Eur Arch Otorhinolaryngol (2014) 271:919–924
DOI 10.1007/s00405-013-2484-2

OTOLOGY

# Benign paroxysmal positional vertigo secondary to vestibular neuritis

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Received: 22 December 2012 / Accepted: 2 April 2013 / Published online: 11 April 2013


**Abstract** The aim of this study was to present the demographic, pathogenetic and clinical features of benign paroxysmal positional vertigo (BPPV) secondary to vestibular neuritis (VN). The medical records of 22 patients, who presented with BPPV within 12 weeks after the onset of VN, were reviewed. Data of a complete otolaryngological, audiological, neurotologic and imaging evaluation were available for all patients. Two hundred and eighty-four patients with idiopathic BPPV were used as a control group. The patients with BPPV secondary to VN presented the following features, in which they differed from the patients with idiopathic BPPV: (1) a lower mean age; (2) involvement of the posterior semicircular canal; (3) presence of canal weakness; (4) more therapeutic sessions needed for cure and a higher rate of recurrence. It may be, thus, concluded that BPPV associated with VN differs from idiopathic BPPV in regard to several epidemiological and clinical features, it responds less effectively to treatment and may follow a protracted course, having a tendency for recurrence.

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**Keywords** Benign paroxysmal positional vertigo · Vestibular neuritis · Vertigo · Hearing loss · Nystagmography · Canalith repositioning procedure

## Introduction

Vestibular neuritis (VN) is a common peripheral vestibular disease, characterized by a sudden onset of vertigo due to unilateral loss of vestibular function. Its main clinical features are severe, incapacitating vertigo and disequilibrium, accompanied by nausea and vomiting [1, 2]. Absence of neurological signs or hearing loss is a distinctive feature of this clinical entity. In most patients, symptoms largely resolve over a period of weeks, but more protracted courses and recurrences commonly occur.

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disease, which can be defined as temporary vertigo induced by a rapid change in head position, accompanied by a characteristic paroxysmal positional nystagmus. The nystagmus may be torsional-vertical, or horizontal and is characterized by findings such as latency, crescendo—decrescendo, transience, reversibility, and fatigability [3, 4]. Although BPVV in most patients is idiopathic, it may be also secondary to various other conditions, such as head trauma, inner ear disease, or it may be the result of surgery and prolonged bed rest [5]. It seems that any inner ear disease that detaches otoliths and yet does not completely damage the function of the posterior semicircular canal (SCC), may induce secondary BPPV. It has been reported that idiopathic and secondary BPPV differ on several aspects, including treatment outcome and prognosis, implying different pathology or pathophysiology of these clinical entities [6].

BPPV secondary to VN has been occasionally reported and studied, mainly in the context of BPPV associated to inner ear disease [7, 8]. However, the specific epidemiological, pathophysiologic, and clinical features of this clinical entity have not been adequately defined. The aim of this study was to investigate a group of patients who presented with BPPV as a consequence of VN, diagnosed and treated in the Neurotology Unit of an ENT Department, during the past 6 years. The demographic, pathogenetic and clinical features of these patients were studied and compared with those of patients with idiopathic BPPV.

## Materials and methods

During the past 6 years, 422 patients examined at the Neurotology Unit of our Department were diagnosed with BPPV. Among them 22 patients had recently (during the previous 12 weeks) suffered from VN and were included in the study. Cases with a history of more remote occurrence of VN were excluded, because either substantial data might be missing or BPPV might not be secondary to VN, but could be idiopathic or owed to other causes, since the causative relation of the two conditions could not be proven. The limit of 12 weeks was accepted as in most patients symptoms have resolved and canal paresis (when not permanent) is restored to normal [9].

