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THE UNIVERSITY OF SOUTH FLORIDA  
COLLEGE OF ARTS AND SCIENCES

**INCIDENCE OF PERIPHERAL VESTIBULOPATHY IN BPPV PATIENTS  
WITH AND WITHOUT PRIOR OTOLOGIC HISTORY**

BY

Allison Hulslander

An Audiology Doctoral Project

Submitted to the Graduate Faculty of the  
Department of Communication Sciences and Disorders  
in partial fulfillment of the requirements  
for the degree of

Doctor of Audiology

Richard A. Roberts, Chair  
Richard E. Gans, Co-Chair  
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## **ABSTRACT**

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The incidence data provided by previous investigations of peripheral vestibulopathy in patients with benign paroxysmal positional vertigo (BPPV) are quite variable. This variability may be explained, in part, by the otologic history of the patients included in these studies. Specifically, patients with a prior history of other otologic disease and BPPV should be more likely to present with peripheral vestibulopathy than patients without no prior history of otologic disease and BPPV. The purpose of this study was to determine the incidence of peripheral vestibulopathy in these two groups of BPPV patients. Caloric responses were analyzed for two groups of patients with posterior canal BPPV, those with a positive history of otologic disease and those with a negative history of otologic disease. Data were analyzed retrospectively for 157 BPPV patients. Patients with a positive history of otologic disease exhibited a greater incidence of peripheral vestibulopathy than the negative history group. The positive history group, on average, also exhibited a larger unilateral weakness than those patients in the negative history group. We conclude that patients with BPPV and a prior history of otologic disease are more likely to present with peripheral vestibulopathy than patients with BPPV and no history of otologic disease

## **INTRODUCTION**

According to the National Institutes of Health, complaints of dizziness and vertigo account for nearly 7 million physician visits a year (National Institute on Deafness and other Communication Disorders, 1995). Vestibular disorders account for approximately 85% of these visits (National Institute of Neurological and Communicative Disorders and Stroke, 1986). Vestibular disorders are typically characterized by vertigo, which is described as a perception of motion even though the patient may not be moving. Patients with vertigo often perceive they are spinning or that the environment is spinning around them.

The pathologies underlying vertigo may be divided into two broad categories: pathologies of peripheral etiology and pathologies of central etiology. Many diseases may affect the peripheral vestibular system, including Meniere's disease, vestibular neuritis/labyrinthine ischemia, acoustic neuroma/vestibular schwannoma, benign paroxysmal positional vertigo (BPPV), perilymphatic fistula, trauma, infection, and autoimmune inner ear disease. A unilateral lesion in the vestibular system is thought to cause the sensation of vertigo present in these peripheral disorders/diseases (Brandt and Daroff, 1980). Central causes of vertigo are due to infarctions, demyelinating disease, degenerative disease, or vascular disorders (Parker and Weiss, 1976; Wennmo and Hindfelt, 1980). Specifically, central disorders known to cause vertigo include vascular loops, vertebrobasilar insufficiency, multiple sclerosis, brainstem disease, migraine, and cervical vertigo (Applebaum and Valvassori, 1984; Kayan and Hood, 1984; Grenman, 1988; Grad and Baloh, 1989).

BPPV is the most common form of vertigo (Bath et al, 2000). BPPV is thought to arise from interaction of the cupula of the affected semicircular canal and displaced otoconia from the otolith organs. The otoconia, which are normally located in the utricle, are dislocated. Subsequently, the otoconia become free floating and affected by gravity. When the head is moved to a provoking position, these otoconia move into the canal and displace the cupula, thus resulting in vertigo and nystagmus (Belafsky et al, 2000; Marcias et al, 2000). Although all three semicircular canals may be affected, posterior canal BPPV is the most common form due to its anatomical position (Herdman and Tusa, 1996).

BPPV was first described by Barany (1921). The classic indicators of this disease include: vertigo and rotary nystagmus (eyes moving clockwise or counterclockwise) towards the affected ear with a latency of several seconds. The nystagmus typically lasts for less than one minute and is fatigable after multiple positioning techniques (Lanska and Remler, 1997; Konrad et al, 1999; Belafsky et al, 2000).

These classic indicators are elicited by specific positioning techniques used in standard vestibular assessment (Dix and Hallpike, 1952; Belafsky et al, 2000; Nunez et al, 2000). These positioning techniques require the clinician to place the patient on his/her back with hyperextension of the neck and the head turned to one side or the other. A positive finding is indicated when this positioning provokes the rotary nystagmus and vertigo already described.

