

Benign paroxysmal positional vertigo and tinnitus

Stefania Barozzi¹
Marina Socci¹
Daniela Ginocchio¹
Eliana Filipponi²
Maria Grazia Troja Martinazzoli¹
Antonio Cesarani¹

Abstract

Introduction: In our clinical experience, some of the patients affected by benign paroxysmal positional vertigo (BPPV) reported the onset of tinnitus shortly before or in association with the positional vertigo. **Objectives:** The aim of this study was to describe the prevalence and the clinical patterns of tinnitus episodes which occurred in association with BPPV and to suggest possible interpretative hypotheses. **Methods:** 171 normal hearing patients affected by BPPV (50 males and 122 females; age range: 25-77 years; mean age 60.3 years \pm 14.9) underwent pure tone audiometry, immittance test and a clinical vestibular evaluation before and after repositioning manoeuvres. Those suffering from tinnitus were also assessed using visual analogue scales and tinnitus handicap inventory. **Results:** 19.3% of the patients reported the appearance of tinnitus concurrently with the onset of the positional vertigo. It was mostly unilateral, localized on the same ear as the BPPV, slight in intensity and intermittent. Tinnitus disappeared or decreased in all patients except two, either spontaneously, before performing the therapeutic manoeuvres, or shortly after. **Conclusions:** A possible vestibular origin of tinnitus determined by the detachment of macular debris into the ductus reuniens and cochlear duct is discussed.

Keywords: tinnitus, vertigo, vestibular diseases.

¹ Audiology Unit, Department of Clinical Sciences and Community Health, Università degli Studi di Milano; Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico. E-mail: stefania.barozzi@unimi.it. E-mail: marina.socci@fastwebnet.it. E-mail: daniela.ginocchio@unimi.it. E-mail: magraziatro@tiscali.it. E-mail: antonio.cesarani@unimi.it

² Rehabilitative area, SITRA, Fondazione IRCCS Ca' Granda, - Ospedale Maggiore Policlinico. E-mail: eliana.filipponi@policlinico.mi.it
Institution: Audiology Unit, Department of Clinical Sciences and Community Health, Università degli Studi di Milano; Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico, via Sforza, 35 - 20122 Milano, Italy.

Send correspondence to:

Stefania Barozzi.

Audiology Unit, Department of Clinical Sciences and Community Health, Università degli Studi di Milano; Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico, Via Pace, 9 - 20122 Milano, Italy. E-mail: stefania.barozzi@unimi.it

Paper submitted to the ITJ-SGP (Publishing Management System) on September 9, 2013; and accepted on January 23, 2014. cod. 140

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder in adults, affecting between 17% and 42% of patients complaining of vertigo¹. BPPV is defined by repeated episodes of acute, short, paroxysmal vertigo, provoked by changes in head position relative to gravity. The most common clinical variant is the posterior canal BPPV, which accounts for approximately 85% to 95% of cases¹. Lateral (horizontal) canal BPPV accounts for between 5% and 15% of cases². Other rare forms of BPPV include anterior canal BPPV and multiple canal PPV. Although debated, the most widely accepted pathophysiological hypothesis is the presence of abnormal debris (thought to be fragmented otolithic particles) upon the cupola (cupololithiasis)³ or within the semicircular canals (canal lithiasis)⁴. Diagnosis is made using Dix Hallpike manoeuvre⁵ for posterior canal BPPV and the supine roll test⁶ for the lateral canal BPPV, both of which provoke vertigo associated with the typical paroxysmal nystagmus. BPPV is treated with particle repositioning manoeuvres (Semont manoeuvre, Epley manoeuvre, barbecue roll manoeuvre)⁷⁻⁹ which have the purpose of moving the particles back into the utricle; in the vast majority of cases, the patients recovered after a few sessions.

In our clinical experience, some of the patients affected by BPPV reported the onset of tinnitus shortly before or in association with the positional vertigo. Such tinnitus often decreased or disappeared after the therapeutic manoeuvres; tinnitus rarely appeared immediately after the manoeuvres. In literature, only one study performed by Gavalas et al.¹⁰ describes tinnitus of vestibular origin. The authors observed that this tinnitus disappeared immediately after the Semont and Epley manoeuvres in some patients and attributed this to a reduction in autonomic activity.