Diagnosis of VN was obtained if the following criteria were fulfilled [7, 10]: (1) history of a single attack of sudden spontaneous vertigo, lasting for at least 24 h and slowly decreasing over days; (2) concomitant horizontal unidirectional spontaneous nystagmus; (3) unilateral canal paresis on caloric testing (>25 % side difference according to Jongkees formula); (4) absence of relevant auditory symptoms or findings; (5) no additional neurological signs or symptoms, and normal brain imaging.

Posterior (or anterior) SCC BPPV was diagnosed with the use of the Dix-Hallpike maneuver, whereas the supine roll test was used for the diagnosis of horizontal canal BPPV [3]. Posterior SCC BPPV was treated by the modified Epley canalith repositioning procedure (CRP) and horizontal SCC BPPV was treated by the barbecue or the Gufoni maneuver. Repeat treatment was performed after 2–3 days from initial treatment, in case of failure or incomplete remission of the symptoms. Assessment of the success of the treatment included both the patient's report of relief from vertigo and a negative Dix-Hallpike test or supine roll test result, for at least 2 months. In case of recurrences, CRPs were repeated, according to the same schedule.

The clinical records of these patients were reviewed and the following data were recorded: age, sex, elapsed time from onset of VN, side of disease, and SCC involved. The severity of the vertiginous symptoms was evaluated according to the following scale [11]: (1) slight vertigo in the provoking position without autonomous symptoms; (2) severe vertigo with nausea; (3) severe vertigo with severe nausea, vomiting, or hypotension. Patients with any clinical, laboratory or imaging findings suggesting pathology of the central nervous system were excluded. Patients with idiopathic BPPV examined and treated during the same period, were used as controls. The protocol of the study was reviewed and approved by the local Institutional Review Board.

All patients underwent a detailed otolaryngologic, audiological and neurotologic evaluation, including audiometry, measurements of acoustic immittance and, occasionally, auditory brainstem responses. Eye movements were recorded by electro- or video-nystagmography using a standard test protocol of visual and vestibular stimulation, described elsewhere [12]. Follow-up data were available for most patients for at least 1 year.

### Statistical analysis

Categorical variables were expressed as frequencies and percentages and continuous variables were expressed as mean ± standard deviation. The Chi-square test was used to evaluate any potential association between categorical variables. The significance of any difference between groups was evaluated by *t* test for independent samples. Odds ratio with 95 % confidence interval were calculated for the estimation of treatment results. The adopted level of statistical significance was 0.05.

## Results

We found 22 patients with BPPV secondary to recent VN, indicating a prevalence of 5.2 % of this clinical entity in patients with BPPV. Nine of these patients had been hospitalized in our setting and the remaining 13 had been diagnosed and treated elsewhere. In all patients, detailed clinical and laboratory data were available for study. Another 23 (5.4 %) patients had a history of VN of more remote origin, and were excluded from this study. From the remaining 377 patients with BPPV, 284 (67.3 %) patients had idiopathic BPPV and were used for comparison (these will be further referred as controls), whereas another 93 patients (22 %) were diagnosed with secondary BPPV due to other possible pathogenetic factors, such as head trauma or Meniere's disease. The demographic and clinical features of patients with BPPV secondary to VN and of the control group with idiopathic BPPV are shown in Table 1 and are compared statistically.

Seventeen patients were female (mean age 41.1 ± 9.7; range 31–64 years) and five were male (mean age

