Otology & Neurotology
29:976–981 © 2008, Otology & Neurotology, Inc.

Daily Exercise Does Not Prevent Recurrence of Benign Paroxysmal Positional Vertigo

*Janet Odry Helminski, †Imke Janssen, and ‡§||Timothy Carl Hain

**Department of Physical Therapy, Midwestern University, Downers Grove; †Department of Preventive Medicine, Rush University Medical Center; Departments of ‡Physical Therapy and Human Movement Performance, §Otolaryngology, and ||Neurology, Northwestern University Medical School, Chicago, Illinois, U.S.A.*

Objective: The purpose of this study was to analyze if a daily routine of self-canalith repositioning procedure (CRP) will increase the time to recurrence and reduce the rate of recurrence of benign paroxysmal positional vertigo (BPPV).
Study Design: Prospective study, nonrandomized control group.
Setting: Outpatient clinic.
Patients: Thirty-nine patients diagnosed with posterior canal BPPV successfully treated with the CRP. Based on a convenience sample, 17 (44%) patients were assigned to the treatment group, whereas 22 (56%) were assigned to the no-treatment group. The number of subjects lost at the time of follow-up were 5 (29.4%) of the treatment group and 2 (9%) of the no-treatment group.
Interventions: Patients assigned to the treatment group performed the self-CRP daily, whereas those assigned to the no-treatment group performed no exercises. Patients were followed for up to 2 years.
Main Outcome Measures: The main outcome measures were the rate of recurrence of BPPV and the time for BPPV to recur.
Results: Of the 39 subjects, symptoms recurred in 16 (41%) of the total population, 6 (35%) of 17 of the treatment group, and 10 (46%) of 22 of the no-treatment group. There was no difference in the frequency of recurrence (Pearson χ^2; $p = 0.522$) or the time to recurrence (survival analysis; log-rank test; $p = 0.242$).
Conclusion: Our results suggest that a daily routine of the self-CRP does not affect the time to recurrence and the rate of recurrence of posterior canal–BPPV. **Key Words:** Benign paroxysmal positional vertigo—Vertigo.
Otol Neurotol **29:**976–981, 2008.

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo. Benign paroxysmal positional vertigo accounts for 26% of all vertigo (1). Most BPPVs are idiopathic (50%) (2). The most common identifiable cause of BPPV is head injury (17%) and viral neurolabyrinthitis (15%) (2). Benign paroxysmal positional vertigo is a major problem of the elderly. Nine percent of geriatric patients in an urban clinic had BPPV (3). The incidence of BPPV increases with each decade of life (4), peaking at the sixth to seventh decade of life (1). Benign paroxysmal positional vertigo affects the quality of life of geriatric patients and is associated with reduced activities of daily living, falls, and depression (3).

Benign paroxysmal positional vertigo is a mechanical problem of the inner ear caused by abnormal stimulation of a semicircular canal. The fluid-filled inner ear (Fig. 1) is composed of 3 continuous sections: the cochlea that senses hearing, the vestibule that detects gravity, and the semicircular canals that sense head rotation. The vestibule contains 2 areas of sensory epithelium, the maculae, composed of the gravity sensing hair cells and supporting cells. The hair cells are embedded within a gelatinous layer, the otolithic membrane, which contains calcium carbonate crystals (otoconia) implanted on top. The vestibule connects to the 3 semicircular canals (posterior canal [PC], horizontal canal, and anterior canal). Each canal contains a dilated area, the ampulla. The ampulla contains the sensory epithelium (cristae ampullaris) composed of hair cells embedded within a gelatinous diaphragm, the cupula. Most BPPVs are thought to be caused by otoconia detaching from the otolithic membrane and moving to the lowest point of the inner ear, usually the PC. This is referred to as canalithiasis (5).

The diagnosis of BPPV is based on the patient's history and the findings on positional testing. If BPPV involves the PC, vertigo occurs when the patient gets in and out of bed, rolls in bed toward 1 side, bends over

Address correspondence and reprint requests to Janet Odry Helminski, Ph.D., Midwestern University, Department of Physical Therapy, 555 31st Street, Downers Grove, IL 60515; E-mail: jhelmi@midwestern.edu

Supported by Grant 1R15AG17567-01A1 from the National Institute of Aging, Bethesda, MD.

