Original Article

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Prevalence of vestibulopathy in benign paroxysmal positional vertigo patients with and without prior otologic history

Prevalencia de vestibulopatía en pacientes con vértigo postural paroxístico benigno (BPPV), con y sin historia previa de patología otológica

Abstract

The purpose of this study was to determine the prevalence of reduced or absent labyrinthine reactivity (vestibulopathy) in two groups of participants with posterior canal BPPV. One group had prior diagnosis of otologic disease (positive history group). No one in the second group had ever been diagnosed with otologic disease (negative history group). Caloric responses were retrospectively analyzed for the two groups. Patients with a positive history exhibited a greater prevalence of vestibulopathy than patients with a negative history. 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 history of otologic disease are more likely to present with vestibulopathy, than patients with BPPV and no history of otologic disease. This finding supports the benefit of complete vestibular evaluation in patients with BPPV to ensure comprehensive and successful treatment outcome.

Sumario

El propósito de este estudio fue determinar la prevalencia de reducción o ausencia de reactivación laberíntica (laberintopatía) en dos grupos de participantes después de BPPV del canal posterior. Un grupo tenía diagnóstico previo de patología otológica (grupo de historia positiva). Nadie del segundo grupo había sido diagnosticado con patología otológica (grupo de historia negativa). Las respuestas calóricas se analizaron retrospectivamente en los dos grupos. Los pacientes con una historia positiva mostraron una prevalencia mayor de vestibulopatía que los pacientes con historia negativa. El grupo de historia positiva, en promedio, también mostró una mayor debilidad unilateral que aquellos con una historia negativa. Concluimos que los pacientes con BPPV e historia de patología otológica presentan vestibulopatía con mayor frecuencia que los pacientes con BPPV sin historia de patología otológica. Estos hallazgos apoyan el beneficio de una evaluación vestibular completa en pacientes con BPPV para asegurar un tratamiento integral y exitoso.

Benign paroxysmal positional vertigo (BPPV) is a common cause of dizziness characterized by intense positionally-provoked vertigo of brief duration (Lanska & Remler, 1997; Bath et al, 2000; Pollak et al, 2002). BPPV is caused by interaction of the cupula of the affected semicircular canal and otoconia from the otolith organs (Schuknecht, 1969; Parnes & McClure, 1992). The otoconia, which are normally located in the utricle, become dislocated and move into the affected semicircular canal. When the head is placed in a provoking position, the otoconia move in the canal and displace the cupula. This results in vertigo and rotary nystagmus towards the affected ear (Belafsky et al, 2000; Macias et al, 2000). The nystagmus typically lasts for less than one minute and is fatigable after multiple positionings (Lanska & Remler, 1997; Konrad et al, 1999; Belafsky et al, 2000). Although all three semicircular canals may be affected, posterior canal BPPV is the most common form due to the anatomical position of this canal in relation to the utricle (Herdman & Tusa, 1996).

The test often considered the most useful in the diagnosis of vestibular diseases is the bithermal caloric test. The caloric test is used to stimulate the horizontal semicircular canals so that the response of each labyrinth can be compared. Each ear is separately stimulated using a warm and cool stimulus. The slow-phase velocity of the nystagmus is the value commonly used for assessment of the caloric response (Jacobson et al, 1997). Analysis of nystagmus slow-phase velocity allows for evaluation of the integrity of the right and left horizontal semicircular canals, separately, as well as for comparison of the strength of the response of each system. A difference of more than 20% between responses from the ears is considered clinically significant for a weak vestibular system (Jongkees et al, 1962). This finding is called a unilateral weakness, implying a 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. The two ears are reacting differently (asymmetrically) to the same type of stimulation. The finding of asymmetric function or unilateral weakness in the vestibular system (Jacobson et al, 1997) may be termed a vestibulopathy.

There are numerous disorders that may cause a concomitant vestibulopathy and BPPV (Pollak et al, 2002). The literature suggests that vestibular neuritis, labyrinthitis, or Meniere's

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disease may be existing inner ear disorders that later cause BPPV once the patient is out of the acute phase (Hughes & Proctor, 1997; Bath et al, 2000). Gans (2000) reports that patients with any of these underlying vestibular disorders are likely to demonstrate a unilateral vestibulopathy 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 vestibulopathy on caloric testing and BPPV (Katsarkas & Kirkham, 1978; Kuritzky et al, 1981; Toglia et al, 1981; Baloh et al, 1987; Hughes & Proctor, 1997; Kumar et al, 1998; Pollak et al, 2002).

