

ORIGINAL PAPER

# Paroxysmal Positional Vertigo: the role of age as a prognostic factor

## *Vertigine posizionale parossistica: il ruolo dell'età come fattore di prognosi della malattia*

M. FARALLI, G. RICCI, E. MOLINI, T. BRESSI, C. SIMONCELLI, A. FRENGUELLI  
Department of Medical-Surgical Specialisation, Otolaryngology and Cervico-Facial Surgery Division,  
University of Perugia, Italy

### Key words

Paroxysmal Positional Vertigo • Vascular vertigo • Prognosis • Age

### Parole chiave

*Vertigine posizionale parossistica • Vertigine vascolare • Prognosi • Età*

### Summary

Aim of this study was to examine possible relationships between several clinical aspects of paroxysmal positional vertigo and factors better defined as “intrinsic” to the patient, above all age. The disorder can affect essentially all age groups; nevertheless, the onset of age-linked degenerative processes, such as vascular damage, can have a negative influence – at least in theory – on the pathogenic mechanisms of cupulolithiasis or canalolithiasis. The study was based on the review of 566 patients with the typical form of paroxysmal positional vertigo. Based on age, the patients were divided into two groups, respectively  $\leq 50$  years and  $> 50$  years. For the purposes of this study, a series of clinical-laboratory conditions associated with the risk of, or clear, vascular damage were also considered. The results indicate that if there are no clinical or case-history elements that can be attributed to an aetiological hypothesis, the clinical behaviour of paroxysmal positional vertigo is not affected by the age factor. However, the existence of generic vascular damage, hypothesised by the presence of the above-mentioned conditions, influences certain clinical aspects of the disorder, particularly recovery time, the trend of the active phase and the number of relapses. In conclusion, paroxysmal positional vertigo with a presumed vascular aetiology, the incidence of which increases with age, presents a worse prognosis, not only with respect to the “idiopathic” form in childhood but also the “idiopathic” type in the elderly. The lithiasic model responds well to pathogenic interpretation requirements, which envisage macular degeneration with a vascular component. However, the observation, via imaging, of diffuse ischaemic lesions in critical areas of the brainstem and the cerebellum in many “vascular” patients, does not exclude the possibility of alternative pathogenic mechanisms that, in the final analysis, can lead to compromised VOR on a central level.

### Riassunto

*Scopo di questo lavoro è stato quello di ricercare eventuali rapporti esistenti tra alcuni aspetti clinici della VPP e fattori più propriamente “intrinseci” del paziente, primo fra tutti l'età. Sappiamo come la malattia possa interessare in pratica tutte le decadi della vita; tuttavia non c'è dubbio che il sopraggiungere con l'età di elementi degenerativi come il danno vascolare siano in grado di interferire, almeno in teoria, in maniera negativa sui meccanismi patogenetici della cupulo-canalolitiasi. Lo studio si è basato sulla revisione di un gruppo di 566 pazienti affetti da VPP in forma tipica. In base all'età i pazienti sono stati suddivisi in 2 gruppi rispettivamente di età  $\leq 50$  anni e  $> 50$  anni. Sono inoltre stati considerati ai fini dell'indagine una serie di condizioni clinico-laboratoristiche associate a rischio o danno vascolare conclamato. I risultati dimostrano come in assenza di elementi clinico-anamnesici in grado di ricondurre ad una qualsiasi ipotesi eziologica, il comportamento clinico di una VPP non risente del fattore età. L'esistenza tuttavia di un generico danno vascolare, ipotizzato dalla presenza delle condizioni precedentemente menzionate, condiziona alcuni aspetti clinici della malattia ed in particolare i tempi di guarigione, l'andamento della fase attiva ed il numero delle recidive. In conclusione, una VPP a presunta eziologia vascolare, la cui incidenza aumenta irrimediabilmente con l'età, presenta una prognosi peggiore rispetto non solo alla forma “idiopatica” giovanile ma anche nei confronti di quella “idiopatica” dell'anziano. Il modello litiasico risponde bene alle esigenze interpretative patogenetiche che prevedono una sofferenza maculare su base vascolare; tuttavia il riscontro mediante imaging di lesioni ischemiche diffuse in aree critiche del tronco-encefalo e del cervelletto, in molti pazienti “vascolari”, non esclude la possibilità di meccanismi patogenetici alternativi che comportino in ultima analisi una sofferenza di tipo centrale del VOR.*