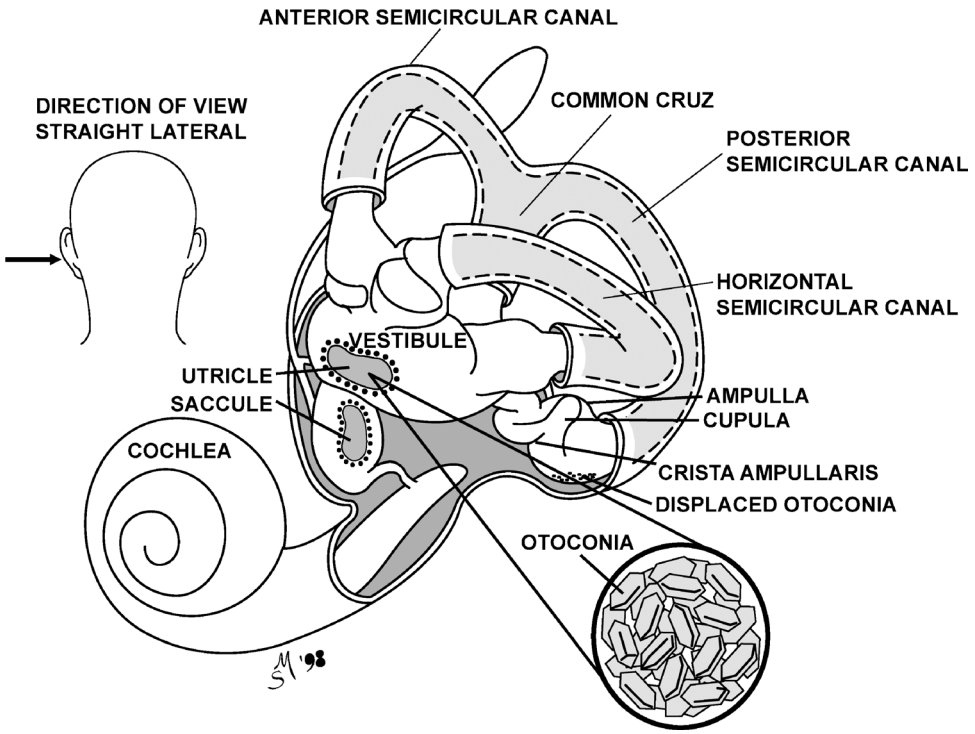

FIG. 1. Mechanisms of BPPV. Reprinted with permission from *American Dizziness and Balance* (2007).

and straightens up, or looks up (top-shelf syndrome) (6,7). Positional testing is used to identify the canal involved. It consists of the Dix-Hallpike maneuver (8), the side-lying test (9), and the roll test (10). If the patient has PC-BPPV, during the Dix-Hallpike maneuver (Fig. 2), when the patient is positioned with the plane of the PC of the lowermost ear perpendicular to the axis of gravity, the debris moves away from the cupula to the lowest part of the canal, generating a burst of torsion (toward the lowermost ear) and upbeating nystagmus. When the patient sits up, the debris moves toward the cupula, generating a burst of nystagmus with reversed direction. If the patient has anterior canal–BPPV, the head-hanging position generates a burst of downbeating nystagmus. If the patient had horizontal canal–BPPV, there is a burst of horizontal nystagmus.

Once the canal involved is identified, the patient may be treated effectively with an appropriate particle repositioning procedure. One such exercise, the canalith repositioning procedure (CRP) developed by Epley (11), was designed to use gravity to treat PC-BPPV. The patient is moved through a series of 4 positions. With each position, the otoconia moves toward the lowest part of the canal, resulting in the movement of the otoconia around the arc of the long arm of the PC into the common crus and depositing the otoconia into the insensitive vestibule. In randomized controlled trials, the short-term success rate of the CRP is 79 ± 16% (12–18). The CRP was modified to enable the patient to self-treat (19) (Fig. 3). The patient performs the CRP with the head extended over the edge of a pillow. In prospective, nonrandomized

