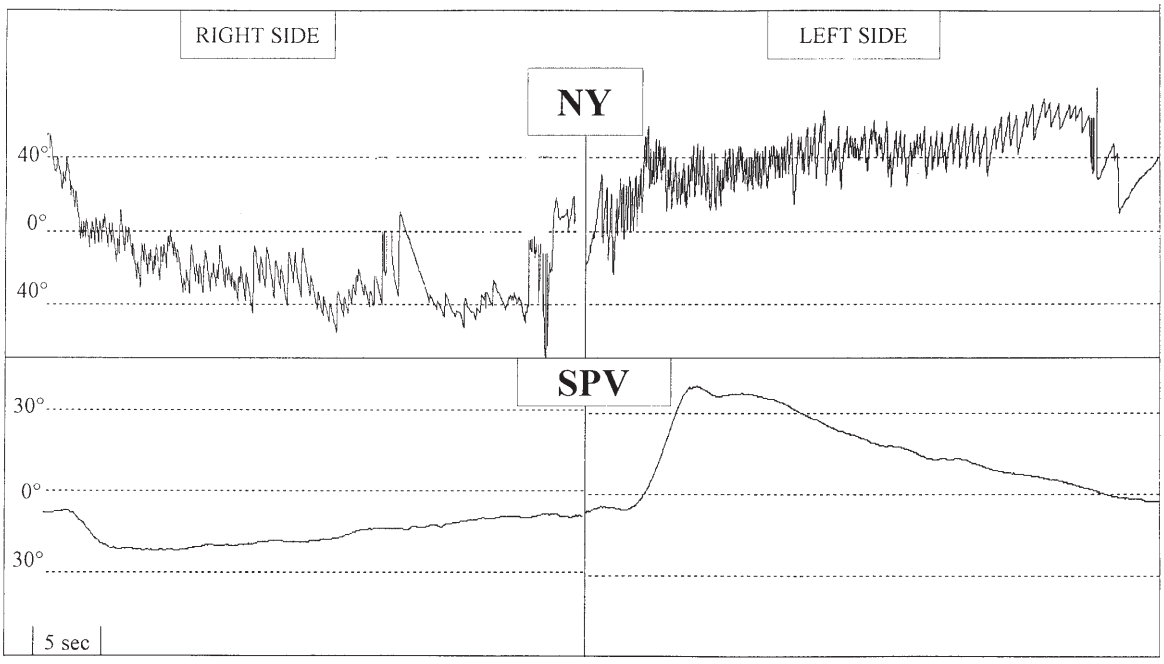


Fig. 1 Electronystagmographic recording shows the typical geotropic nystagmus (*NY*) and slow phase velocity (*SPV*) of the left horizontal canal paroxysmal positional vertigo

head from side to side while supine, a linear horizontal nystagmus toward the lower ear when the head of the supine patient was rapidly turned from side to side, beating stronger toward the affected ear, with short latency, poorly fatigable and lasting 30 to 90 seconds [1, 13, 16, 17, 18] (Fig. 1). Patients with a history, symptoms, physical findings or laboratory tests indicating the presence of central nervous system ocular motility or middle ear diseases were discarded.

Data from these patients were compared to data from a control group of 20 healthy normal subjects (mean age 45.2 years) and from a group of 40 BPPV-PSC patients (mean age 43.3).

In patients presenting with apogeotropic variant nystagmus [17, 21] directed toward the upperear when the head was rotated to either side, a manoeuvre was performed to change the position of the debris and the direction of nystagmus [5].