The aim of this study was to describe the prevalence and the clinical patterns of tinnitus episodes which occurred in association with BPPV and to suggest possible pathophysiological mechanisms.

METHODS

A total of 171 normal hearing patients affected by BPPV, 50 males and 122 females, of an age ranging from 25 to 77 years (mean age 60.3 years \pm 14.9) were enrolled in this study. BPPV had been diagnosed according to the criteria proposed by the American Academy of Otolaryngology - Head and Neck Surgery (2008) when the patients reported a history of repeated episodes of vertigo provoked by changes in head position relative to gravity and when, upon physical examination, characteristic nystagmus was provoked by the positioning manoeuvres. 136 patients (79.5%) had posterior canal

BPPV, 24 (14%) lateral canal BPPV, 9 (5%) multiple canal BPPV and 2 (1.1%) anterior canal BPPV.

The exclusion criteria were: external or middle ear diseases, temporomandibular joint dysfunctions, a pure tone threshold at 0.5, 1.0 and 2.0 kHz (PTA) > 25 dB HL, BPPV resistant to three repositioning manoeuvres.

The patients were divided into two groups: Group 1 included subjects who did not suffer from tinnitus or who had been suffering from tinnitus for at least one month before the onset of BPPV; Group 2 consisted of subjects whose tinnitus had appeared in association with BPPV or during the previous month.

All of the patients underwent an audiovestibular evaluation including:

- Detailed history with special attention to tinnitus. Tinnitus subjects belonging to Group 2 were also assessed using Visual Analogue Scales (VAS) from 1 to 10 (in terms of volume and disturbance), and the Italian version of the Tinnitus Handicap Inventory (THI)¹¹.
- Otoloscopic examination in order to exclude possible external ear and tympanic membrane pathologies.
- Pure-tone audiometry performed in a sound-attenuated booth using an Amplaid 309 audiometer (Amplifon, Italy) and calibrated earphones (TDH 49). Pure-tone thresholds were measured in each ear separately at the frequencies of 0.25-8 kHz for air conduction and 0.25-4 kHz for bone conduction. Patients with a PTA > 25 dB were excluded from this study.
- Tympanometry and measurement of acoustic reflex in order to study middle ear function using the Amplaid A766 Middle Ear Analyzer (Amplifon, Italy) with a 226-Hz probe. The tympanograms were classified as *type A* (normal middle ear pressure), *type B* (flat curve), *type C* (negative peak pressure). In patients with *type A* tympanograms, we determined the contralateral acoustic reflex using pure-tone signals at 0.5-4 kHz. Patients with middle ear dysfunctions defined by tympanograms other than *type A* and/or absence of acoustic reflex were excluded.
- Clinical vestibular examination, including Dix-Hallpike manoeuvre and supine roll test.

After diagnosing BPPV, all of the patients were treated with 1-3 repositioning manoeuvres: Semont or Epley manoeuvres for posterior canal BPPV and barbecue manoeuvre for lateral canal BPPV.

Seven days after each manoeuvre, patients were retested to verify the disappearance of both vertigo and paroxysmal nystagmus. If the manoeuvre had been successful, Group 1 patients were asked if they had noticed the appearance of tinnitus and Group 2 patients were asked if their tinnitus had changed; if the tinnitus was still present, they repeated VAS and THI.

RESULTS

Of the 171 BPPV patients, 138 (80.7%) belonged to Group 1 and 33 (19.3%) to Group 2.

For each group, age and gender of the patients, type (semicircular canal involved) and side of BPPV are reported in Table 1.

Group 2 patients described tinnitus as: ringing/ticking (9 patients), buzzing (6), hissing (5), creaking (5), blowing (3), like the sea (3) and pounding (2). Tinnitus was intermittent in 25 (75.8%) patients, with a duration of few seconds/minutes and continuous in 8 (24.2%). Tinnitus was unilateral and localized in the same ear as BPPV in 75.8% (25) of the patients, and in the contralateral ear in 18.2% (6). Two patients complained of bilateral tinnitus. Tinnitus was modified by changes of head position in 30.3% (10) of the patients.

The intensity of tinnitus, as shown by the VAS scale was < 5 in 28 (84.8%) patients and > 5 in 5 (15.2%) patients. The mean THI score was: 25.2 ± 7.2 .