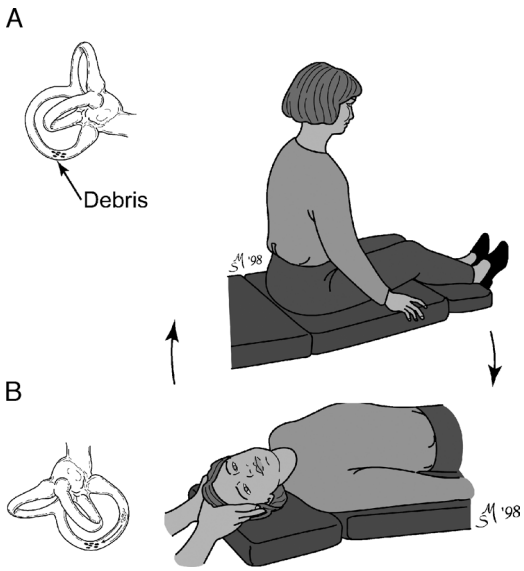

FIG. 2. *A*, Dix-Hallpike maneuver illustrated for the head right position without Frenzel goggles. The patient is positioned in long sitting (sitting on the treatment table with the legs extended). The patient's head is rotated 45 degrees toward the right. The patient is then lowered into supine, with the neck extended 20 degrees over the edge of the treatment table. The position is maintained for 45 seconds. The procedure is then repeated toward the left. For each position, the clinician notes the direction and characteristics of the nystagmus. *B*, In the head right position, if BPPV involves the right PC, a torsional toward the lowermost ear and upbeating nystagmus is observed. Reprinted with permission from *American Dizziness and Balance* (2007).

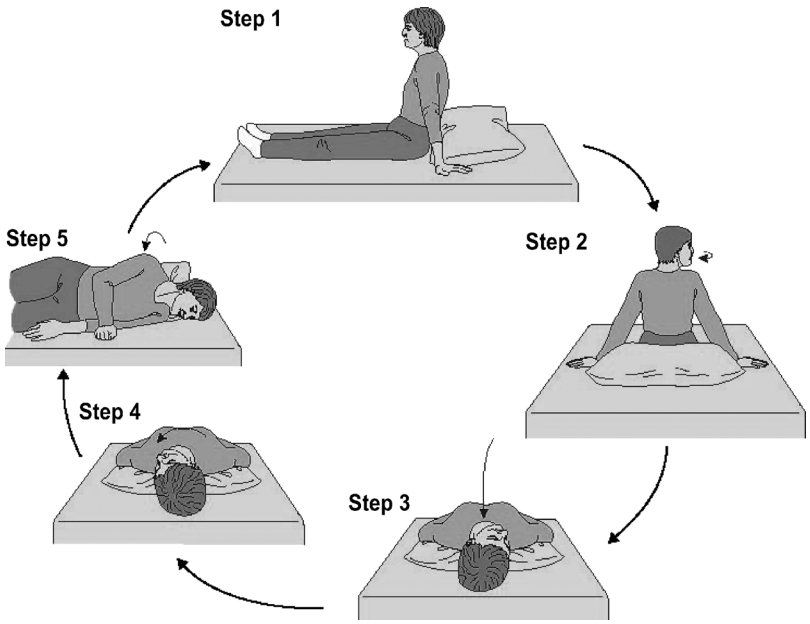

FIG. 3. Self-canalith repositioning procedure illustrated for treatment of the right PC. The patient moves through a series of 4 positions, starting with the placement of the involved canal in the head-hanging position of the Dix-Hallpike maneuver. The head is extended over the edge of the pillow instead of the edge of the bed. Then, the patient rotates the head 90 degrees toward the uninvolved side, followed by rolling onto the uninvolved side maintaining the head on trunk position, and finally sitting up from lying on the side. Each position is maintained for a minimum of 30 seconds or as long as the nystagmus lasts. Reprinted with permission from *American Dizziness and Balance* (2007).

trials, the average success rate of the self-CRP was 93 ± 4% (17,20) after 1 week.

Once successfully treated, BPPV often recurs. Of patients with PC-BPPV successfully treated with the CRP, 44% redevelop BPPV within the first 2 years (21). We previously questioned if a daily routine of a particle-repositioning maneuver would reduce the rate of recurrence of BPPV. We found that a daily routine of Brandt-Daroff exercises (22) did not affect the time to recurrence or the rate of recurrence of PC-BPPV (23). We suggested that the rate of recurrence of BPPV was not reduced because the Brandt-Daroff exercises were minimally effective in the treatment of BPPV within 1 week, the success rate being 24% (15). Recent studies have shown that the self-CRP is more effective than the Brandt-Daroff exercises in the treatment of BPPV within 1 week (17,20). Therefore, we hypothesized that the self-CRP may be effective in preventing recurrence of BPPV. The purpose of this study was to analyze if a daily routine of the self-CRP significantly reduces the rate of recurrence of BPPV and increases the time for BPPV to recur.