## Introduction

Paroxysmal positional vertigo (PPV) is the most commonly observed form of labyrinthine vertigo and is undoubtedly the balance disorder that most bene-

fits from physical therapy. Despite numerous reports in the literature, the aetiology of this disorder is still uncertain, as none of the theories advanced so far can individually act as the prototype for all the forms of PPV. In fact, even if traumatic origins<sup>1-4</sup> are the eas-

iest to identify, the case studies of most patients do not indicate the presence of significant trauma that, from a temporal standpoint, is compatible with the onset of symptoms. Likewise, the vascular theory<sup>5-7</sup> that may be feasible in patients over the age of 50 years, with a significant clinical history, does not always appear to justify PPV, particularly in young subjects who present no risk factors. Furthermore, it is difficult to demonstrate a viral<sup>8</sup>, dysendocrine<sup>9</sup>, dysmetabolic<sup>9</sup>, deficiency-related<sup>10</sup>, or autoimmune<sup>11-14</sup> aetiology. These hypotheses are probably all valid, thereby allowing the possibility that various aetiological factors may play a role in determining the disorder. On the other hand, the pathogenesis of the disorder seems to be firmly established, above all in the typical forms of PPV, in which the cupulolithiasis or canalolithiasis model respond well to the interpretative requirements of nystagmus induced by positioning at diagnosis<sup>15-18</sup>. Regardless of the techniques used, PPV shows high recovery rates, both spontaneously and following treatment. Nevertheless, the observation, in clinical practice, of forms of PPV that are resistant to therapy or are characterised by an active phase with a recurrent or relapse phase, as well as the evidence – not unusual – of atypical oculomotor patterns, triggers a series of questions regarding differential diagnostics as well as pathogenic and therapeutic aspects. Aim of this study was to examine possible relationships between several clinical aspects of the disorder, such as recovery time (i.e., the period between the beginning of treatment and resolution of the objective picture), the trend of the active phase, and factors more appropriately defined as “intrinsic” to the patient, above all age. It is well known that the disorder can occur essentially at any age, including childhood, in which it also acquires predictive value for certain forms of headache in adulthood<sup>19</sup>. However, undoubtedly, with aging, degenerative elements such as vascular damage can negatively interfere – at least in theory – with the pathogenic mechanisms of cupulolithiasis or canalolithiasis.

## Materials and methods

Taking part in the study were 566 patients (204 male, 362 female; mean age 56.8 years) with a typical form of PPV, referred to our Unit between 1 January 2000 and 31 December 2002. All patients underwent a complete otoneurological evaluation, including audiometry, tympanometry, and vestibular examinations with thermal stimulation. In those cases in which asymmetrical mono- or bilateral hypoacusia or significant areflexia/hyporeflexia according to Jongkees' formula were observed, the diagnostic examination was completed with auditory evoked poten-

tials. Diagnostic imaging techniques (computed tomography (CT), magnetic resonance (MR)) were used only for patients with Auditory Brainstem Responses (ABR) alterations and those with negative diagnostic examinations who failed to respond to physical therapy.

After diagnosis of the nature and site of PPV, based on objective clinical elements (paroxysmal positional nystagmus), patients were included in a rehabilitation protocol that included Semont's liberatory manoeuvre – as modified by Toupet<sup>20-23</sup>, as well as Epley's repositioning manoeuvre<sup>24-27</sup> in cases of PPV of the vertical semicircular canals (VSC). Instead, for forms of PPV of the horizontal semicircular canal (HSC), Vanucchi's position<sup>28</sup>, Baloh's position<sup>29</sup> and Gufoni's position<sup>30</sup> were used.

Patients in the study population were classified into two groups, based on age:

- Group A – Patients aged  $\leq 50$  years;
- Group B – Patients aged  $> 50$  years.

The study also focused on an analysis of the clinical elements representing factors that could potentially favour and/or trigger onset of the disorder. For the purposes of our study, in particular, we considered the noxae that most commonly cause or are associated with vascular damage, the incidence of which increases significantly with age. These included hypertension undergoing drug treatment; hyperglycaemia being treated with oral hypoglycaemic agents or insulin; dyslipidaemia (hypercholesterolaemia, in particular), not necessarily being treated with drugs but with total cholesterol values exceeding 250 mg/dl; vascular brain diseases documented via imaging and positive clinical neurological findings (stroke, Transient Ischaemic Attack); previous acute or chronic ischaemic heart disease. Based on the presence of these factors, we considered two types of patients:

- Vascular type – patients with two or more factors associated with the risk of or clear vascular damage;
- Non-vascular type – patients without or with only one factor associated with the risk of, or clear, vascular damage.