**Table 1** Demographic and clinical features of patients with BPPV secondary to VN and in patients with idiopathic BPPV

| | BPPV with VN ($N = 22$) | Idiopathic BPPV ($N = 284$) | $p$ value |
|---|---|---|---|
| Gender | | | |
| Male | 5 (22.7 %) | 114 (40.1 %) | 0.19 |
| Female | 17 (77.3 %) | 170 (59.9 %) | |
| Age (years) | | | |
| Mean | 41.5 ± 9.7 | 54.4 ± 16.5 | <0.001 |
| Range | 31–64 | 24–86 | |
| Elapsed time from onset of VN (days) | | | |
| Mean | 22.2 ± 18.5 | | |
| Range | 3–75 | | |
| Side of involvement | | | |
| R | 12 (54.5 %)[a] | 165 (58.1 %) | 0.32 |
| L | 10 (45.5 %)[a] | 99 (34.9 %) | |
| Bilateral | – | 20 (7.0 %) | |
| Semicircular canal involved | | | |
| Posterior | 22 (100 %) | 219 (77.1 %) | <0.05 |
| Horizontal | – | 31 (10.9 %) | |
| Anterior-multiple | – | 34 (12.0 %) | |
| Vertigo severity | | | |
| 1 | 0 | 28 (9.9 %) | <0.001 |
| 2 | 9 (41.0 %) | 228 (80.2 %) | |
| 3 | 13 (59.0 %) | 28 (9.9 %) | |

[a] Involvement was always on the same side with VN

42.4 ± 10.8; range 33–60 years). Neurologic examination was normal in all patients, but to exclude a central disorder, in all patients magnetic resonance imaging of the brain had been performed, which was normal. The mean elapsed time from the onset of VN until manifestation of BPPV was 22.2 (±18.5) days (range 3–75 days). Audiological investigation proved hearing within normal limits for all patients and none of them had a history of vascular disease.

All patients had unilateral BPPV of the posterior SCC, on the side ipsilateral to the involved side during VN. No patient with involvement of the anterior, horizontal canal or multiple canals was found. Thirty-one patients from the control group had horizontal canal involvement and another 34 had multiple or anterior canal BPPV. In the majority of patients with BPPV secondary to VN (59.0 %) vertigo was intense with accompanying symptoms (grade 3). Statistically significant differences in the patient group, as compared to the control group of patients with idiopathic BPPV, were found in the following variables: age (younger); SCC involvement (absence of horizontal, anterior or multiple canal BPPV); vertigo severity (more intense). Gender and side of involvement did not differ significantly between groups.

We performed video- or electro-nystagmography in all patients and compared statistically the results with those of a group of 78 controls, recruited from the group of patients with idiopathic BPPV (Table 2). Seventeen patients (77.3 %) of the first group had a mild or moderate spontaneous nystagmus of peripheral type, without optic fixation. In 12 of them slow phase velocity exceeded 6°/sec. The nystagmus was horizontal with the fast phase beating always towards the normal ear. All patients of this group had also unilateral canal weakness (mean 65.2 % ± 18.5, range 35–100 %).

Spontaneous nystagmus was observed in only three (3.8 %) patients of the idiopathic BPPV group and canal weakness was found in 19 (24.3 %) of them. In addition, 14 (63.6 %) patients with BPPV secondary to VN had directional preponderance, always towards the healthy side, in comparison to 17 patients (21.8 %) from the control group with idiopathic BPPV who had directional preponderance, either as a sole finding or in conjunction with canal paresis. No central nystagmographic findings were found, with the exception of one patient with idiopathic BPPV who had impaired fixation suppression of the vestibulo-ocular reflex. Statistically significant differences between the two groups in all the nystagmographic measures of peripheral vestibular dysfunction were found.

We performed the modified Epley CRP in all patients with BPPV secondary to VN and obtained successful treatment in 13 (59.1 %) of them, after one (3 patients) or multiple (10 patients) treatment sessions (Table 3). Comparison of treatment success between patients with BPPV secondary to VN and controls with idiopathic BPPV yielded an odds ratio of 8.67 (95 % CI 3.32–22.62; $p < 0.001$), in favor of patients with idiopathic BPPV. Furthermore,