MATERIALS AND METHODS

Subjects diagnosed with PC-BPPV and treated successfully with the CRP were recruited from the practices of J.O.H. and T.C.H.. This study was approved by Midwestern University's institutional review board.

A neurootologic examination was performed. The diagnosis of PC-BPPV was based on history and findings on the Dix-Hallpike maneuver (8). The response of the patient to positional testing was monitored with video-oculography to prevent the patient from using visual fixation to suppress the nystagmus. Three criteria were required for diagnosis: 1) a 1- to 20-second latency before the onset of vertigo and nystagmus, 2) observation of a rotatory (toward the lowermost ear) and/or upward-directed nystagmus in the head-hanging position, and 3) vertigo and nystagmus of less than 60 seconds in duration. The patients quantified their symptom intensity on a scale of 1 to 3 (mild, moderate, and severe). Positional testing was performed before the initial treatment procedure and at the time of each follow-up.

If diagnosed with PC-BPPV, patients were treated with the CRP without vibration as previously described (11,21). After the maneuver, patients remained seated in the office for 20 minutes. To prevent otoconia from moving back into the semicircular canals, patients were placed on activity restrictions postmaneuver to avoid symptom-provoking positions. Patients were instructed to sleep upright or at a 45-degree angle for 2 nights and to avoid lying on the involved side and refrain from vertical and rapid head movements for 1 week.

One week after treatment, the patient's response to the Dix-Hallpike maneuver was evaluated in the clinic. Results, defined as change, were categorized on a scale of 1 to 4 (remission, much better, better, and no change) based on clinical examination. If symptoms resolved or were much better, patients were asked to participate in the study, and consent was obtained. If patients were unable to perform the exercises because of physical limitations or lack of motivation, patients were assigned to the no-treatment group, Group 1. If patients agreed to perform the self-CRP daily, the patients were assigned to the treatment group, Group 2. Subjects in Group 2 were trained in and instructed to perform 2 cycles of the self-CRP (19) (Fig. 3) 1 time

TABLE 1. *Characteristics of subjects with benign paroxysmal positional vertigo*

Characteristic	No treatment (n = 22)	Treatment (n = 17)	Total population (n = 39)	Statistical test	*p*
Age, mean ± SD (median), yr	61 ± 14 (64)	58 ± 16 (55)	59 ± 15 (63)	*t*	0.571
Male/female, n	7/15	4/13	11/28	Pearson χ^2	0.568
Duration, mean ± SD (median), mo	3 ± 6 (1)	3 ± 3 (3)	3 ± 5 (2)	Kruskal-Wallis	0.535
Intensity, n (%)				Pearson χ^2	0.089
Unknown	1 (5)	0 (0)	1 (3)		
Mild	10 (45)	2 (12)	12 (31)		
Moderate	9 (41)	13 (76)	22 (56)		
Disabling	2 (9)	2 (12)	4 (10)		
History of recurrence, n (%)				Pearson χ^2	0.458
No	9 (41)	5 (29)	14 (36)		
Yes	13 (59)	12 (71)	25 (64)		
Response after treatment, n (%)				Pearson χ^2	0.373
Remission	21 (95)	17 (100)	38 (97)		
Much better	1 (5)	0	1 (3)		
Better	0	0	0		
No change	0	0	0		

SD indicates standard deviation.

per day and were provided an illustrated handout and digital video disc of the exercises.

Patients were excluded from the study if the diagnosis of bilateral PC-BPPV or atypical BPPV was established on positional testing; if central nervous system involvement was identified based on history, magnetic resonance imaging, or neurologic examination; or if an alternative maneuver was performed such as the Semont maneuver (24) or Brandt-Daroff exercises (22).

