

## VESTIBOLOGY

# Observational study on risk factors determining residual dizziness after successful benign paroxysmal positional vertigo treatment: the role of subclinical BPPV

## *Studio osservazionale sui fattori di rischio che causano residual dizziness dopo il trattamento della vertigine parossistica posizionale benigna: il ruolo della VPPB subclinica*

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## SUMMARY

After successful treatment for benign paroxysmal positional vertigo, many patients may complain of residual dizziness. Possible explanations may be the persistence of otolith into canal insufficient to provoke noticeable nystagmus, utricular dysfunction and undiagnosed coexisting vestibular disorder. We conducted a prospective observational case-control study, focusing on the role of risk factors in determining residual dizziness after BPPV treatment. In the present study, 148 patients were recruited and residual dizziness was documented in the 57.5% of the cohort. Among patients with residual dizziness 36 had subclinical BPPV and after retreatment, although nystagmus was not clinically evident, there was resolution of dizziness. We conclude that residual otoliths may play a role in determining post-maneuver residual dizziness that is often linked to subclinical BPPV; this conclusion is also supported by the high prevalence of BPPV recurrence in patients with residual dizziness, as confirmed by our analysis. The main cause appears to be linked with dispersed otolith in semicircular canals.

**KEY WORDS:** Residual dizziness • Benign paroxysmal positional vertigo • Subjective BPPV • Dizziness • Nystagmus

## RIASSUNTO

*Alcuni pazienti, dopo il trattamento della vertigine parossistica posizionale benigna concluso con successo, possono lamentare un disequilibrio residuo. La possibile spiegazione potrebbe essere: la persistenza di otoliti canalari insufficienti a provocare un nistagmo clinicamente evidente, una disfunzione utricolare, coesistenza di altri disordini del sistema vestibolare. Abbiamo condotto uno studio osservazionale prospettico caso-controllo, focalizzando l'attenzione sul ruolo di fattori di rischio che possono causare un disequilibrio residuo dopo il trattamento della VPPB. Abbiamo reclutato 148 pazienti e un disequilibrio residuo è stato documentato nel 57,5% dei casi. Tra i pazienti con disequilibrio residuo in 36 è stata diagnosticata una VPPB subclinica, che dopo il ritrattamento, sebbene senza evidenza clinica di nistagmo, hanno avuto una risoluzione dei sintomi. Possiamo concludere che gli otoliti residui possono avere un ruolo nel determinare il disequilibrio residuo post-manovra, poiché legato a una VPPB subclinica. Questa conclusione è testimoniata anche dall'alta prevalenza di recidive nei pazienti con disequilibrio residuo. La causa principale sembra legata alla persistenza di otoliti dispersi nei canali semicircolari.*

**PAROLE CHIAVE:** Disequilibrio residuo • Vertigine parossistica posizionale benigna • VPPB soggettiva • Disequilibrio • Nistagmo

## Introduction

Benign paroxysmal positional vertigo (BPPV) accounts for about 20% of vestibular complaints <sup>1</sup>. Being a mechanical disorder of the semicircular canals, management

consists of “mechanical” repositioning of the otoconial debris, also called otolith or canalith, detached from vestibular sensorineural epithelia. Posterior semicircular canal (PSC) is the most involved by BPPV with approxi-

mately 90% of cases, while horizontal semicircular canal (HSC) is the next most common <sup>2</sup>; superior semicircular canal (SSC) involvement is rare. The canalithiasis consists of dispersed fragments of otoliths into semicircular canals, which are able to cause vertigo when, by gravity, move into canals. The repositioning maneuvers to treat canalithiasis are well established and used widespread, with some variations recently reported in literature <sup>3</sup>.

In clinical practice, among patients admitted to emergency for vertigo, 8-9% are diagnosed with BPPV <sup>4</sup>. The treatment of BPPV is often simple and immediate, giving the patient a prompt resolution of the symptoms. Some patients with resistant BPPV require several maneuvers to reach adequate results, while other patients, after initial resolution of symptoms, show delayed positional nystagmus due to canal reentry of otoliths <sup>5</sup>.

Furthermore, patients without noticeable nystagmus during diagnostic assessment for BPPV, although experiencing vertigo and autonomic symptoms while the diagnostic test are performed, are diagnosed as subjective (subclinical) BPPV, since dispersed otolith are unable to give a clinical manifestation of nystagmus, and is manageable in the same manner of traditional BPPV <sup>6</sup>.

Despite successful BPPV treatments, many patients complain of residual dizziness (RD) that is described variously by patients and can be classified as non-vestibular dizziness, based on the characteristics of the disequilibrium and absence of nausea and vomiting <sup>7</sup>.