Although the positioning techniques described above are able to definitively diagnose BPPV, the test often considered the most useful in the diagnosis of peripheral vestibular diseases, in general, is the bithermal caloric test. The caloric test, a component

of videonystagmography (VNG) assessment, is used to stimulate the horizontal semicircular canals so that the response of each ear can be compared. The patient lies supine with the head elevated to 30°, aligning the horizontal semicircular canals in a vertical position. Each ear is separately stimulated using a warm and cool stimulus. A warm stimulus heats the endolymph in the test ear and causes a convection current that flows upward and deflects the cupula toward the utricle. Movement of the cupula toward the utricle results in an excitatory response. This excitation creates an asymmetry in primary afferent firing between the inner ears and triggers nystagmus with the fast phase beating in the direction of the stimulated ear and the slow phase toward the opposite direction. Irrigating the ear with cool air or water will decrease the temperature of the endolymph, causing it to flow downward and deflecting the cupula away from the utricle. This results in an inhibitory response and causes the fast phase component of the nystagmus to beat away from the stimulated ear. The slow-phase velocity of the nystagmus is the value most commonly used for assessment of the caloric response (Jacobson et al, 1997).

Caloric testing allows evaluation of the integrity of the right and left horizontal semicircular canals, separately, and allows for comparison of the strength of the response of each system. A difference of more than 20% between responses from the ears is considered significant for a weak vestibular system (Jongkees et al, 1962). This finding is called a unilateral weakness, implying a peripheral vestibular disorder on the side with the diminished response (Jacobson et al, 1997). In a unilateral weakness, there is an asymmetry in the magnitude of information entering the brainstem, thus generating different nystagmus characteristics from each ear. In other words, the two ears are

reacting differently (asymmetrically) to the same type of stimulation. Caloric testing allows for diagnosis of asymmetric function or unilateral weakness in the peripheral vestibular system (Jacobson et al, 1997), a peripheral vestibulopathy.

There are numerous disorders that may cause a unilateral peripheral vestibulopathy and are reportedly associated with BPPV (Pollak et al, 2002). The literature suggests that vestibular neuritis, labyrinthitis, or Meniere's disease may be existing inner ear disorders that later cause BPPV once the patient is out of the acute phase (Hughes and Proctor, 1997; Bath et al, 2000). Gans (2000) reports that patients with any of these underlying vestibular disorders would likely demonstrate a unilateral weakness on the caloric subtest. Cerebrovascular disease including stroke/transient ischemic attacks (TIAs), basilar artery insufficiency, vertebrobasilar insufficiency, or migraine have also been reported to cause a unilateral weakness on caloric testing and may precede BPPV (Katasarkas and Kirkham, 1978; Kurizky et al, 1981; Togliola et al, 1981; Baloh et al, 1987; Hughes and Proctor, 1997; Kumar et al, 1998; Pollak et al, 2002).

Head trauma has also been reported as precipitating the onset of BPPV, but has not been reported to cause peripheral vestibulopathy (Karlberg et al, 2000). Disorders affecting the peripheral vestibular system that may be present prior to BPPV without a reported incidence of unilateral weakness are chronic ear infections and a history of ear surgery (Hughes and Proctor, 1997; Karlberg et al, 2000; Pollak et al, 2002).

The incidence of peripheral vestibulopathy for BPPV patients has been reported in the range of 3% to 50% (Hughes and Proctor, 1997; Gans, 2000; Karlberg et al, 2000; Pollak et al, 2002). This broad range may reflect variability among the patients or in the

methodology of the various studies. For example, patients with an underlying vestibular disorder may be more likely to present with a unilateral weakness on the caloric subtest than patients without an underlying vestibular disorder. Based on the published data, it is clear that not all patients diagnosed with BPPV will have a peripheral vestibulopathy on the affected side. However, for patients with BPPV and a unilateral weakness on the caloric test, there is sufficient evidence to be concerned about a more serious underlying inner ear disorder. This underlying vestibular disorder may cause imbalance and instability for the patient. Therefore, the purpose of this study was to determine the incidence of peripheral vestibulopathy in two groups of BPPV patients using data collected during caloric testing on these patients. One group had a positive history of prior otologic disease and the other group had a negative history of prior otologic disease. It was hypothesized that a greater incidence of peripheral vestibulopathy would be observed for the group with prior otologic disease and differences between the groups in the current investigation may explain the variability among reports investigating the incidence of peripheral vestibulopathy in patients with BPPV.