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

Patients with BPPV but without a prior history of otologic pathology have been described as presenting with primary BPPV (Karlberg et al, 2000). Patients with BPPV and a definite history of prior otologic pathology are described as presenting with secondary BPPV (Karlberg et al, 2000). The prevalence of vestibulopathy in BPPV patients has been reported in the range of 13% to 50% (e.g., Blessing et al, 1986; Hughes & Proctor, 1997; 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. Studies investigating patients with primary BPPV report prevalence rates from 30% (Baloh et al, 1987) to 50% (Hughes & Proctor, 1997). On the other hand, studies investigating patients with secondary BPPV report prevalence rates of 43% (Pollak et al, 2002) and 44% (Karlberg et al, 2000). The report by Blessing et al (1986) indicates a 13% prevalence of vestibulopathy, but this appears to include participants with both primary and secondary BPPV. The report by Stockwell (2000) indicates a 20% prevalence of vestibulopathy but also does not distinguish between primary and secondary BPPV, as the data were from a remote testing service and patient history information were unavailable. To our knowledge, no one has reported the prevalence of vestibulopathy in patients with primary and secondary BPPV in the same investigation. This makes it difficult to rule out methodological issues as explaining the variability between different studies.

Based on the published data, it is clear that not all patients diagnosed with BPPV will have a vestibulopathy. However, for patients with BPPV and a vestibulopathy on the caloric test, there is sufficient evidence to be concerned about an underlying vestibular disorder. This underlying vestibular disorder may cause imbalance, instability, oscillopsia, and head motion intolerance for the patient. Further, more extensive therapy may be indicated after the patient has been treated for BPPV (Pollak et al, 2002). Therefore, the purpose of this study was to determine the prevalence of vestibulopathy in two groups of BPPV 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 prevalence of vestibulopathy would be observed for the group with prior otologic disease. Any differences observed in prevalence between the two groups in the current investigation may explain the variability among reports investigating the prevalence of vestibulopathy in patients with BPPV.

Methods

Participants

A retrospective review of 157 charts of patients assessed and treated between January, 1999, and August, 2002, at the American Institute of Balance (AIB) was conducted. Approval was obtained by the Institutional Review Board of the University of South Florida prior to initiation of this investigation. The charts selected for review in this study were from patients seen for evaluation and treatment of vertigo, who were subsequently diagnosed with BPPV of the posterior canal. The patients meeting this inclusion criterion were divided into two participant groups based on their prior otologic history. Using this criterion, 49 participants were included in the group with positive history of otologic disease (PosHx), and 108 participants were included in the group with negative history of otologic disease (NegHx). Specific information regarding participant groups is found in Table 1. A t-test indicated no difference in age between the two groups, t (48) = −1.83, p = 0.70.

Procedures

DIAGNOSIS OF BENIGN PAROXYSMAL POSITIONAL VERTIGO
Diagnosis of posterior canal BPPV was based on a positive finding for the modified Dix-Hallpike test. The modification is that the examiner is positioned behind the patient. This results in enhanced ease of performance of the maneuver. Classic positive indicators were used to confirm the presence of BPPV and also the involved ear. These included: 1) transient rotary-torsional nystagmus toward the undermost ear; 2) subjective vertigo with the nystagmus; and 3) latency of nystagmus onset. All patients were evaluated with infra-red video-oculography with a video recording for review and confirmation.

OTOLOGIC HISTORY
The PosHx group consisted of those participants previously diagnosed with labyrinthitis, vestibular neuritis/labyrinthine ischemia, or Meniere's disease, as indicated on their case history form or during their initial case history interview. Figure 1 shows the distribution of otologic diagnoses for this group. As shown, 37 (76%) of the participants had an otologic diagnosis of vestibular neuritis/labyrinthine ischemia. Those participants with no diagnosed otologic disorders were placed in the NegHx group.