We conducted a prospective case-control study on BPPV treated by canalith repositioning maneuvers, focusing on the role of residual debris in determining subclinical BPPV as a cause of RD.

## Materials and methods

All consecutive patients admitted for BPPV to our ENT divisions in the period 2012-2014 were included in the study, according to the approval of the institutional review board. Recent vertigo other than BPPV, head trauma and lifetime history of previous episodes of vertigo other than BPPV were considered as exclusion criteria. Residual dizziness was expressed as sensation of unsteadiness or lightheadedness without rotational vertigo <sup>8</sup>. Data of included patients were prospectively collected in an electronic database. The following variables were recorded: age (< 40; 40-65; > 65), gender (male/female), side (left/right), tinnitus (yes/not), hearing loss (yes/not), previous BPPV episodes (yes/not), affected semicircular canal (PSC, LSC, SSC), recurrence (yes/not), liberatory nystagmus (yes/not), number of maneuvers done, success of maneuvers (yes/not) and residual dizziness (yes/not). All pa-

tients underwent otolaryngologic examination, pure tone audiometry, evaluation of nystagmus with infrared video-Frenzel lens, diagnostic test for positional nystagmus with Dix-Hallpike manoeuvre for PSC, supine roll-test for HSC, head-hanging manoeuvre for SSC, video Head Impulse Test (vHIT) done with Interacoustics Eysecam<sup>®</sup> and Vestibular Evoked Myogenic Potentials (VEMPs) with Hedera Biomedics Socrates<sup>®</sup>. Ocular and Cervical VEMPs were done by Air-conducted stimulus examining both ears separately by tone burst 130 dB at 500 Hz. Normative values considered in all patients for VEMPs were: latency values, inter-amplitude and inter-latency asymmetry between range 0-45%, and absent or not reproducible wave; for vHIT symmetric gain and absence of overt and/or covert saccades was considered as normal, the gain value was not considered because several issue about to assess normative <sup>9</sup>. The PSC diagnostic test was considered positive when nystagmus was appropriate with head position as torsional type with up-beating component. The HSC was considered positive when during supine head roll-test a direction changing horizontal nystagmus was detected, and for SSC when during head-hanging test a down-beating nystagmus with latency, crescendo and transience was observed with or without torsional component <sup>10</sup>. The treatment included the same manoeuvre in all patients related to canal involved to avoid bias due to type of manoeuvre: Gans manoeuvre for PSC BPPV <sup>3</sup>, Gufoni and Yacovino manoeuvre respectively for HSC and SSC involvement <sup>2,11</sup>. The success of treatment was defined as disappearance of both symptoms and nystagmus at diagnostic tests performed 45 minutes after treatment. The clinical features of BPPV were recorded: side involved, canal involved, number of manoeuvres done to treat the BPPV, presence of liberatory nystagmus, canal re-entry or canal switch <sup>5</sup> and recurrence of BPPV after successful treatment. Follow-up was done with clinical control at one and two weeks after treatment and with control visits at 6 months and 12 months even if symptoms were absent. The presence of dizziness even without nystagmus at clinical control was recorded, and the vertigo elicited during the diagnostic manoeuvre for BPPV without clinical evidence of nystagmus was considered as subclinical (subjective) BPPV <sup>6</sup>. The recurrence was defined as further BPPV episodes with noticeable nystagmus at otoneurologic examination in the follow-up period. All patients with persistence of untreatable dizziness underwent to imaging to exclude pathologies of the central nervous system. Distributions of continuous variables in different groups were analysed by T-student test parametric method. For categorical variables, comparisons were performed using Chi-square test with Yates correc-

tion and Fisher's exact Test. Univariate and multivariate logistic regression analysis were performed. Odds ratios and 95% confidence intervals were calculated. Statistical significance was set at  $p < 0.05$ . Data were analysed using the R statistical software package, version 2.2.0. Informed consent was obtained from all participants.