## **METHODS**

### **Participants**

A retrospective study of 157 patients assessed and treated between January, 1999 and August, 2002 at the American Institute of Balance (AIB) was conducted. The patients selected for review in this study were seen for evaluation and treatment of vertigo and were diagnosed with BPPV of the posterior canal. Any patient with a negative finding on the modified Dix-Hallpike (Gans, 2002) was excluded from this study. Those patients identified as having horizontal canal, anterior canal, or recurrent



BPPV upon initial diagnosis or secondary to migration following initial treatment, were excluded from the study. The patients meeting the inclusion criteria were divided into two subject groups: 49 subjects aged 40 - 89 years (mean = 66) with a prior history of otologic disease (PosHx) and 108 subjects aged 25 - 95 years (mean = 70.4) without a prior history of otologic disease (NegHx). A t-test indicated no difference in age between the two groups,  $t(48) = -1.83, p = 0.70$ . There were 34 females (56%) in the PosHx group and 78 females (62%) in the NegHx group.

### **Procedures**

All patients received a complete assessment using the standard test protocol of AIB. Results from this protocol were used to determine subject eligibility and subsequent grouping. The three main factors considered included diagnosis of posterior canal BPPV, otologic history, and caloric testing results.

#### ***A.I.B. Standard Test Protocol***

Case history information regarding detailed presenting complaints, family history, drug history, and associated symptoms (hearing loss, tinnitus, and aural pressure) was obtained. History of prior otologic disease such as labyrinthitis, vestibular neuritis/labyrinthine ischemia, and Meniere's disease were recorded. Prior medical history including cardiovascular findings, chronic intractable motion intolerance, stroke/TIA, diabetes, migraine, and head trauma were also noted.

The otologic examination consisted of otoscopic inspection of the tympanic membranes. A vertebral artery screening test and Babinsky reflexes were performed. Balance was assessed by the Sensory Organization Performance test, including the Romberg and Fukuda step test. Audiometry, immittance, and otoacoustic emissions

results were obtained. If the patient was not previously referred for imaging studies, an auditory brainstem response (ABR) was performed. Vestibular autorotation and a complete VNG test battery including: ocular motor pursuit, gaze, high frequency head-shake, air calorics, and positional tests were performed. All testing was performed in one appointment.

### ***Diagnosis of BPPV***

Diagnosis of posterior canal BPPV was based on the presence of a positive finding for the modified Dix-Hallpike test. Classic positive indicators include: 1) transient rotary nystagmus toward the undermost ear; 2) subjective vertigo that parallels the nystagmus; 3) latency of onset of nystagmus.

### ***Otologic History***

The PosHx group consisted of those patients reporting labyrinthitis, vestibular neuritis/ labyrinthine ischemia, or Meniere's disease on their case history form or during their initial case history interview. Those reporting none of these disorders were placed in the NegHx group. Patients placed in the NegHx group reporting head trauma, cardiovascular disease, stroke/TIA, diabetes, migraine, and chronic intractable motion intolerance on their case history forms, or during their initial history interview were further categorized by these conditions.

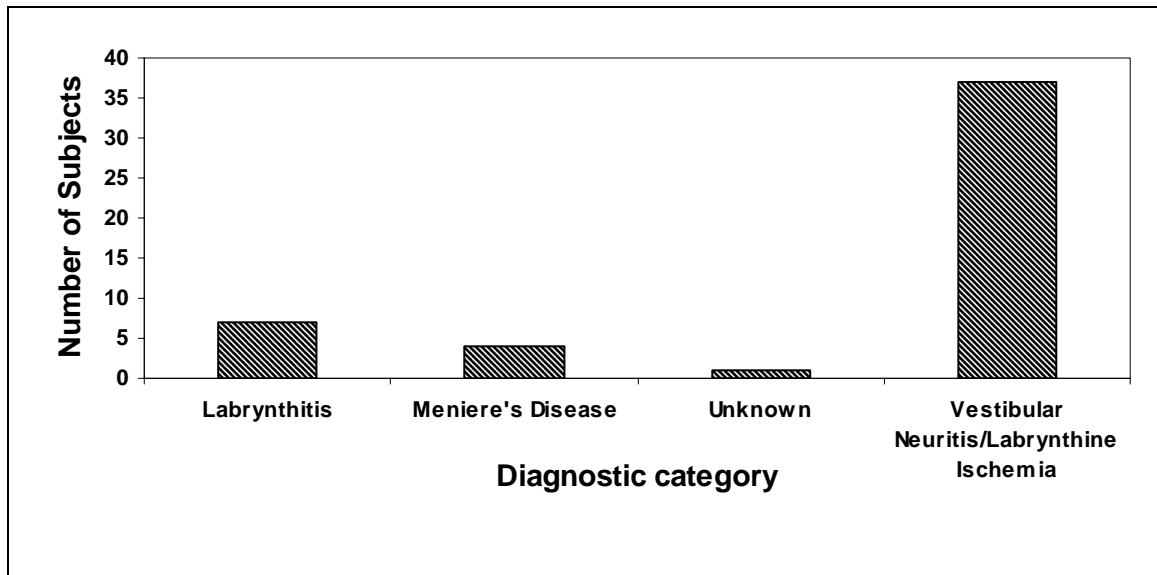
### ***Unilateral Weakness***

The incidence of peripheral vestibulopathy in each of these groups was determined using caloric testing results and the Jongkees et al. (1962) formula. Unilateral weakness was defined as a difference in the mean slow phase velocity of 23% or greater between ears. This is a stricter criterion than the 20% difference used in some