**Table 2** Nystagmographic findings in patients with BPPV secondary to VN and in patients with idiopathic BPPV

| Nystagmographic findings | BPPV secondary to VN ($N = 22$) | Idiopathic BPPV ($N = 78$) | $p$ value |
|---|---|---|---|
| Spontaneous nystagmus | 17 (77.3 %) | 3 (3.8 %) | <0.001 |
| Canal paresis | 22 (100.0 %) | 19 (24.3 %) | <0.001 |
| Bilateral | 0 | 6 | |
| Unilateral | 22 | 13 | |
| Diseased ear | 22 | 10 | |
| Normal ear | 0 | 3 | |
| Directional preponderance | 14 (63.6 %) | 17 (21.8 %) | <0.001 |
| Alone | 0 | 12 | |
| With canal weakness | 14 | 5 | |
| Central findings | 0 | 1 (1.3 %) | 0.59 |

**Table 3** Treatment results in patients with BPPV secondary to VN and in patients with idiopathic BPPV

| | *N* (%) | Success of 1st treatment (%) | Success of repeat treatment (%) | Failures (%) |
|---|---|---|---|---|
| BPPV secondary to VN | | | | |
| Posterior SCC BPPV | 22 (100.0) | 3 (13.6) | 10 (45.5) | 9 (40.9) |
| Idiopathic BPPV | | | | |
| Posterior SCC BPPV | 219 (77.1) | 192 (67.6) | 11 (3.9) | 16 (5.6) |
| Horizontal SCC BPPV | 31 (10.9) | 18 (6.3) | 10 (3.5) | 3 (1.1) |
| Anterior SCC BPPV | 17 (6.0) | 11 (3.9) | 6 (2.1) | – |
| Multiple canal BPPV | 17 (6.0) | – | 15 (5.3) | 2 (0.7) |
| Total | 284 (100) | 221 (78.9) | 42 (13.7) | 21 (7.4) |

patients with BPPV secondary to VN needed more therapeutic sessions for treatment of the disease, since in only 13.6 % of them CRPs were initially successful, vs. 78.9 % in the idiopathic group. Patients who did not respond to treatment were advised to follow a program of Brandt–Daroff exercises [13] and in case of failure were offered surgery. In 19 patients, a follow-up of more than 12 months was obtained. Among 12 of them, in whom treatment had been successful, eight patients (66.6 %) presented recurrence of BPPV, as compared to 13.5 % recurrence rate in the idiopathic BPPV group ($p < 0.001$). In the remaining seven patients, who did not respond favorably to treatment, the disease persisted in spite of vestibular rehabilitation, with the exception of one patient who recovered after a few months. However, none of these patients has consented to surgical treatment yet.

## Discussion

BPPV secondary to VN, has been occasionally reported, in the context of BPPV owed to inner ear disease [7, 8, 14]. However, several issues concerning the incidence, the pathogenesis, the specific clinical characteristics and any particularity in the treatment and prognosis of this clinical entity are not still quite clear. Most studies included all cases of BPPV, with a history positive to VN, either of recent or of late onset [10, 15, 16]. In the present study, we included only patients with BPPV of recent onset after VN. Accordingly, 22 patients with BPPV secondary to VN, with complete medical records including brain imaging studies and nystagmographic data, were available for study.

In most reports of patients with VN, the incidence of BPPV complicating its course appears to range approximately from 10 to 15 %. For example, Huppert et al. [17] reported that among 103 patients who had VN, BPPV occurred in 14 patients (13.6 %). Kim et al. [16] found that 20 out of 131 patients (15.3 %) with VN, experienced BPPV during the follow-up period. In addition, Mandala et al. [10], in a study of a group of 51 patients with VN, reported that BPPV was more frequent (5/51 patients, 9.8 %) in VN patients than in the general population. However, Bergenius and Perols [18] suggested a higher occurrence of secondary BPPV (31.6 %), in a long-term follow-up of patients with VN, but the authors admitted that a Dix-Hallpike positioning test was not specifically performed in their patients. It could be, thus, speculated that several patients might had suffered from some other type of positional vertigo and not true BPPV. In addition, Murofushi et al. [19] reported an occurrence of secondary BPPV in a higher rate of patients (21.3 %).