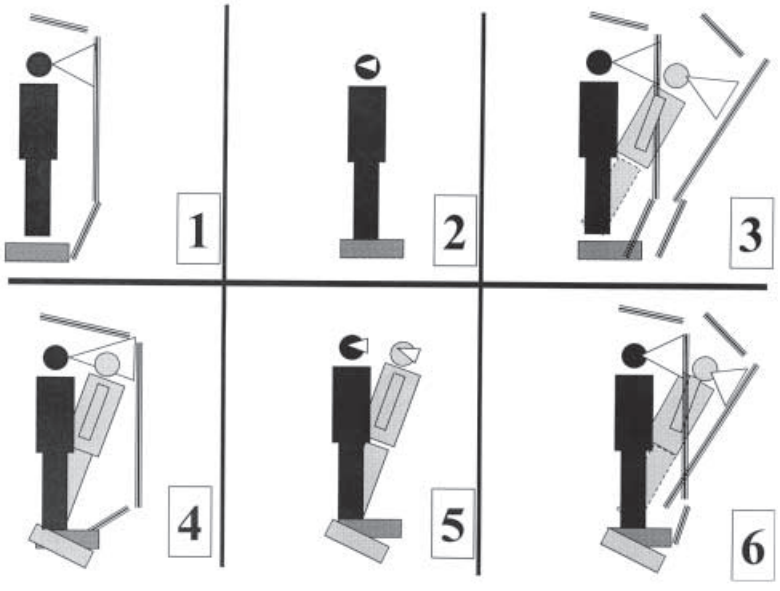
Electronystagmography was recorded during bithermal stimulation before and after treatment.

The liberative manoeuvre for BPPV-HSC was the one suggested by Lempert [10, 11] and named "barbecue rotation". It consists of a single 270° rotation around the supine patient's yaw longitudinal axis, performed in rapid steps of 90° at 30 s intervals, starting from the affected ear. After the manoeuvre, as recommended by Nuti [16], we suggested to all patients that they should lie on the healthy side during the following night [24].

The success of barbecue rotation for BPPV-HSC was indicated by apogeotropic nystagmus when the patient's head reached the affected-ear-down position (five patients), or by the absence of paroxysmal vertigo in the following 7 days.

In order to avoid possible interference caused by the diagnostic manoeuvre, dynamic posturography (Equitest, Neurocom Int. Inc., Clackamas, Oregon) was performed 1 h later.

Fig. 2 The six-test condition of dynamic posturography



Postural control scores were recorded in six test conditions (Fig. 2) and sensory analysis was calculated on the relationship among the equilibrium scores. This evaluation identifies the sensory dysfunction and individual preference for different inputs: somatosensory, visual, vestibular and vision preferential.

The patients were re-assessed 1 month after the liberative manoeuvre. Before and after recovery labyrinthine function was evaluated during bithermal stimulation.

Statistical analysis

All results are expressed as means ±1 SD. Analysis of variance with repeated measures was performed and differences at $P \leq 0.05$ were considered significant. The Scheffé test was used to make paired comparison tests among controls and BPPV groups.

Results

Nystagmus and vertigo disappeared after the first treatment in nine patients, and after the second liberative manoeuvre in six patients, but in one patient the manoeuvre caused the BPPV-HSC to change to ipsilateral BPPV-PSC.

Electronystagmographic recordings during bithermal stimulation before treatment revealed significant hypoexcitability of the affected ear in 8/16 patients. After the liberative manoeuvre a symmetric bilateral response to caloric stimulation was recorded in all patients.

No postural deficit could be observed in the BPPV-HSC group before or after the Lempert manoeuvre in comparison to controls. In the BPPV-PSC group significant impairment of postural control, mainly confined to the vestibular afferents, was present compared to controls or the BPPV-HSC group.

After a successful liberative manoeuvre a residual postural deficit was also registered in the BPPV-PSC group compared to controls (Tables 1, 2, 3).

Before the liberative manoeuvre 11 of 40 BPPV-PSC patients failed during the test while no falls were observed in the BPPV-HSC group before or after treatment.

The postural control of the BPPV-HSC patient who changed to BPPV-PSC was normal before the Lempert manoeuvre. A significant worsening of the postural score was recorded after the onset of the BPPV-PSC.

Table 1 Sensory organization test and composite equilibrium score in the three groups before the liberative manoeuvre. Values are shown as mean ±1 SD and ANOVA with repeated measures. *n.s.* not significant

	Normal	BPPV-PSC	BPPV-HSC	ANOVA
Composite	80.65 ± 0.69	64.65 ± 2.5	78.23 ± 5.54	n.s.
Somatosensory	0.98 ± 0.00	0.97 ± 0.01	0.98 ± 0.04	n.s.
Visual	0.89 ± 0.01	0.74 ± 0.05	0.83 ± 0.11	n.s.
Vestibular	0.71 ± 0.01	0.48 ± 0.05	0.67 ± 0.11	n.s.
Preferential	1.00 ± 0.01	0.95 ± 0.03	1.01 ± 0.11	n.s.