In 24.2% (8) of the patients, the tinnitus, which had originated in association with BPPV, had already disappeared spontaneously before the therapeutic manoeuvres were performed. At the control visit, after a successful manoeuvre, tinnitus had disappeared in another 48.5% (24) of the patients. In 18.2% (6) it had decreased in intensity and duration; only in 2 patients did it remain unchanged.

In 2 patients belonging to Group 1, a transitory tinnitus appeared after the repositioning manoeuvres, but resolved spontaneously in a few days.

DISCUSSION

As far as we know, this is one of the few studies dealing with tinnitus in BPPV.

In our experience, 19.3% of the patients affected by BPPV reported the appearance of tinnitus in association with the onset of positional vertigo. It was mostly unilateral and localized in the same ear as the BPPV, slight in intensity and intermittent. In about one third of the subjects it was modified by changes of head position. Tinnitus disappeared or decreased in all patients except two, either spontaneously, before performing the therapeutic manoeuvres, or immediately after them. Conversely, the manoeuvres provoked temporary tinnitus in 2 patients.

Gavalas et al.¹⁰ observed that, in some patients, tinnitus associated with recent vestibular symptomatology, was reduced after Semont and Epley exercises. How it is possible to explain the presence of tinnitus in BVPP and/or its disappearance through rehabilitative procedures?

There are three possible pathophysiological hypotheses.

The existence of anatomical connections between the vestibular and cochlear systems could justify the involvement of the auditory pathways secondary to a labyrinth stimulus and the occurrence of tinnitus in BPPV patients. Nervous connections between the two systems have been described in the internal auditory canal, at the nuclear level and also in the auditory cortex¹²⁻¹⁴. The existence of these neural fibre connections explains the persistence of tinnitus after cochlear neurectomy performed for intractable tinnitus or the modification of some kind of tinnitus through caloric vestibular stimulation. It is unlikely, however, that this could justify the tinnitus of patients in which it does not coincide in time with the positioning manoeuvres.

According to the model of Jastreboff, tinnitus may be caused by the interaction between the vestibular and the limbic systems. In fact, the vertiginous symptoms can be so acute and intense as to induce emotional responses such as fear and terror of provoking the vertigo through the movements. The disappearance of the tinnitus could be due to the relief they experience when symptoms resolve. According to Gavalas et al.¹⁰, it is exactly this reduction in autonomic activity that could mediate the decrease of tinnitus after repositioning manoeuvres. However, this theory does not explain the appearance of tinnitus before the onset of positional vertigo as experienced by some of our patients.

The third hypothesis considers changes in the hydrodynamics of the inner ear fluids. There are two main theories concerning the secretion and absorption of endolymph in the membranous labyrinth. In the "longitudinal flow theory"¹⁴, the endolymph of the cochlea is produced in the scala media and normally flows through the ductus reuniens, the saccule and the endolymphatic duct where it is reabsorbed; in the vestibular system, on the other hand, there is a flow of endolymph from the utricle and semicircular canals towards the endolymphatic sac. According to the "theory of radial flow", the endolymph is produced and absorbed locally in the scala media and utricle (there

Table 1. Descriptive statistics for Group 1 and Group 2 patients. Age, gender, type (semicircular canal involved) and side of BPPV are reported.

	N.	Age (years) (Min/Max/Med)	Gender (males/females)	Semicircular Canal Post/Lat/Ant/Multi	Side (right/left/bilat.)
Group 1	138 (80,7%)	25 77 59.3	43 95	113 18 0 7	68 63 7
Group 2	33 (19.3%)	39 77 59.9	7 26	23 6 2 2	17 14 2

Inclusion criteria required that all of the patients had normal hearing sensitivity.

are no secretory cells - dark cells - in the saccule)¹⁵ In both cases, it seems unlikely that debris upon the cupola or within the semicircular canals in BPPV can cause an acute modification of the cochlear endolymphatic pressure. It is possible, however, according to the theory of radial flow, that otoliths or saccular macular debris may slide through the ductus reuniens into the cochlear canal. Gussen¹⁶ reported that the atrophy of the saccular macula in humans causes an accumulation of otolith debris within the ductus reuniens and cochlear duct. According to this theory, just as the detachment of debris from the utricle into the semicircular canals determines vertiginous episodes in BPPV, the detachment of macular debris from the saccule into the ductus reuniens and cochlear duct might result in tinnitus. This theory would also explain why some kind of tinnitus in patients affected by recurrent BPPV can disappear with liberatory manoeuvres also during periods of remission from vertiginous symptoms.