Considering the same factors in relation to age (Table I), we subdivided the patients as follows:

- Group A Vascular type – patients aged  $\leq 50$  years with two or more factors associated with the risk of, or clear, vascular damage;
- Group A Non-vascular type – patients aged  $\leq 50$  years without or with only one factor associated with the risk of or clear vascular damage;
- Group B Vascular type – patients aged  $> 50$  years with two or more factors associated with the risk of, or clear, vascular damage;
- Group B Non-vascular type – patients aged  $> 50$  years without or with only one factor associated with the risk of, or clear, vascular damage.

**Table I.** Subdivision of patients according to age and typology.

Group A vascular type 6 patients	age $\leq$ 50 years with 2 or more factors associated with risk or clear vascular damage
Group A non-vascular type 158 patients	age $\leq$ 50 years without or with only 1 factor associated with risk or clear vascular damage
Group B vascular type 105 patients	age $>$ 50 years with 2 or more factors associated with risk or clear vascular damage
Group B non-vascular type 291 patients	age $>$ 50 years without or with only 1 factor associated with risk or clear vascular damage

Recovery time was evaluated and expressed as the mean number of manoeuvres required to negativise the clinical picture; data obtained from the patients in the two groups under examination were also compared. Moreover, the number of relapses was evaluated starting with the second month after negativisation of the clinical picture.

The study also involved quantitative and qualitative analysis of the forms of PPV with an active phase characterised by immediate negativisation of the objective picture with the first rehabilitative manoeuvre and return to positivity at the next follow-up, scheduled 2-3 days after the previous session. The cases in which the clinical behaviour was repeated for at least three successive sessions were defined as Type 1M (manoeuvre) x 3S (sessions) PPV (Table II).

## Results

Of the 566 patients observed, six were excluded from our analysis as they never achieved negativisation, despite all the treatment measures undertaken. In one case, we observed a severe neurological pathology (32-year-old patient with astrocytoma of the brainstem and cerebellar compression); in the other five cases, the patients presented at least two of the vas-

cular factors considered and they belonged to the age range of  $>$  50 years. Instead, 560 patients completed the rehabilitation protocol and were thus considered cured (98.2%).

There were 164 patients in Group A and 396 in Group B. Of the pathological conditions considered, arterial hypertension was the most frequent, being observed in 191 patients; hyperglycaemia – under treatment – was found in 46 cases, whereas 62 patients had hypercholesterolaemia. Ischaemic heart disease was observed in 39 patients, whereas 21 had vascular cerebral diseases. Based on the type previously described, 111 patients were defined as the vascular type; in this group, 90 had 2 of the vascular factors under consideration, whereas 3 or more of these factors were documented in 21 patients. There were 449 non-vascular type patients.

A total of 6 patients belonged to Group A vascular type, 158 patients belonged to Group A non-vascular type, 105 patients to Group B vascular type, and 291 patients to Group B non-vascular type (Table I).

On average, 2.78 ( $\pm$  0.31) manoeuvres were required to negativise the clinical picture. The mean for patients in Group A was 2.40 ( $\pm$  0.72), whereas the value recorded for Group B was 2.96 ( $\pm$  1.02). The comparison between the two groups (Group A vs. Group B) shows a statistically significant increase ( $p =$

**Table II.** Clinical behaviour of Type 1M x 3S PPV.

	Control	1st manoeuvre	Control	2nd manoeuvre
1st Session	+	performed	-	not performed
2nd Session	+	performed	-	not performed
3rd Session	+	performed	-	not performed

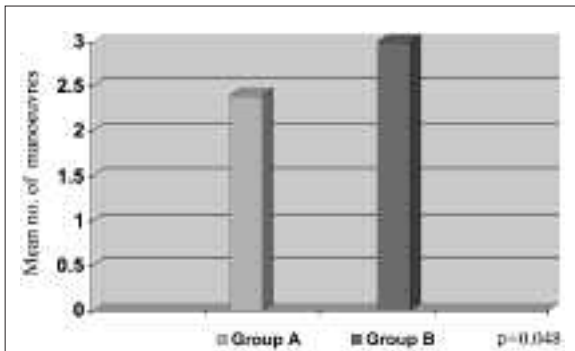


Fig. 1. Comparison between recovery times of groups examined.