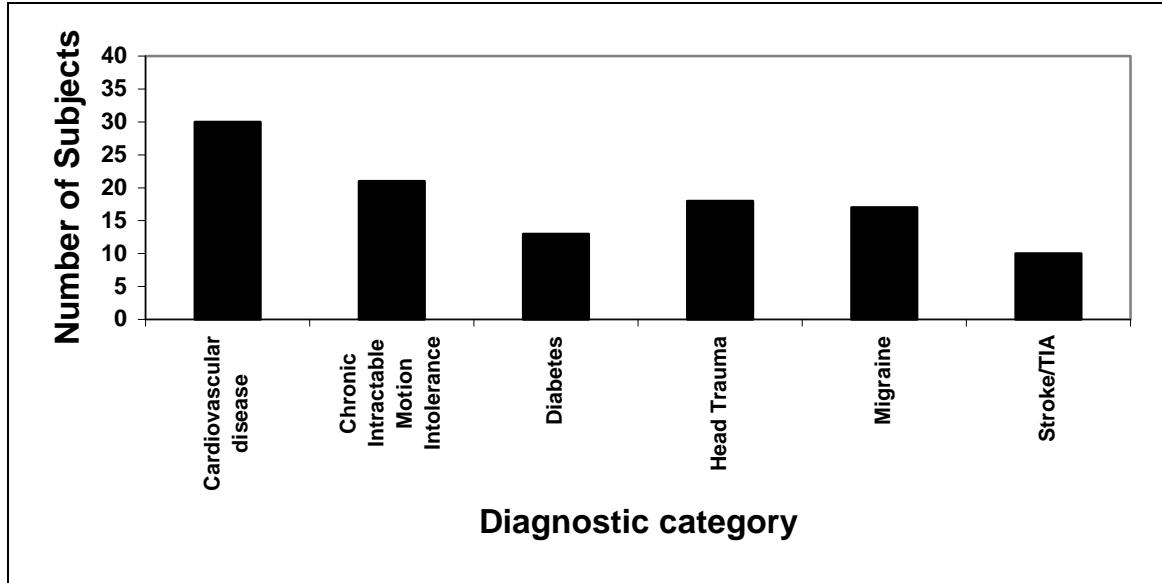
labs, but is less strict than the criterion reported in other studies (Karlberg et al, 2000). Subjects meeting the 23% or greater unilateral weakness were considered to have an underlying vestibular disorder or peripheral vestibulopathy.

## RESULTS

Each group was analyzed in terms of diagnostic factors associated with BPPV. This information is shown for the PosHx group in Figure 1 and for the NegHx group in Figure 2. As shown, the most common diagnostic category for the PosHx group was vestibular neuritis/ labyrinthine ischemia. This was the factor for 37 of the subjects (79.5%) in the PosHx group. For the subjects in the NegHx group, cardiovascular disease was the most prominent associated factor (27.8%). The right ear was the ear with BPPV in 32 (65%) of the subjects in the PosHx group and 57 (53%) of the subjects in the NegHx group.