Table 2 Sensory organization test and composite equilibrium score in the three groups after liberative manoeuvre. Values are shown as mean ±1 SD and ANOVA with repeated measures. *n.s.* not significant

	Normal	BPPV-PSC	BPPV-HSC	ANOVA
Composite	80.65 ± 0.69	73.27 ± 1.74	80.3 ± 2.08	n.s.
Somatosensory	0.98 ± 0.00	1.00 ± 0.02	0.99 ± 0.03	n.s.
Visual	0.89 ± 0.01	0.81 ± 0.04	0.85 ± 0.03	n.s.
Vestibular	0.71 ± 0.01	0.68 ± 0.04	0.74 ± 0.02	n.s.
Preferential	1.00 ± 0.01	0.91 ± 0.02	0.98 ± 0.04	n.s.

Discussion

Utricular dysfunction has been documented in BPPV-PSC by ocular counter-rolling (OCR) [12] and an eccentric rotatory test [23]. This otolithic dysfunction induced by the unequal loads of the macula beds has been considered responsible for the postural impairment following recovery from paroxysmal vertigo in BPPV-PSC [6]. If such a hypothesis were true, similar findings would also have been found in BPPV-HSC patients. Instead, in the present study normal postural scores were recorded before and after recovery, ruling out such a pathological mechanism.

An alteration in the dynamics of the semicircular canals ensuing from the presence of a clot in the affected canal must therefore be considered. Worsening of the postural score during paroxysmal vertigo attacks in a BPPV-PSC group has been clearly demonstrated [9]. Hypofunction of the pathological horizontal semicircular canal in BPPV-HSC patients, suggesting involvement of a canal, was recorded in about half of our patients by means of bithermal caloric stimulation. After recovery, responses became symmetric and of normal magnitude [22]. The reversible ipsilateral caloric hypoexcitability can be explained by a functional plugging of the HSC caused by the dislodged otolithic material, which would abolish the convective current, as observed in squirrel monkeys [19] in

Table 3 Scheffé test of sensory analysis and composite scores in controls (*N*), *PSC* and *HSC* groups. *n.s.* not significant

Group	Composite	Somato-sensory	Visual	Vestibular	Prefer-ential
N vs Pre-PSC	0.0005	0.07	0.002	0.0001	0.001
N vs Post-PSC	0.001	n.s.	0.007	0.001	0.001
N vs Pre-HSC	n.s.	n.s.	n.s.	n.s.	n.s.
N vs Post-HSC	n.s.	n.s.	n.s.	n.s.	n.s.
Pre-HSC vs Pre-PSC	0.001	n.s.	0.05	0.001	0.001
Post-HSC vs Post-PSC	0.01	n.s.	0.05	0.01	0.01
Pre-HSC vs Post-HSC	n.s.	n.s.	n.s.	n.s.	n.s.
Pre-PSC vs Post-PSC	0.0001		0.003	0.0001	0.004

which experimental plugging of the horizontal canal reduced the caloric response. Thus, we can assume that dysfunction of a canal is responsible for the postural deficit detectable in BPPV-PSC patients [9, 25] and is the cause of about 50% of reduced responses after caloric stimulation in BPPV-HSC- patients [1, 13, 18]. The different figures may be due to the different sensitivity of posturographic and caloric tests and to the physiological differences between the horizontal and the posterior canals, as is also suggested by the different fatigability and latency of the paroxysmal nystagmus.

In conclusion, three main considerations can be drawn from the present data. First, BPPV-HSC does not affect postural control. Second, dynamic posturography seems able to detect postural imbalance due to posterior semicircular canal dysfunction during and even after the resolution of paroxysmal vertigo attacks. Third, a role for utricular dysfunction in the postural deficit observed in the BPPV-PSC group can be ruled out. Therefore the recovery delay observed even 1 month after the liberative manoeuvre in the BPPV-PSC group might be due to the persistence of small amounts of residual debris in the canal, to paresis of ampullar receptors, or to the time needed for vestibular re-adaptation after a peripheral vestibular disorder.