In our study, the ear affected by tinnitus corresponds in most cases to the side of BPPV; this could be helpful to the physician before performing repositioning manoeuvres when the side of BPPV is uncertain, as in some cases of lateral canal BPPV.

CONCLUSIONS

In some patients with BPPV, a characteristic tinnitus may appear, mostly slight, intermittent and localized in the same ear affected by BPPV; it often regresses either spontaneously or after repositioning manoeuvres.

REFERENCES

- Bhattacharyya N, Baugh RF, Orvidas L, Barrs D, Bronston LJ, Cass S, et al.; American Academy of Otolaryngology-Head and Neck Surgery Foundation. Clinical practice guideline: benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 2008;139(5 Suppl 4):S47-81. PMID: 18973840 DOI: <http://dx.doi.org/10.1016/j.otohns.2008.08.022>
- Cakir BO, Ercan I, Cakir ZA, Civelek S, Sayin I, Turgut S. What is the true incidence of horizontal semicircular canal benign paroxysmal positional vertigo? *Otolaryngol Head Neck Surg.* 2006;134(3):451-4. PMID: 16500443 DOI: <http://dx.doi.org/10.1016/j.otohns.2005.07.045>
- Schuknecht HF. Cupulolithiasis. *Arch Otolaryngol.* 1969;90(6):765-78. DOI: <http://dx.doi.org/10.1001/archotol.1969.00770030767020>
- Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. *Laryngoscope.* 1992;102(9):988-92. PMID: 1518363
- Dix MR, Hallpike CS. The pathology symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc R Soc Med.* 1952;45(6):341-54. PMID: 14941845
- Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). *CMAJ.* 2003;169(7):681-93. PMID: 14517129
- Semont A, Freyss G, Vitte E. Curing the BPPV with a liberatory maneuver. *Adv Otorhinolaryngol.* 1988;42:290-3. PMID: 3213745
- Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 1992;107(3):399-404. PMID: 1408225
- Lempert T, Tiel-Wilck K. A positional maneuver for treatment of horizontal-canal benign positional vertigo. *Laryngoscope.* 1996;106(4):476-8. PMID: 8614224 DOI: <http://dx.doi.org/10.1097/00005537-199604000-00015>
- Gavalas GJ, Passou EM, Vathilakis JM. Tinnitus of vestibular origin. *Scand Audiol Suppl.* 2001;(52):185-6. DOI: <http://dx.doi.org/10.1080/010503901300007470>
- Monzani D, Genovese E, Marrara A, Gherpelli C, Pingani L, Forghieri M, et al. Validity of the Italian adaptation of the Tinnitus Handicap Inventory; focus on quality of life and psychological distress in tinnitus-sufferers. *Acta Otorhinolaryngol Ital.* 2008;28(3):126-34.
- Ozdoğmuş O, Sezen O, Kubilay U, Saka E, Duman U, San T, et al. Connections between the facial, vestibular and cochlear nerve bundles within the internal auditory canal. *J Anat.* 2004;205(1):65-75. DOI: <http://dx.doi.org/10.1111/j.0021-8782.2004.00313.x>
- Fredrickson JM, Scheid P, Figge U, Kornhuber HH. Vestibular nerve projection to the cerebral cortex of the rhesus monkey. *Exp Brain Res.* 1966;2(4):318-27. DOI: <http://dx.doi.org/10.1007/BF00234777>
- Odkvist LM, Rubin AM, Schwarz DW, Fredrickson JM. Vestibular and auditory cortical projection in the guinea pig (*Cavia porcellus*). *Exp Brain Res.* 1973;18(3):279-86. DOI: <http://dx.doi.org/10.1007/BF00234598>
- Kimura RS. Distribution, structure, and function of dark cells in the vestibular labyrinth. *Ann Otol Rhinol Laryngol.* 1969;78(3):542-61.
- Gussen R. Saccule otoconia displacement into cochlea in cochleosaccular degeneration. *Arch Otolaryngol.* 1980;106(3):161-6. PMID: 7356437 DOI: <http://dx.doi.org/10.1001/archotol.1980.00790270025006>