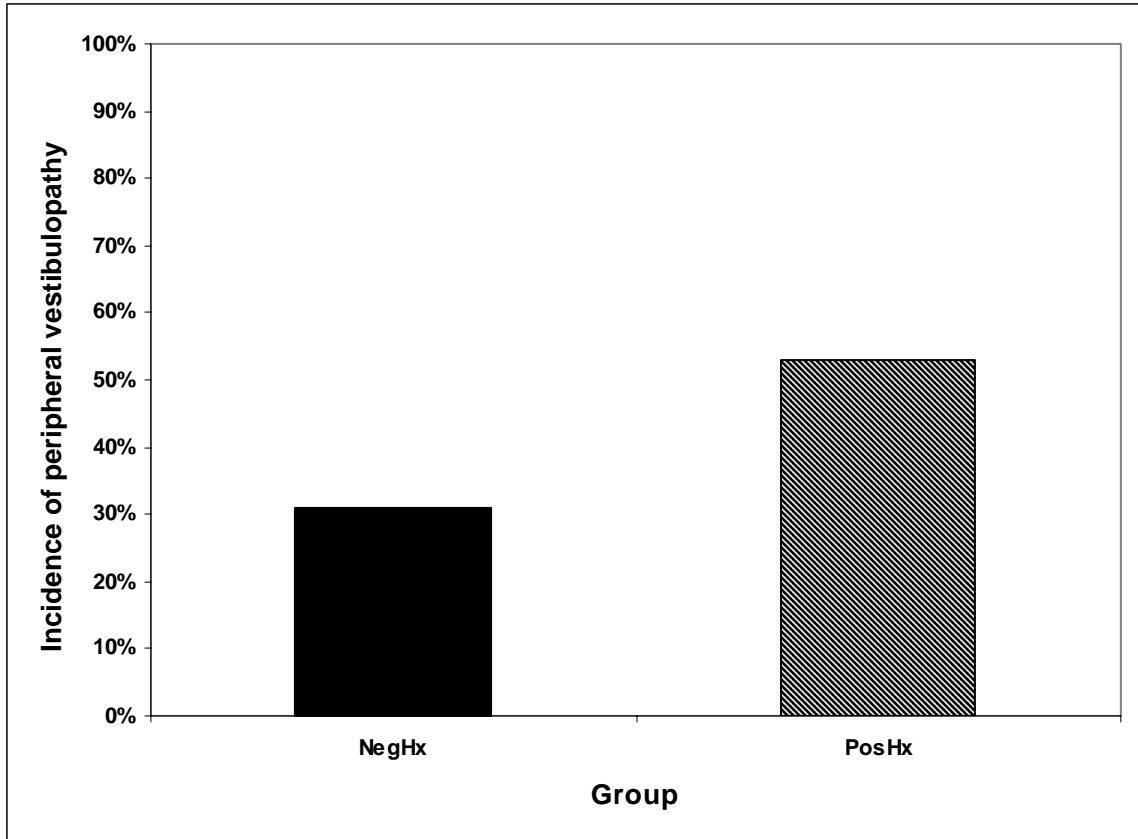
**Figure 1.** Distribution of subjects by diagnostic category for the PosHx group.



**Figure 2.** Distribution of subjects by diagnostic category for the NegHx group.

Results were averaged and examined for trends. As shown in Figure 3, the incidence of peripheral vestibulopathy (unilateral weakness greater than 23%) was 53.1% for the PosHx group and 30.6% for the NegHx. To determine if the incidence of peripheral vestibulopathy for each group was different, the data were examined using a chi-square ( $X^2$ ) analysis. Using this analysis, it was determined that the PosHx group had a significantly higher incidence of peripheral vestibulopathy than the NegHx group ( $X^2 = 7.28, p < 0.05$ ). In addition, the number of patients with BPPV and peripheral vestibulopathy in the same ear was 17 (65%) in the PosHx group and 17 (52%) in the NegHx group.

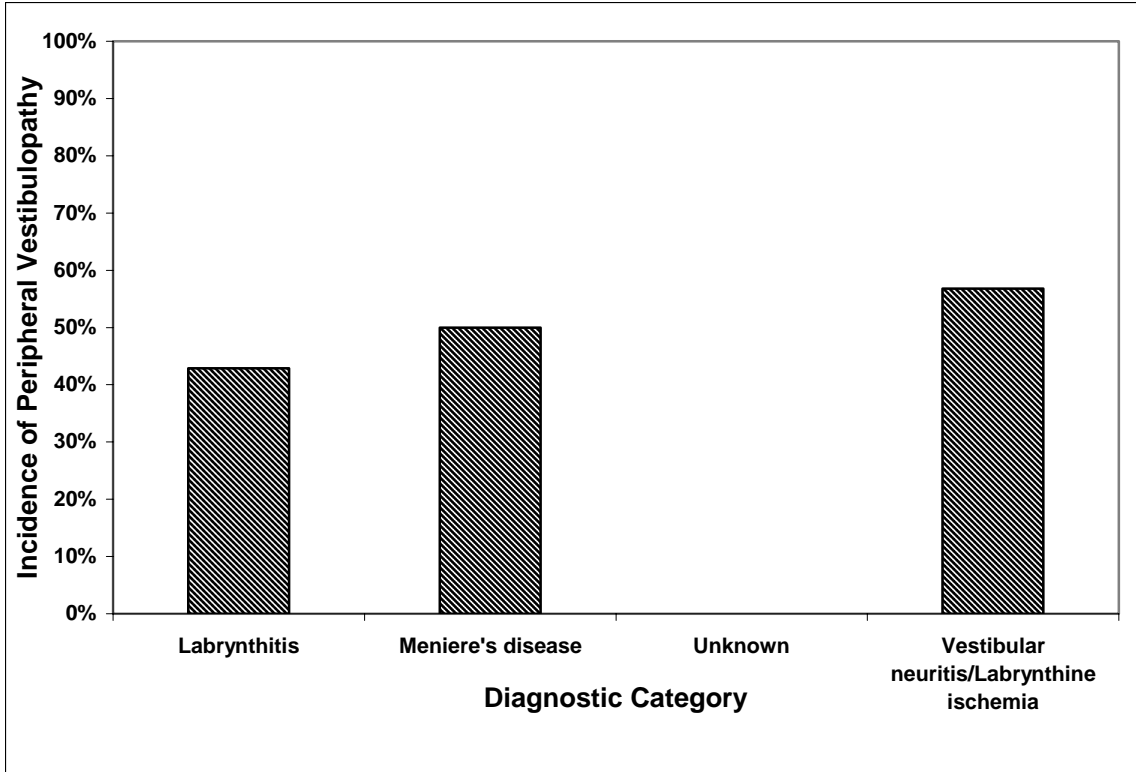
Average unilateral weakness results were also determined. All values of unilateral weakness, including those that did not meet the 23% criterion, were included in this analysis. An independent t-test revealed a significant difference between the mean unilateral weakness results for the two groups,  $t(48) = 2.14, p = 0.03$ . This result indicated