On the other hand, in various studies of BPPV, reported rates of secondary BPPV, especially in cases owed to VN, are inconsistent, ranging from 0.8 to 24 % [6]. Karlberg et al. [7] found only 24 patients with VN in a large group of 2,847 patients (0.8 %), although VN was the most common cause of BPPV secondary to inner ear disease. Lee et al. [8] found an incidence of 1.8 % of BPPV secondary to VN, with an average onset of 17.9 days (range of 0–50 days). Caldas et al. [15], also, found occurrence of this clinical entity in 3 % of their patients, whereas Roberts et al. [14], in a study of vestibulopathy in BPPV patients with and without prior otologic history, reported the presence of 37 out of 157 patients (23.5 %) with an otologic diagnosis of vestibular neuritis/labyrinthine ischemia.

We found a prevalence of 5.2 % of BPPV secondary to VN in our patients, whereas another 5.4 % of the patients had a history of VN of more remote origin, and were excluded from this study. However, a bias towards more increased rates than the actual ones could be hypothesized, since our setting is specialized in the management of patients with BPPV and frequently, patients with this disease, presenting difficulties in diagnosis and treatment, are referred from other settings.

The pathogenetic mechanism underlying BPPV secondary to VN seems to derive from the distribution of the vestibular nerve in the inner ear [20]. The superior vestibular nerve innervates the cristae of the anterior and lateral SCCs and the macula of the utricle. VN, usually of viral etiology, often preferentially affects the superior vestibular nerve and the structures it innervates. Gianoli et al. [21] have shown that anatomic features may explain this predilection: the bony canal of the superior vestibular nerve is longer and has more spicules than the singular and the inferior vestibular nerve. Consequently, the superior

vestibular nerve may be more susceptible to conduction problems in the inflammation [22]. Impairment of lateral SCC and the superior vestibular nerve function is associated with vertigo, horizontal spontaneous nystagmus and a canal paresis on caloric testing. Simultaneous damage to the utricle could detach the otoconia and cause posterior canal BPPV, if the otoconia enters the posterior SCC duct. Morgenstein and Seung [23] reported degeneration in the utricular neuroepithelium in a patient with VN, which favors this hypothesis. Manifestation of posterior canal BPPV implies either preservation of at least some function in the inferior vestibular nerve or recovery of its function despite initial involvement. This is also supported by the presence of vestibular-evoked myogenic potentials in postneurolabyrinthitis patients [19]. These potentials are of saccular origin and both the macula of the saccule and the crista of the posterior canal are innervated by the inferior vestibular nerve.

The following findings from our study support the above pathogenetic mechanism: (1) all our patients, as well as the subjects in previous reports [7, 10], had BPPV on the same side which had been involved during VN, implying a pathogenetic relation between the two conditions; (2) BPPV was characterized by severe clinical symptoms and difficulty in treatment, whereas all patients had canal paresis, indicating severe nonresolved peripheral vestibular involvement after the course of VN; (3) BPPV secondary to VN was always of the posterior canal type. Occasionally, cases of BPPV secondary to VN, with horizontal or anterior canal BPPV have been reported [8], owed probably either to involvement of the inferior vestibular nerve or to typical superior VN, with incomplete paresis or functional recovery of the horizontal or the anterior canal.

Another possible pathogenetic mechanism, originally proposed by Hemenway and Lindsay [24], is ischemic distress related to anterior vestibular artery, which supplies the horizontal semicircular canal and the utricle. Ischemic necrosis in this territory is characterized by an initial episode of acute vertigo followed by benign paroxysmal positional vertigo, owed to degeneration of otolithic macula and accumulation of otoconia into the semicircular canals. Although in our patients, findings of brain ischemia in magnetic resonance imaging were not evident, this etiology cannot be excluded, at least in some cases, and may explain the increased rate of recurrences, as well as the lower success rate of CRPs.