## Results

During the study period, 165 patients were treated for BPPV at our institutions, but 17 patients were lost during follow-up. We conducted our analysis on 148 patients, 92 (62.2%) females and 56 (37.8%) males, average age 53 (s.d. = 13.9) and median age 53, who were recruited according to the inclusion criteria. 63.5% of cases were in the 40-64 age category. No spontaneous nystagmus was recorded. Residual dizziness was documented in 57.5% of the sample. Most of the cases (76.4%) had PSC involvement at clinical examination. Tinnitus was present in 23 subjects (15.5%). The audiometric test revealed a sensorineural hearing loss (SNHL) in 65 patients: 22 mild SNHL and 43 severe SNHL. Imaging performed in 25 patients with persistence of dizziness after retreatment excluded a central nervous system pathology. VEMPs were present in all subjects

complaining of residual dizziness or recurrent BPPV; the mean P1/N1 latency for c-VEMPs was 15.9/23.6 msec (SD 1.9/2.4); no latency or amplitude asymmetry was recorded with interaural difference under 30% (SD 5%). vHIT was also normal (absence of covert and/or overt saccades) in all patients with no asymmetric gain value recorded.

At least one episode of recurrence was documented during follow-up in 18 patients (12.2%), which statistically differed between the comparison group by age, canal reentry and absence of liberatory nystagmus during the first session of treatment (Table I). In 65 patients, more than one manoeuvre was needed to obtain BPPV resolution.

The logistic regression model documented significant risk excess for recurrence of BPPV associated with age (OR = 1.063; C.I. = 1.014-1.12), while a significant high reduction associated with success of therapeutic manoeuvre (OR = 0.028; C.I. = 0.001-0.33).

In Table II, comparison between patients complaining residual dizziness (57.4%) and the ones without residual dizziness (42.6%) is shown. Female gender ( $p = 0.00$ ), advanced age ( $p = 0.00$ ), previous episodes of BPPV ( $p = 0.01$ ), more than one manoeuvre for treatment ( $p = 0.00$ ) and recurrence of BPPV ( $p = 0.00$ ) explained the statistical difference between the two groups.

**Table I.** Comparison between patients with and without recurrence. Age, canal reentry and presence of liberatory nystagmus were significant predictor of recurrence.

		No recurrence	Recurrence	P-value
		N 130 (%) 87.8%	N 18 (%) 12.2%	
Gender	Male	48 (85.7%)	8 (14.3%)	0.7
	Female	82 (89.1%)	10 (10.9%)	
Age	Average	52.7 S.d. (13,94)	67.3 s.d. (13,79)	0.006
Previous BPPV	None	60 (93.7%)	4 (6.3%)	0.1
	> 1	70 (83.3%)	14 (16.7%)	
Number of CRM	1	76 (91.6%)	7 (8.4%)	0.2
	> 1	54 (83.1%)	11 (16.9%)	
Canal reentry	No	126 (89.4%)	15 (10.6%)	0.04
	Yes	4 (57.1%)	3 (42.9%)	
Liberatory Ny	No	1 (25%)	3 (75%)	0.006
	Yes	129 (89.6%)	15 (10.4%)	

**Table II.** Comparison between patients with and without persistence of dizzy symptoms after treatment; numbers of manoeuvres and recurrence were predictive of persistent dizziness.

		Residual dizziness Yes	Residual dizziness No	P-value
		85 (%) 57.4%	63 (%) 42.6%	
Gender	Male	24 (42.9%)	32 (57.1%)	0.005
	Female	61 (66.3%)	31 (33.7%)	
Age	Average	57 S.d. (14,41)	47 s.d. (10,78)	0.000001
Previous BPPV	None	29 (45.3%)	35 (54.7%)	0.01
	More than one	56 (66.7%)	28 (33.3%)	
Numbers of manoeuvres	One	34 (41%)	49 (59%)	0.000004
	More than one	51 (78.5%)	14 (21.5%)	
Liberatory Ny	No	26 (68.4%)	12 (52.6%)	0,1
	Yes	59 (28.2%)	51 (71.8%)	
Canal reentry	No	80 (56.7%)	61 (43.7%)	0.7
	Yes	5 (71.4%)	2 (28.6%)	
Success of manoeuvres	No	4 (100%)	0 (0%)	0.1
	Yes	81 (56.3%)	63 (43.7%)	
Recurrence	No	68 (52.3%)	62 (47.7%)	0.0005
	Yes	17 (94.4%)	1 (5.6%)	

Among patients readmitted for residual dizziness following clinical vestibular examination, 36 were diagnosed with a subclinical BPPV, while only 2 patients had subclinical BPPV at follow-up in the group without RD ( $p = 0.00$ ). No recurrence was detected in patients with subclinical BPPV who underwent retreatment of the same canal.