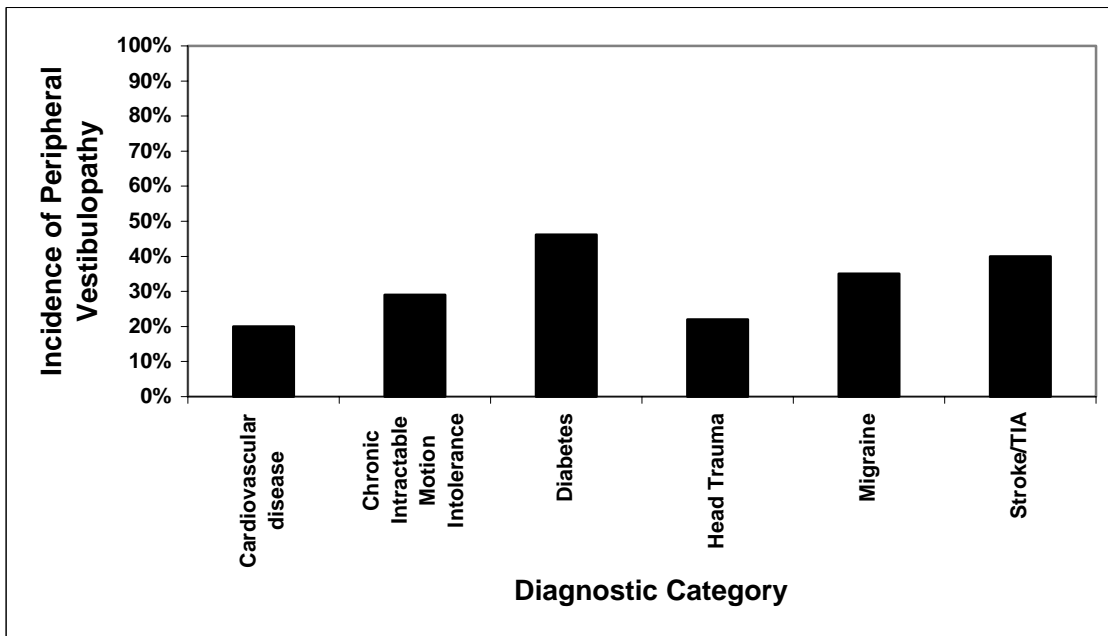
**Figure 3.** Incidence of peripheral vestibulopathy in the PosHx and NegHx groups. The filled bar represents the percentage of subjects with peripheral vestibulopathy from the NegHx group and the hashed bar represents the percentage of subjects with peripheral vestibulopathy from the PosHx group.

that the PosHx group had a significantly larger average caloric weakness (25.8%) compared to the NegHx group (17.8%).

As shown in Figure 4, a history of vestibular neuritis/labyrinthine ischemia resulted in the greatest incidence of caloric weakness in the PosHx group with over half (56.8%) of these subjects exhibiting peripheral vestibulopathy. Meniere's disease (50%) and labyrinthitis (42.9%) produced slightly lower incidences of caloric weakness in the PosHx group.



**Figure 4.** Incidence of peripheral vestibulopathy by diagnostic category in the PosHx group.



**Figure 5.** Incidence of peripheral vestibulopathy by diagnostic category in the NegHx group.

Subjects with diabetes and stroke/TIA yielded the greatest incidences of unilateral weakness in the NegHx group with 46.2% and 40%, respectively. Subjects with migraine exhibited an incidence of 35.3% and those with a history of head trauma demonstrated the smallest incidence of caloric weakness (22.2%) in the NegHx group. This data is shown in Figure 5.