UNILATERAL WEAKNESS
The prevalence of vestibulopathy in each of these groups was determined using caloric test results and the Jongkees et al (1962)

Table 1. Number of participants, age, gender, and involved ear information for the two groups

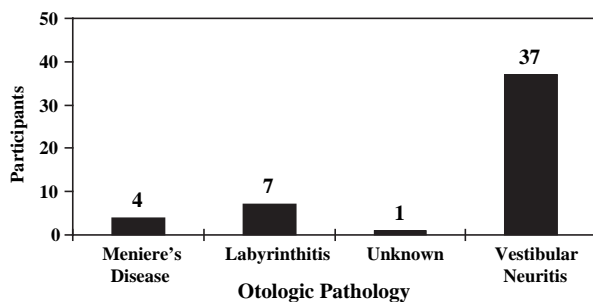| Group	n	Mean Age	Gender	Involved Ear
PosHx	49	66 years; (40−89)	22 males; 27 females	32 right; 17 left
NegHx	108	70 years; (25−95)	41 males; 67 females	57 right; 51 left

Figure 1. Number of participants with specific otologic diagnoses is shown for the PosHx group.

formula. Unilateral weakness was defined as a difference in the mean slow phase velocity of 23% or greater between ears. This is a more strict criterion than the 20% difference used in some labs, but is less strict than the criterion reported in some other studies (Karlberg et al, 2000). Participants with unilateral weakness of 23% or greater were considered to have a vestibulopathy.

Results

As shown in Figure 2, the prevalence of vestibulopathy was 53.1% (26) for the PosHx group and 30.6% (33) for the NegHx group. To determine if the prevalence for each group was different, the data were examined using a chi-square (χ^2) analysis. Using this analysis, it was determined that the PosHx group had a significantly higher prevalence of vestibulopathy than the NegHx group ($\chi^2 = 7.28$, p <0.05).

To further explore the differences regarding labyrinthine reactivity between these two groups, average unilateral weakness results were also determined. All values of unilateral weakness for each participant in each group, 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 that the PosHx group had a significantly larger (25.8%) asymmetry in labyrinthine reactivity (caloric weakness) compared to the NegHx group (17.8%).

Discussion

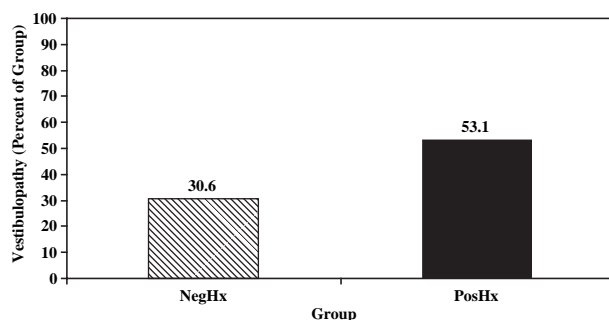The prevalence of vestibulopathy in BPPV patients is reported to range from 13 to 50% (Blessing et al, 1986; Hughes & Proctor, 1997; Karlberg et al, 2000; Pollak et al, 2002). The purpose of the current investigation was to attempt to explain the variability among these studies by determining if there was a greater prevalence of vestibulopathy in BPPV patients with a positive history of otologic disease compared to the prevalence of vestibulopathy in BPPV patients without such a history. To our knowledge, prevalence rates have not been reported for these two groups in a single study.

Results of the current study indicated that BPPV patients with a prior history of ear disease not only have a greater prevalence of vestibulopathy than those without a history of inner ear disorder; but the BPPV patients with a positive history of otologic disease also show a larger average unilateral weakness than those BPPV patients without a prior otologic disorder.

Comparison to previous studies
The results of this investigation are consistent with the hypothesis that patients with BPPV and a prior history of otologic disease are more likely to exhibit a vestibulopathy on caloric testing than patients with BPPV and no history of otologic disease. Previous studies investigating primary BPPV suggest the prevalence of vestibulopathy is from 30% to 50% (Baloh et al, 1987; Hughes & Proctor, 1997) (see Table 2). The 30% prevalence of vestibulopathy reported by Baloh et al (1987) is in close agreement with the results for the NegHx group in the current study (30.6%). On the other hand, the data of Hughes and Proctor (1997) suggests a higher prevalence (50%) of vestibulopathy for patients with primary BPPV. It is difficult to explain this difference, but one possibility is that a lower criterion for unilateral weakness was used compared to the 23% asymmetry used in the current study. As stated previously, some labs use a value of 20% to determine the presence of a clinically significant caloric asymmetry, while others may use a stricter criterion of 25%. Using a 20% asymmetry would have increased the prevalence of vestibulopathy for both groups in the current study.