## Discussion

Residual dizziness is a frequent complaint of patients after treatment for BPPV, even if therapeutic success was achieved, which might be present in two-thirds of cases. Four theories have been hypothesised to explain the RD: 1) remaining otoconial debris due to incomplete repositioning that can produce soft positional vertigo, because

the remaining debris are insufficient to deflect the cupula to a degree able to provoke overt nystagmus<sup>12,13</sup>; 2) BPPV is not only a disorder of the semicircular canals, but also of otolith organs that sense orientation in the space, and otolith dysfunction might account for transient mild dizziness<sup>14,15</sup>; 3) another vestibular lesion that is difficult to identify from history alone might coexist with BPPV, and the prevalence of less-specific dizziness was significantly higher in BPPV patients with additional peripheral or central vestibular dysfunction<sup>16</sup>; 4) delayed recovery might be due to the longer time needed for central adaptation after particle repositioning.

The English literature also reports that patients with residual dizziness have higher anxiety scores than patients with no residual dizziness<sup>8</sup>. Anxiety has been demonstrat-

ed to play a role in dizziness, and anxiety and dizziness are comorbid in a larger percentage of patients than would be expected from chance alone<sup>17,18</sup>.

The vestibular system participates in autonomic regulation adjusting cardiovascular control during body movement and change in posture<sup>19,20</sup>. Patients with BPPV occasionally experience postural light-headedness when righting from a sitting position, despite successful repositioning procedures<sup>21</sup>; it is similar to orthostatic dizziness reported by patients with orthostatic hypotension<sup>22</sup>.

One-third of patients with BPPV have some abnormality of autonomic system response as shown by orthostatic hypotension tilting test or blood pressure response during Valsalva manoeuvre; the rate of autonomic dysfunction is higher in patient with residual dizziness than in those without<sup>23</sup>.

Residual dizziness was found to be related to duration of vertigo before repositioning manoeuvre. A longer duration of BPPV was associated with the presence of residual dizziness after the particle repositioning maneuver<sup>7</sup>. In our observation, patients with more than one episode of BPPV in their history had a significantly increased risk to develop a RD, which is increased if the patient is more than 65 years of age. Our dataset showed that the elderly population has a generically high risk of BPPV recurrence, effectively confirming our previous results, where we described some risk factors (hypertension, diabetes, osteoporosis) that influence the high rate of recidivism in patients over 65 years<sup>24</sup>. The increased prevalence in the elderly population is considered to be caused by changes in otoconia morphology, possibly related to vascular damage in the inner ear<sup>25</sup>, although signs of inner ear aging such as tinnitus and hearing loss have showed no relationship with RD in our patients. Elderly patients affected by BPPV also complain of dizziness and unsteadiness instead of typical positional vertigo; this may be due to unconscious avoidance of positions provoking vertigo rather than decreased perception of vestibular stimuli related to otolith organ damage<sup>26</sup>. Furthermore, in the elderly we found a reduced success rate of repositioning manoeuvres that may be linked to that chronic vascular damage of the inner ear and modification of otoconia. This reduced success rate of treatment was parallel to a significantly increased rate of RD in those patients, which leads to the consideration that some dispersed otolith into semicircular canals may play the main role in RD.

The responsibility of dispersed fragments of otolith could also be hypothesised by our observation that patients who underwent more than one repositioning manoeuvre in the same session had an increased risk to have RD in the post-manoeuvre period, as shown by logistic regression where the success of the manoeuvre reduced the recurrence rate.

In a previous report, we described the linkage of canal re-entry BPPV risk with number of manoeuvres, thinking that the otolith may be dispersed into canals<sup>5</sup>. Although patients do not complain a true positional vertigo after treatment, they may have dizziness due to otolith fragmentation, the mass of which is not enough to elicit a true positional vertigo. Effectively, we noted a high percentage of subclinical BPPV in patients with RD, which after re-treatment, even if nystagmus was not clinically evident, had resolution of dizziness<sup>27,28</sup>. However, the RD may be linked not only with dispersed otolith (main cause of subclinical BPPV), but also with age, BPPV recurrence and absence of liberatory nystagmus that could predict the chance of RD as shown in our analysis. In our opinion, following the reported dataset, residual otoliths play a main role in determining post-manoeuvre RD that is often linked to subclinical BPPV; this conclusion is also supported by the high prevalence of BPPV recurrence in patients with RD, as confirmed in our analysis.

## Conclusions

RD may be a long lasting complaint in patients treated for BPPV. The pathophysiology may be related with several diseases and comorbidities. Our study focused on dispersed otolith into semicircular canals as a risk factor for RD. The high prevalence of subclinical BPPV among patients readmitted for RD is one of the possible explanations. Advanced age and recurrence of BPPV may be predictive of post-treatment RD. However, more than half of patients with RD remain without an explanation of the likely cause, and warranting further studies.

## Conflict of interest statement

None declared.

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