Subjects in both groups were instructed to notify an investigator within 24 hours if dizziness recurred. Every 2 months, subjects were mailed a questionnaire asking if dizziness recurred and, in the treatment group, if they were still doing their exercises. If dizziness recurred, within 1 to 2 weeks, subjects were evaluated in the clinic with positional testing. During testing, eye movements were recorded with videooculography. If symptomatic, patients quantified the intensity on a scale of 1 to 3 (mild, moderate, and severe). If BPPV did not recur, subjects were evaluated in the clinic at the end of 2 years or at the end of the study.

Statistical analysis of the data was performed using Systat (version 10.2). To analyze if the 2 groups were similar, the demographic characteristics of the groups were compared. To analyze whether the time to recurrence differed between the treatment and no-treatment groups and to account for patients entering the study at different times, the Kaplan-Meier product-limit method was used to estimate the survival function. Survival time was defined as the number of days from the day of the last treatment session to the day the symptoms of BPPV recurred. For patients without a recurrence of BPPV, times were censored at the last day of the follow-up. Level of significance for all analyses was $p < 0.05$.

RESULTS

We identified 39 patients with PC-BPPV treated between December 2003 and December 2005. Of the 39 patients, 17 (44%) were assigned to the treatment group, and 22 (56%) were assigned to the no-treatment group. The number of subjects lost at the time of follow-up were 5 (29.4%) of the treatment group and 2 (9%) of the no-treatment group. The difference between the 2 groups was not significant (Pearson χ^2; $p = 0.101$). The Table 1 summarizes the demographic characteristics of the no-treatment and treatment groups. There were no significant differences in age, sex, duration of symptoms before treatment, or history of recurrent BPPV. There was no significant difference in response to treatment. The number of subjects with idiopathic cause was 20 (91%) of the no-treatment group and 14 (82%) of the treatment group. The known etiologies included posttraumatic, viral neurolabyrinthitis, and Ménière's disease.

Of the 39 subjects, symptoms recurred in 16 (41%) of the total population, 10 (43%) of the no-treatment group, and 6 (35%) of the treatment group. The difference in frequency of recurrence between the 2 groups was not significant (Pearson χ^2; $p = 0.522$). The estimated survival function for the no-treatment and treatment groups was plotted (Fig. 4). The log-rank test revealed that there was no significant difference in the time to recurrence between the 2 groups ($p = 0.242$).

Of the 16 subjects where symptoms had recurred, 6 (38%; 3 from each group) initiated self-treatment before evaluation in the clinic and treated themselves.

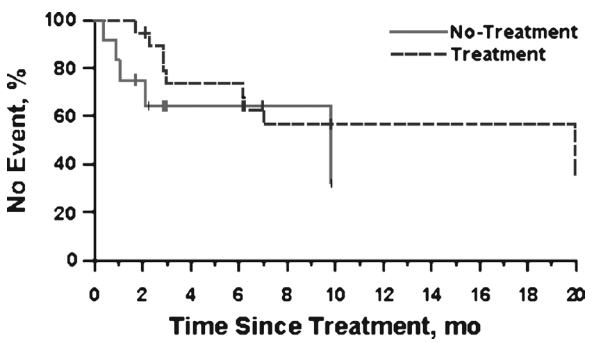

FIG. 4. Kaplan-Meier estimation of time to recurrence for the treatment and no-treatment groups. The log-rank statistic = 1.370, and $p = 0.242$. Reprinted with permission from *American Dizziness and Balance* (2007).

DISCUSSION

Our results suggest that a daily routine of the self-CRP does not reduce recurrence of PC-BPPV. There was no significant difference in the rate of recurrence or the time to recurrence of PC-BPPV between the treatment and no-treatment groups (Fig. 4; $p < 0.05$). These results are similar to those of Helminski et al. (23), that a daily routine of Brandt-Daroff exercises does not affect recurrence of PC-BPPV. The authors suggested that a daily routine of Brandt-Daroff exercise did not prevent recurrence of BPPV because the exercises were not effective. In prospective trials, after 1 week of exercises, the success rate of the Brandt-Daroff exercises was 24% (16), whereas the average success rate of the self-CRP was 93 ± 4% (17,20). However, even with a daily routine of the more effective self-CRP, exercises do not affect recurrence of BPPV.