References

1. Baloh R W, Jacbbson K, Honrubia V (1993) Horizontal semicircular canal variant of benign positional vertigo. Neurology 43:2542–2549
2. Black FO, Nashner LM (1984) Postural disturbance in patients with benign paroxysmal positional nystagmus. Ann Otol Rhinol Laryngol 93:595–599
3. Boniver R (1991) Posturographie et vertige paroxystique benign. Acta Oto-Rhino-Laryngol Belg 45:331–334
4. Brandt T (1991) Traumatic otolith vertigo. In: Vertigo. Springer-Verlag, Berlin Heidelberg New York, p 194–195
5. Casani A, Vannucchi G, Fattori B, Ghilardi PL (1997) Positional vertigo and ageotropic bidirectional nystagmus. Laryngoscope 107:807–13
6. Di Girolamo S, Paludetti G, Briglia G, Cosenza A, Santarelli R, di Nardo W (1998) Postural control in benign paroxysmal positional vertigo before and after recovery. Acta Otolaryngol (Stockh) 118:289–293
7. Futaki T, Ikeda T (1989) The auto-tilt test in patients with positional vertigo and Menière's disease. Am J Otol 10:289–292
8. Guidetti G (1985) La posture dans le vertige paroxystique positionnel. RevOto-Neuro-Ophtalmol 5:17–18
9. Katsarkas A, Kearney R (1990) Postural disturbances in paroxysmal positional vertigo. Am J Otol 11:144–148
10. Lempert T, Tiel-Wilck K (1996) A positional maneuver for treatment of horizontal-canal benign positional vertigo. Laryngoscope 106:476–478
11. Lempert T (1994) Horizontal benign positional vertigo. Neurology 44:2213–2214
12. Markham CH, Diamond SG, Ito J (1987) Utricular dysfunction in benign paroxysmal positional vertigo. In: Graham MD, Kemink JL (eds) The vestibular system: neurophysiologic and clinical research. Raven Press, New York
13. McClure J (1985) Horizontal canal BPV. J Otolaryngol 14:30–35
14. Norré ME, Forrez G, Beckers A (1986) Posture testing (posturography) in diagnosis of peripheral vestibular pathology. Arch Oto-Rhino-Laryngol 243:186
15. Norré ME (1990) Posture in otoneurology. Acta Oto-Rhino-Laryngol Belg 44:355–64
16. Nuti D, Agus G, Barbieri MT, Passali D (1998) The management of horizontal-canal paroxysmal positional vertigo. Acta Otolaryngol (Stockh) 118:455–460
17. Nuti D, Vannucchi P, Pagnini P (1996) Benign paroxysmal positional vertigo of the horizontal canal: a form of canalolithiasis with variable clinical features. J Vestib Res 6:173–84
18. Pagnini P, Nuti D, Vannucchi P (1989) Benign paroxysmal vertigo of the horizontal canal. ORL J Otorhinolaryngol Relat Spec 51:161–170
19. Paige GD (1985) Caloric responses after horizontal canal inactivation. Acta Otolaryngol (Stockh) 100:321–327
20. Semont, Freyss G, Vitte E (1988) Curing the BPPV with a liberative maneuver. Adv Oto-Rhino-Laryngol 42:290–293
21. Steddin S, Bandt T (1996) Horizontal canal benign paroxysmal positioning vertigo (H-BPPV): transition of canalolothiasis to cupulolithiasis. Ann Neurol 40:918–922
22. Strupp M, Brandt T, Steddin S, Eng D (1995) Horizontal canal benign paroxysmal positioning vertigo: reversible ipsilateral caloric hypoexcitability caused by canalolithiasis? Neurology 45:2072–2076
23. Takeda N, Nishiike S, Kitahara T, Kubo T, Ogino H, Koizuka I (1997) Clinical features and utricular dysfunction in patients with benign paroxysmal positional vertigo. Nippon Jibiinkoka Gakkai Kaiho 100:449–456
24. Vannucchi P, Giannoni B, Pagnini P (1997) Treatment of horizontal semicircular canal benign paroxysmal positional vertigo. J Vestib Res 7:1–6
25. Vicini C, Campanini A (1993) Standing and walking abnormalities in BPPV; a static and dynamic posturographic study. In: Passali D, Nuti D (eds). Advances in otoneurology. Tipografia senese, Siena, p 109–123