## **DISCUSSION**

Presently in the literature, the incidence of peripheral vestibulopathy in BPPV patients ranges from 3 to 50% (Hughes and Proctor, 1997; Gans, 2000; Karlberg et al, 2000; Pollak et al, 2002). The purpose of this study was to attempt to explain the variability among these studies by determining if there was a greater incidence of unilateral weakness in BPPV patients with a positive history of otologic disease compared to the incidence of unilateral weakness in BPPV patients without such a history. Results indicated that BPPV patients with a prior history of ear disease not only have a greater incidence of underlying peripheral vestibulopathy (53.1%) than those without a history of an inner ear disorder (30.6%), but the BPPV patients with a positive history of otologic disease also show a larger average unilateral weakness (25.8%) than those BPPV patients without a prior otologic disorder (17.8%).

It is important to understand that a prerequisite for posterior canal BPPV is, at least, partially intact functioning of the posterior semi-circular canal. Karlberg et al. (2000) suggest that any inner ear disorder that causes displacement of otoconia without complete impairment of posterior canal function could produce posterior canal BPPV. Karlberg et al. also indicate that audiometry and caloric testing should be performed on

all patients with BPPV in order to recognize any inner ear disorder that might be causing the BPPV.

The present study found that a prior history of vestibular neuritis/labyrinthine ischemia produced the greatest incidence of peripheral vestibulopathy (56.8%). Approximately 43% of the patients reporting a history of labyrinthitis also had a peripheral vestibulopathy. The literature confirms this relationship between BPPV and acute unilateral peripheral vestibulopathy (i.e. vestibular neuritis/labyrinthine ischemia and labyrinthitis) (Katsarkas and Kirkham, 1978; Baloh et al, 1987; Harada et al, 1993; Karlberg et al, 2000). In these other reports, patients were considered to have an acute unilateral peripheral vestibulopathy if they had a history of a single attack of sudden spontaneous vertigo slowly decreasing over days, a unilateral weakness (>25% caloric weakness), and no relevant auditory symptoms or findings. They stated that the finding of posterior canal BPPV nystagmus is a sign of the functional integrity of the vestibulo-ocular reflex pathway from the posterior semi-circular canal (Harada et al, 1993). Vestibular neuritis and labyrinthitis often affect the superior vestibular nerve and the structures it innervates, including the cristae of the anterior and horizontal semi-circular canals and the macula of the utricle. Vertigo and a unilateral weakness on caloric testing are produced by an impairment of the horizontal canal. Damage to the utricle could also lead to displacement of the otoconia and cause posterior canal BPPV (Karlberg et al, 2000).

Meniere's disease has also been demonstrated to cause BPPV and peripheral vestibulopathy (Stahle and Bergman, 1967; Thomas and Harrison, 1971; Hulshof and Baarsma, 1981; Hughes and Proctor, 1997; Bath et al, 2000). In the current study



Meniere's disease was present in 2% of the patients. This finding is consistent with earlier studies reporting that 1 - 2% of BPPV cases were associated with Meniere's disease (Karlberg et al, 2000). Fifty percent of these Meniere's patients also had a peripheral vestibulopathy. This is slightly lower than the 60 - 74% incidence of peripheral vestibulopathy reported to occur with Meniere's disease in the literature (Stahle and Bedrgman, 1967; Thomas and Harrison, 1971; Hulshof and Baarsma, 1981). This apparent discrepancy could be associated with the fact that the stage of the Meniere's disease, active or destructive, was not taken into account.

The pathophysiology underlying the relationship between Meniere's disease and BPPV is not yet completely known. It has been proposed that loose otoconia could cause a decrease in endolymphatic absorption, resulting in Meniere's disease secondary to BPPV. But, it has also been proposed that endolymphatic hydrops might damage the utricle, resulting in loose otoconia and BPPV secondary to Meniere's disease. The latter explanation is thought to be the most realistic (Karlberg et al, 2000).

The literature also supports the finding of cerebrovascular disease, including stroke/ TIA and migraine, resulting in a peripheral vestibulopathy (Grad and Baloh, 1989; Hughes and Proctor, 1997; Kumar et al, 1998; Pollak et al, 2002). Migraine and BPPV in the same individual have also been frequently reported (Schiller and Herdberg, 1960; Kayan and Hood, 1984). For patients with a history of migraine, it is thought that the BPPV is caused by vascular labyrinthine injury associated with the migraine disorder (Hughes and Proctor, 1997). The current study found a 35.3% incidence of peripheral vestibulopathy in the patients reporting migraine during case history. This is in agreement with reports in the literature suggesting 22 - 44% of patients with migraine

will exhibit peripheral vestibulopathy (Kuritzky et al, 1981; Toglia et al, 1981).