The current study reports a 53.1% prevalence of vestibulopathy for patients with a positive history of otologic disease. This is slightly higher than the 43–44% prevalence of vestibulopathy reported by investigations for patients with secondary BPPV. Pollak et al (2002) reported data on 58 patients with BPPV. Of the 58 patients, 35 had additional vestibular pathology and could be considered to have secondary BPPV. Fifteen (43%) of the patients with secondary BPPV had a vestibulopathy as demonstrated by caloric testing. This is in close agreement with Karlberg et al (2000) who reported on 81 patients with secondary BPPV. The Karlberg et al study was specific regarding criteria for secondary BPPV. Patients were only included in this group if they had an ipsilateral inner ear disease. Of the patients assigned to this group, 36 (44%) had either an acute or chronic unilateral peripheral vestibulopathy. Karlberg et al (2000) also used a criterion of >25% asymmetry for a caloric weakness to be present. If these authors had used 23% asymmetry as the criteria, it is possible they would have reached a prevalence rate similar to the current study.

Another factor that may have increased the prevalence rate in the current study is the fact that 76% of the participants in the PosHx group had a diagnosis of vestibular neuritis. There is evidence that patients with vestibular neuritis will exhibit a

Figure 2. Prevalence of vestibulopathy for participants in the PosHx and NegHx groups.

Table 2. Comparison of prevalence of vestibulopathy from current study to previous studies

Current Study	Previous Studies
NegHx Group 30.6%	Primary Benign Paroxysmal Positional Vertigo 30–50%, (Baloh et al, 1987; Hughes & Proctor, 1997)
PosHx Group 53.1%	Secondary Benign Paroxysmal Positional Vertigo 43–44%, (Pollak et al, 2002; Karlberg et al, 2000)

unilateral weakness on the caloric test 97–100% of the time (Bergenius & Borg, 1983; Corvera & Davalos, 1985). It is possible that given a majority of participants in our PosHx group with a diagnosis of vestibular neuritis, the prevalence of vestibulopathy found in the current study may be slightly higher than what would be expected for other reports with fewer patients with this diagnosis. This will be explored in greater detail in the next section.

Otologic disease and vestibulopathy
Table 3 lists otologic disease and comparison of prevalence of vestibulopathy on the current study and previous studies. The present study found that of the 37 patients with a prior history of vestibular neuritis/labyrinthine ischemia, 21 (56.8%) had a vestibulopathy. Three (43%) of the patients reporting a history of labyrinthitis also had a vestibulopathy. The literature confirms the relationship between BPPV and vestibular neuritis, labyrinthine ischemia and labyrinthitis (Katsarkas & 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 vestibulopathy, if they had a history of a single attack of sudden spontaneous vertigo slowly decreasing over days, a unilateral weakness ($>25\%$ caloric asymmetry), and no relevant auditory symptoms or findings. 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 horizonal semi-circular canals and the macula of the utricle. Vertigo and a vestibulopathy 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 vestibulopathy (Stahle & Bergman, 1967; Thomas & Harrison, 1971; Hulshof & Baarsma, 1981; Hughes & Proctor, 1997; Bath et al, 2000). In the current study, Meniere's disease

was present in 4 (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). Two (50%) of these Meniere's patients also had a vestibulopathy. This is slightly lower than the 60–74% prevalence of vestibulopathy reported to occur with Meniere's disease in the literature (Stahle & Bergman, 1967; Thomas & Harrison, 1971; Hulshof & 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).

Co-morbid factors and vestibulopathy
Although a higher prevalence of vestibulopathy was observed for the PosHx group, 30.6% of the participants in the NegHx group also had a vestibulopathy. Given this relatively high prevalence of vestibulopathy in the NegHx group, the possibility of non-otologic co-morbid factors that could cause a vestibulopathy were considered for both groups. Please see Table 4 for a listing of co-morbid factors and the prevalence of vestibulopathy for the current study.

The literature supports the finding of cerebrovascular disease, including stroke/TIA and migraine, resulting in a vestibulopathy (Grad & Baloh, 1989; Hughes & Proctor, 1997; Kumar et al, 1998; Pollak et al, 2002). Migraine and BPPV in the same individual have also been frequently reported (Schiller & Herdberg, 1960; Kayan & 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 & Proctor, 1997). The current study found a 35.3% (6) prevalence of vestibulopathy in the patients with no history of otologic disease and reporting migraine during the case history. This is in agreement with reports in the literature suggesting 22–44% of patients with migraine will exhibit vestibulopathy (Kuritzky et al, 1981; Toglia et al, 1981). None of the three patients reporting migraine in the PosHx group had a vestibulopathy.