0.048) in patients > 50 years (Group B) (Fig. 1). As far as concerns this group, the vascular type patients, with a mean of  $3.77 (\pm 0.24)$ , showed a highly significant increase ( $p = 0.000$ ) in the mean number of manoeuvres used to solve the clinical picture with respect to the non-vascular type patients who, inversely, had a mean value of  $2.50 (\pm 0.16)$  (Fig. 2). There were 77 relapses, with onset 2 months after recovery, for an incidence of 13.7% (77/560). Overall, the patients in Group A presented 13 relapses, 12 in non-vascular type patients and one in the vascular type. The patients from Group B had a total of 64 relapses; in 43 cases they were from the non-vascular type group, and in 21 from the vascular type. The relapse rate was 8% (13/164) in Group A vs. 16.3% (64/396) in Group B. With regard to Group A, the non-vascular type patients showed an incidence of 7.7% (12/158), whereas in the vascular type patients, the incidence was 16.6% (1/6). In Group B, the incidence was 14.9% (43/291) in non-vascular type patients and 20.1% (21/105) in vascular type patients (Table III). We observed recurrent Type 1M x 3S PPV in 54 patients; 44 (81.4%) belonging to Group B and only 10 (18.5%) to Group A, they all were of the non-vascular type. Of the 44 patients from Group B, 16 were of

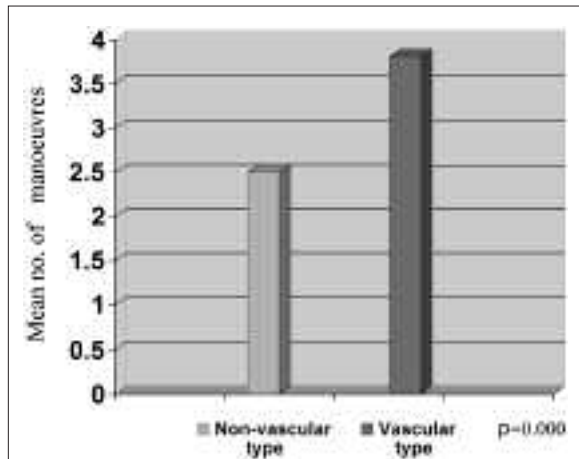


Fig. 2. Comparison between recovery times in relation to type examined.

the non-vascular type and 28 of the vascular type. To summarise, the overall incidence of Type 1M x 3S PPV was 9.6% (54/560); in Groups A and B, it was 6.2% (10/164) and 11.2% (44/396), respectively. In Group B, we observed a significant increase in Type 1M x 3S PPV in the vascular type patients, who presented an incidence of 26% (28/105), as opposed to 5.6% (16/291) recorded in the non-vascular type patients (Table IV).

## Discussion

The patient's age, in itself, does not represent a negative factor for the prognosis of the disorder. When there are no clinical/case-history elements that can lead to any type of aetiological hypothesis, "idiopathic" PPV, in the elderly, does not differ significantly from "idiopathic" PPV in childhood, in terms of clinical behaviour. In other words, no primary process of physiological involution of the resumed

Table III. Incidence of relapses.

	No. relapses	Incidence %
Total no. patients	77	13.7 (77/560)
Group A	13	8 (13/164)
Group B	64	16.3 (64/396)
Group A non-vascular type	12	7.7 (12/158)
Group A vascular type	1	16.6 (1/6)
Group B non-vascular type	43	14.9 (43/291)
Group B vascular type	21	20.1 (21/105)