BPPV secondary to VN has been categorized according to the time of manifestation as of early or of late onset. In the first category, cases within 1–3 months after the onset of the primary disease have been included [20]. In our study group, in which only patients of the first category were included, mean elapsed time from onset of VN was 22 days, which agrees with other reports [8]. However, cases with onset as early as 0 days [25] and as late as 20 years [20], have been reported. Our patients with secondary BPPV were younger than those with idiopathic BPPV (mean age 41.5 versus 54.4), which agrees with some previous reports [8, 20, 26] and their clinical picture was more severe. The latter finding has not been specifically reported so far, but it may be concluded from the fact that difficulties in treatment and frequent recurrences have been admitted [8, 10].

Treatment of patients with BPPV secondary to VN appears to be less effective and more time consuming than that of patients with idiopathic BPPV. We obtained treatment with the first session of CRP in only 13.6 % of our patients and 45.5 % needed multiple sessions, whereas in 40.9 % treatment was not successful and the disease followed a protracted course. The corresponding percentages in idiopathic BPPV were statistically significantly better, with corresponding values of 78.9, 13.7, and 7.4 %. In several previous reports, multiple sessions of treatment [27] and increased rate of recurrences have been found [16]. Lee et al. [8] reported mean duration of treatment 2.28 days for idiopathic BPPV and 5.07 days for unilateral vestibulopathy, which was significantly longer. Mandalà et al. [10] found 3 out of 5 patients with BPPV secondary to VN, who had recurrent (more than three) episodes of BPPV, and three patients with BPPV difficult to treat (multiple CRPs). We found high recurrence rates, in accordance with Huppert et al. [17], who reported recurrent BPPV (more than three episodes) in 71.4 % of their patients.

Worse treatment results and higher rate of recurrences in BPPV secondary to VN might challenge the typical explanation of BPPV, as originating from cupulo/canalolithiasis. In some of these patients, a different pathogenetic mechanism could be implicated, such as brainstem ischemia, other abnormalities of the function of the central nervous system, or even metabolic or hormonal dysfunction [28]. Anterior vestibular artery occlusion might also cause an extensive utricular damage and increased rate of failure of CRPs, as previously mentioned.

## Conclusion

In conclusion, patients with BPPV secondary to VN differed from patients with idiopathic BPPV in the following ways: (1) a lower mean age; (2) involvement of the posterior SCC only; (3) presence of canal weakness; (4) poorer treatment results and higher rate of recurrence. The above findings may indicate that BPPV secondary to VN differs from idiopathic BPPV on several demographic and clinical aspects, that it is more resistant to treatment and that it may follow a protracted course, having a tendency for recurrence.