Stroke/TIA have also been reported to produce peripheral vestibulopathy and precede BPPV (Katsarkas and Kirkham, 1978; Baloh et al, 1987; Hughes and Proctor, 1997; Kumar et al, 1998; Pollak et al, 2002). In the current study, forty percent of the BPPV patients reporting a history of stroke/TIA also have a peripheral vestibulopathy. Grad and Baloh (1989) reported that 42% of patients with cerebrovascular disease also had a peripheral vestibulopathy. A history of a stroke/TIA has also been reported to be a central cause of dizziness (Bath et al, 2000).

In previous studies, a history of head trauma was present in 14 - 27% of patients (Karlberg et al, 2000). The current study found a history of head trauma in 11% of the subjects selected. Head trauma has been established to precede BPPV, but not to cause a unilateral weakness (Karlberg et al, 2000). This study found 4 of the 18 (22.2%) patients reporting head trauma also had a unilateral weakness on caloric testing.

Cardiovascular disease, such as postural hypotension or congestive heart failure, has also been reported to cause dizziness (Oghalai et al, 2000). However, cardiovascular disease has not been proven to cause a peripheral vestibulopathy or to precede BPPV. In the current study, 27% of the BPPV patients reporting a history of cardiovascular disease also had a peripheral vestibulopathy.

Diabetes mellitus was the diagnostic category producing the greatest incidence of peripheral vestibulopathy (46.2%) in the NegHx group. Diabetes accelerates atherosclerosis, which may damage the labyrinthine, visual, and somatosensory function. It also causes peripheral neuropathy and diabetic retinopathy (Konrad et al, 1999).

Patients presenting with BPPV and an additional vestibulopathy may have a higher incidence of ongoing symptoms following a particle repositioning maneuver than those patients with pure BPPV (Pollak et al, 2002). This may suggest that a patient with BPPV secondary to vestibular neuritis may be less likely to clear from a single treatment versus an idiopathic occurrence of BPPV. This may be caused by an uncompensated horizontal canal induced non-paroxysmal dizziness or by an unidentified vertical canal, otolith, or central vestibular dysfunction leading to positional vertigo (Pollak et al, 2002). It has also been demonstrated that patients with peripheral vestibulopathy have a substantially better outcome than patients with central vestibular dysfunction (Pollak et al, 2002).

In general, 90% of patients with dizziness can be diagnosed by history alone (Bath et al, 2000). Obtaining a detailed patient history allows the clinician to determine if the patient has had an otologic event, often in the absence of hearing loss. It also allows the clinician to properly triage patients for vestibular rehabilitation therapy including canalith repositioning maneuvers, adaptation exercises, balance re-training, and fall prevention in the elderly population. This is extremely important since treatment for BPPV, vestibulopathy, and disequilibrium has been shown to be highly efficacious when properly administered (Gans and Harrington-Gans, 2002). Some individuals with BPPV complain of reduced balance, ataxia, and/ or lightheadedness following the treatment of the positional vertigo caused by BPPV (Konrad et al, 1999). An underlying vestibular disorder may be the cause of balance problems in these patients (Nadol and Schuknecht, 1989). The findings of this study may prove to be clinically significant in the management of these patients.

## SUMMARY AND CONCLUSIONS

The purpose of this experiment was to investigate the incidence of peripheral vestibulopathy in BPPV patients with and without a prior history of otologic disease. Data were collected for a group of patients with a positive history of otologic disease and for a group of patients with a negative history of otologic disease. All patients included in this study were diagnosed with posterior canal BPPV.

A greater incidence of peripheral vestibulopathy and a larger average unilateral weakness was found for the patients with a positive otologic history than for the patients with a negative otologic history. Specific conclusions were: 1) patients with posterior canal BPPV and a positive otologic history are more likely to have a peripheral vestibulopathy than patients with posterior canal BPPV and a negative otologic history, 2) incidence rates of peripheral vestibulopathy reported in the literature for posterior canal BPPV are likely to be influenced by the relative numbers of subjects with and without prior otologic history, 3) for this study, the most common diagnostic category for the positive history group was vestibular neuritis/labyrinthine ischemia and this category also presented with the highest incidence of peripheral vestibulopathy, 4) the most common diagnostic category for the negative history group was cardiovascular disease but the category that presented with the highest incidence of peripheral vestibulopathy for this group was diabetes.

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