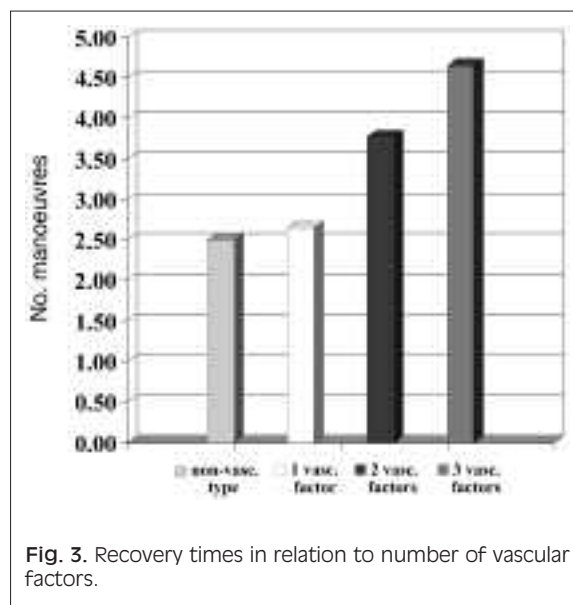
**Table IV.** Incidence of Type 1M x 3S PPV.

	PPV type 1M x 3S	Incidence %
Total no. patients	54	9.6 (54/560)
Group A	10	6.2 (10/164)
Group B	44	11.2 (44/396)
Group A non-vascular type	10	6.2 (10/164)
Group A vascular type	0	0 (0/6)
Group B non-vascular type	16	5.6 (16/291)
Group B vascular type	28	26 (28/105)

mechanism emerges that envisages a delicate balance between the production of otoconia and their destruction by the dark cells that surround the maculae of the otolithic organs, the alteration of which would definitively lead to the accumulation of intracanal masses.

Certain clinical aspects of the disorder, considered in our studies, are, nevertheless, affected by the presence of elements that can potentially cause vestibular damage: these, without any doubt, include vascular factors, the incidence of which increases progressively with age.

Of the 6 patients not responding to the different therapeutic measures undertaken, 5 were from Group B, all of whom presented at least 2 vascular risk factors. Moreover, the mean number of manoeuvres required to negativise the clinical picture showed a highly significant increase in the group of patients defined as “vascular”. In short, the presence of the factors described above seems to affect recovery time and, in some cases, actual recovery itself. This is confirmed by the clinical behaviour observed in non-vascular type patients, as no significant differences emerged between Groups A and B with regard to the parameters being examined. Moreover, what does emerge from the study is a close correlation between the extent of the damage theorised, based on the number of vascular factors noted and the clinical aspects of the disorders examined. In particular, the presence of only one risk factor did not lead to significant changes in recovery time with respect to vascular patients. Inversely, the mean number of manoeuvres required to negativise the clinical picture increased significantly and progressively as the number of vascular factors increased (Fig. 3). Therefore, it would appear that several of the factors under examination must be combined in order to trigger – at anterior vestibular artery level – vascular stress that can cause macular damage severe enough to influence recovery time, through the detachment of larger otoconial masses that thus require a larger number of manoeuvres to remove them. Another significant element lies in the fact that, among the vascular patients, we observed a higher

**Fig. 3.** Recovery times in relation to number of vascular factors.

incidence of the type of PPV that we defined as 1M x 3S, based on the clinical behaviour described above. In this case, the active phase of the disorder seems to be affected by persistent macular degeneration, leading to the slow and continuous – albeit not necessarily massive – detachment of otoliths. This leads to the build-up, in the canal, of otoconial masses that can easily be removed with liberating manoeuvres, but they rapidly reform due to continuous macular degeneration. Also in this case, the decisive role of the vascular factor emerges by examining non-significant differential data obtained by comparing the two groups of non-vascular patients.

In agreement with various Authors, our experience also confirms that PPV is a recurrent disorder, despite the difficulties in quantifying clinical data. These difficulties are due primarily to the behaviour of the patient, who often fails to attend follow-up visits, above all in cases of transient recurrence followed by the rapid resolution of symptoms. Even



though the clinical data are less significant with respect to the parameters considered above, the study shows that the presence of vascular factors appears to influence an increase in the incidence of relapses of PPV, and its negative effects are inevitably felt as the patient grows older.

## Conclusions

To summarize, as age advances, there is a higher rate of paroxysmal positional vertigo as well as worse prognosis, but this is strictly due to the fact that advanced age is also associated with a higher incidence of vascular risk factors.

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■ Received: February 18, 2005  
Accepted: December 20, 2005

■ Address for correspondence: Dr. G. Ricci, Clinica Otorinolaringoiatrica, via Enrico Dal Pozzo, 06126 Perugia, Italy. Fax +39 075 5726886