## References


1. Ryu JH (1993) Vestibular neuritis: an overview using a classical case. Acta Otolaryngol Suppl 503:25–30
2. Baloh RW (2003) Clinical practice. Vestibular neuritis. N Engl J Med 348(11):1027–1032
3. Korres SG, Balatsouras DG (2004) Diagnostic, pathophysiologic, and therapeutic aspects of benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 131:438–444
4. Balatsouras DG, Koukoutsis G, Ganelis P, Korres GS, Kaberos A (2011) Diagnosis of single- or multiple-canal benign paroxysmal positional vertigo according to the type of nystagmus. Int J Otolaryngol 2011:483965
5. Korres S, Balatsouras DG, Kaberos A, Economou C, Kandiloros D, Ferekidis E (2002) Occurrence of semicircular canal involvement in benign paroxysmal positional vertigo. Otol Neurotol 23:926–932
6. Riga M, Bibas A, Xenellis J, Korres S (2011) Inner ear disease and benign paroxysmal positional vertigo: a critical review of incidence, clinical characteristics, and management. Int J Otolaryngol 2011:709469
7. Karlberg M, Hall K, Quickert N, Hinson J, Halmagyi GM (2000) What inner ear diseases cause benign paroxysmal positional vertigo? Acta Otolaryngol 120:380–385
8. Lee NH, Ban JH, Lee KC, Kim SM (2010) Benign paroxysmal positional vertigo secondary to inner ear disease. Otolaryngol Head Neck Surg 143:413–417
9. Matsuo T, Sekitani T (1985) Vestibular neuronitis: neurotological findings and progress. ORL J Otorhinolaryngol Relat Spec 47: 199–206
10. Mandalà M, Santoro GP, Awrey J, Nuti D (2010) Vestibular neuritis: recurrence and incidence of secondary benign paroxysmal positional vertigo. Acta Otolaryngol 130:565–567
11. De la Meilleure G, Dehaene I, Depondt M, Damman W, Crevits L, Vanhooren G (1996) Benign paroxysmal positional vertigo of the horizontal canal. J Neurol Neurosurg Psychiat 60:68–71
12. Korres SG, Balatsouras DG, Ferekidis E (2004) Electronystagmographic findings in benign paroxysmal positional vertigo. Ann Otol Rhinol Laryngol 113:313–318
13. Brandt T, Daroff RB (1980) Physical therapy for benign paroxysmal positional vertigo. Arch Otolaryngol 106:484–485
14. Roberts RA, Gans RE, Kastner AH, Listert JJ (2005) Prevalence of vestibulopathy in benign paroxysmal positional vertigo patients with and without prior otologic history. Int J Audiol 44:191–196
15. Caldas MA, Ganança CA, Ganança FF, Ganança MM, Caovilla HH (2009) Clinical features of benign paroxysmal positional vertigo. Braz J Otorhinolaryngol 75:502–506
16. Kim YH, Kim KS, Kim KJ, Choi H, Choi JS, Hwang IK (2011) Recurrence of vertigo in patients with vestibular neuritis. Acta Otolaryngol 131:1172–1777
17. Huppert D, Strupp M, Theil D, Glaser M, Brandt T (2006) Low recurrence rate of vestibular neuritis: a long-term follow-up. Neurology 28(67):1870–1871
18. Bergenius J, Perols O (1999) Vestibular neuritis: a follow-up study. Acta Otolaryngol 119:895–899
19. Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG (1996) Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis. An indicator of inferior vestibular nerve involvement? Arch Otolaryngol Head Neck Surg 122:845–848
20. Harada K, Oda M, Yamamoto M, Nomura T, Ohbayashi S, Kitsuda C (1993) A clinical observation of benign paroxysmal positional vertigo (BPPV) after vestibular neuronitis (VN). Acta Otolaryngol Suppl 503:61–63
21. Gianoli G, Goebel J, Mowry S, Poomipannit P (2005) Anatomic differences in the lateral vestibular nerve channels and their implications in vestibular neuritis. Otol Neurotol 26:489–494
22. Fetter M, Dichgans J (1996) Vestibular neuritis spares the inferior division of the vestibular nerve. Brain 119(Pt 3):755–763
23. Morgenstein KM, Seung HI (1971) Vestibular neuronitis. Laryngoscope 81:131–139
24. Hemenway WG, Lindsay JR (1956) Postural vertigo due to unilateral sudden partial loss of vestibular function. Ann Otol Rhinol Laryngol 65:692–706
25. Zapala DA, Shapiro SA, Lundy LB, Leming DT (2006) Simultaneous acute superior nerve neurolabyrinthitis and benign paroxysmal positional vertigo. J Am Acad Audiol 17:481–486
26. Bagger-Sjöbäck D, Perols O, Bergenius J (1993) Audiovestibular findings in patients with vestibular neuritis: a long-term follow-up study. Acta Otolaryngol Suppl 503:16–17
27. Korres S, Balatsouras DG, Ferekidis E (2006) Prognosis of patients with benign paroxysmal positional vertigo treated with repositioning manoeuvres. J Laryngol Otol 120:528–533
28. Boniver R (2008) Benign paroxysmal positional vertigo: an overview. Int Tinnitus J 14:159–167