The absence of a significant difference between the treatment and no-treatment groups might be attributed to an insufficient sample size—in particular, the self-CRP may have such a small effect that the effect is not detected with our sample size. Although this may be the case, the study's recurrence rate is similar to that reported in association with previous studies. The 2-year symptom recurrence rate in this study is 41%, whereas in previous studies, the rates were 43% (23) and 44% (21). This suggests that even if a larger sample size did document a significant effect of treatment, the effect of daily exercise on recurrence is so small as to be negligible.

Contacting subjects every 2 months may have underestimated the rate of recurrence. To improve reliability and validity, subjects may have been asked to maintain a dairy of symptoms and may have been contacted more frequently. However, this does not seem necessary. The 2-year symptom recurrence rate in this study and in previous studies (23,21) were the same.

The number of subjects lost to follow-up may have biased the results of our study. The lost-to-follow-up rate was significantly higher for the treatment group (29%) versus the no-treatment group (9%). If the data are analyzed assuming that symptoms recurred in all patients who dropped out of the study, there is still no significant difference between the treatment and the no-treatment groups. If the rate of recurrence is compared between the treatment group and the no-treatment group, for the total population (Pearson χ^2; $p = 0.522$) or for the total population excluding those lost to follow-up (Pearson χ^2; $p = 0.854$), there is no significant difference in rate of recurrence between the 2 groups. Therefore, it seems that the number of patients lost to follow-up did not affect our results. To avoid introducing bias into the statistical analysis and to provide information regarding the potential effects of the treatment policy, subjects lost to follow-up were included in the calculations.

Recurrence of BPPV may be caused by some other process that is not affected by exercise such as reactivation of a latent virus, a disorder that is vascular, autoimmune, or metabolic in origin, or positional such as prolonged bed rest. Current evidence suggests that 1 cause of vestibular neuritis is a latent infection of the vestibular ganglia by herpes simplex virus Type 1 (25). A stressful life event may result in reactivation of the latent virus infection, which could manifest as BPPV (25,26). Migraine headaches are 3 times more prevalent in patients with BPPV of unknown cause as compared with BPPV of known cause such as trauma or a surgical procedure (27). Autoimmune alterations (48.5%) and the level of antithyroid bodies in the blood (27.1%) are more common in patients with BPPV as compared with controls (28). In women whose ages range from 50 to 85 years, 75% of patients with BPPV have osteoporosis or osteopenia (29). However, a relationship between the incidence of BPPV and osteoporosis or osteopenia has not been made. A case reported the development of BPPV may follow prolonged bed rest (30). Therefore, several factors may contribute to recurrence of BPPV that may not be affected by exercise. Our study was not designed to identify these factors.

Daily particle-repositioning exercises may not prevent recurrence of BPPV. However, educating the patient in identifying the canal involved based on positional symptoms and self–particle-repositioning procedures such as the self-CRP may enhance the patient's control of the disease process and reduce health care costs by reducing the number of office treatment sessions. In this study, 38% of patients whose symptoms recurred initiated treatment, and 100% of these patients successfully resolved their symptoms by themselves.

In summary, a daily routine of the self-CRP does not affect the time to recurrence of BPPV or the rate of recurrence of BPPV. The cause of recurrent BPPV may be due to several factors not affected by exercise.

REFERENCES

1. Baloh RW, Sloane PD, Honrubia V. Quantitative vestibular function testing in elderly patients with dizziness. *Ear Nose Throat J* 1989;68:935–9.
2. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology* 1987;37:371–8.
3. Oghalai JS, Manolidis S, Barth JL, Stewart MG, Jenkins HA. Unrecognized benign paroxysmal positional vertigo in elderly patients. *Otolaryngol Head Neck Surg* 2000;122:630–4.
4. Froehling DA, Silverstein MD, Mohr DN, Beatty CW, Offord KP, Ballard DJ. Benign positional vertigo: incidence and prognosis in a population-based study in Olmsted County, Minnesota. *Mayo Clin Proc* 1991;66:596–601.
5. Hall SF, Ruby RR, McClure JA. The mechanics of benign paroxysmal vertigo. *J Otolaryngol* 1979;8:151–8.
6. Furman JM, Cass SP. Benign paroxysmal positional vertigo. *N Engl J Med* 1999;341:1590–6.
7. Whitney SL, Marchetti GF, Morris LO. Usefulness of the dizziness handicap inventory in the screening for benign paroxysmal positional vertigo. *Otol Neurotol* 2005;26:1027–33.
8. Dix MR, Hallpike CS. The pathology symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc R Soc Med* 1952;45:341–54.
9. Cohen HS. Side-lying as an alternative to the Dix-Hallpike test of the posterior canal. *Otol Neurotol* 2004;25:130–4.
10. Bisdorff AR, Debatisse D. Localizing signs in positional vertigo due to lateral canal cupulolithiasis. *Neurology* 2001;57:1085–8.

11. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1992;107:399–404.
12. Froehling DA, Bowen JM, Mohr DN, et al. The canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo: a randomized controlled trial. *Mayo Clin Proc* 2000;75:695–700.
13. Lynn S, Pool A, Rose D, Brey R, Suman V. Randomized trial of the canalith repositioning procedure. *Otolaryngol Head Neck Surg* 1995;113:712–20.
14. Sherman D, Massoud EA. Treatment outcomes of benign paroxysmal positional vertigo. *J Otolaryngol* 2001;30:295–9.
15. Soto Varela A, Bartual Magro J, Santos Perez S, et al. Benign paroxysmal vertigo: a comparative prospective study of the efficacy of Brandt and Daroff exercises, Semont and Epley maneuver. *Rev Laryngol Otol Rhinol (Bord)* 2001;122:179–83.
16. Sridhar S, Panda N, Raghunathan M. Efficacy of particle repositioning maneuver in BPPV: a prospective study. *Am J Otolaryngol* 2003;24:355–60.
17. Tanimoto H, Doi K, Katata K, Nibu KI. Self-treatment for benign paroxysmal positional vertigo of the posterior semicircular canal. *Neurology* 2005;65:1299–300.
18. von Brevern M, Seelig T, Radtke A, Tiel-Wilck K, Neuhauser H, Lempert T. Short-term efficacy of Epley's manoeuvre: a double-blind randomised trial. *J Neurol Neurosurg Psychiatry* 2006; 77:980–2.
19. Radtke A, Neuhauser H, von Brevern M, Lempert T. A modified Epley's procedure for self-treatment of benign paroxysmal positional vertigo. *Neurology* 1999;53:1358–60.
20. Radtke A, von Brevern M, Tiel-Wilck K, Mainz-Perchalla A, Neuhauser H, Lempert T. Self-treatment of benign paroxysmal positional vertigo: semont maneuver vs Epley procedure. *Neurology* 2004;63:150–2.
21. Hain TC, Helminski JO, Reis IL, Uddin MK. Vibration does not improve results of the canalith repositioning procedure. *Arch Otolaryngol Head Neck Surg* 2000;126:617–22.
22. Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. *Arch Otolaryngol* 1980;106:484–5.
23. Helminski JO, Janssen I, Kotaspouikis D, et al. Strategies to prevent recurrence of benign paroxysmal positional vertigo. *Arch Otolaryngol Head Neck Surg* 2005;131:344–8.
24. Semont A, Freyss G, Vitte E. Curing the BPPV with a liberatory maneuver. *Adv Otorhinolaryngol* 1988;42:290–3.
25. Arbusow V, Theil D, Strupp M, Mascolo A, Brandt T. HSV-1 not only in human vestibular ganglia but also in the vestibular labyrinth. *Audiol Neurootol* 2001;6:259–62.
26. Gacek RR. Pathology of benign paroxysmal positional vertigo revisited. *Ann Otol Rhinol Laryngol* 2003;112:574–82.
27. Ishiyama A, Jacobson KM, Baloh RW. Migraine and benign positional vertigo. *Ann Otol Rhinol Laryngol* 2000;109:377–80.
28. Modugno GC, Pirodda A, Ferri GG, Montana T, Rasciti L, Ceroni AR. A relationship between autoimmune thyroiditis and benign paroxysmal positional vertigo? *Med Hypotheses* 2000;54:614–5.
29. Vibert D, Kompis M, Hausler R. Benign paroxysmal positional vertigo in older women may be related to osteoporosis and osteopenia. *Ann Otol Rhinol Laryngol* 2003;112:885–9.
30. Gyo K. Benign paroxysmal positional vertigo as a complication of postoperative bedrest. *Laryngoscope* 1988;98:332–3.