A history of stroke/TIA has also been reported to produce vestibulopathy and BPPV (Katsarkas & Kirkham, 1978; Baloh et al, 1987; Hughes & Proctor, 1997; Kumar et al, 1998; Pollak et al, 2002). In the current study, 40% (4) of the BPPV patients in the NegHx group with a reported history of stroke/TIA also had a vestibulopathy. This is in close agreement with Grad & Baloh (1989) who reported that 42% of patients with cerebrovascular

Table 3. Comparison of prevalence of vestibulopathy for certain otologic diseases in the current study and in previous studies

Otologic Disease	Current Study	Previous Studies
Vestibular Neuritis/Labyrinthine Ischemia	56.8%	97–100%, (Bergenius & Borg, 1983; Corvera & Davalos, 1985)
Labyrinthitis	43%	
Meniere's Disease	50%	60–74%, (Stahle & Bergman, 1967; Thomas & Harrison, 1971; Hulshof & Baarsma, 1981)

Table 4. Co-morbid factors and prevalence of vestibulopathy for each group

	Prevalence of Vestibulopathy		
Pathology	PosHx	NegHx	Possible Vestibular Influence
Cardiovascular Disease	57%	27%	Compromised labyrinthine blood supply; Ischemic/athersclerotic changes
Diabetes	33%	46%	Accelerated atherosclerotic changes
Head Trauma	50%	22%	Labyrinthine concussion; Temporal bone fracture
Migraine	0%	35%	Vasoconstriction of labyrinth or vestibulo-cerebellum (22–44%, Kuritzky et al, 1981; Toglia et al, 1981)
Stroke/Ischemia	100%	40%	Cerebrovascular disease (42%, Grad & Baloh, 1989)

disease also had a vestibulopathy. All three of the patients in the PosHx group with a history of stroke/TIA also had a vestibulopathy.

In previous studies, a history of head trauma was present in 14–27% of patients (Katsarkas & Kirkham, 1978; Karlberg et al, 2000). The current study found a history of head trauma in 17% (18) of the participants in the NegHx group and 4% (2) participants in the PosHx group. Head trauma has been established to cause BPPV, but not to cause a unilateral weakness (Karlberg et al, 2000). This study found 22.2% (4) of the 18 patients in the NegHx group and reporting head trauma also had a vestibulopathy on caloric testing. Only one of the two patients in the PosHx group who reported head trauma had a vestibulopathy.

Cardiovascular disorders, such as postural hypotension or congestive heart failure, have also been reported to cause dizziness (Oghalai et al, 2000). However, cardiovascular disorders have not been proven to cause a vestibulopathy or BPPV. In the current study, 27% (8) of the NegHx patients reporting a history of cardiovascular disease also had a vestibulopathy. The prevalence of vestibulopathy in PosHx patients with a history of cardiovascular disease was 57% (8).

In the NegHx group, 46% (6) of the participants with a history of diabetes mellitus had a vestibulopathy on the caloric test. Only one of the three participants with a history of diabetes mellitus in the PosHx group also had a vestibulopathy. Diabetes accelerates athersclerosis, which may damage labyrinthine, visual, and somatosensory function. It also causes neuropathy and diabetic retinopathy (Konrad et al, 1999).

Clinical significance of benign paroxysmal positional vertigo and a vestibulopathy
Patients presenting with BPPV and an additional vestibulopathy may have a higher prevalence of ongoing symptoms following treatment than those patients with pure BPPV (Pollak et al, 2002). This would be true regardless of whether the vestibulopathy is on the same side as the BPPV or on the contralateral side. In the current study, 16% (17) of the patients in the NegHx group had a vestibulopathy on the same side as the BPPV. Thirty-seven percent (18) of the patients in the PosHx group had a vestibulopathy on the side with the BPPV. All of the patients in Karlberg et al (2000) had a vestibulopathy and BPPV on the same side. This was a criterion for inclusion in their secondary BPPV group. Five (14%) of the 35 patients with a positive otologic history had a vestibulopathy on the ipsilateral side in the Pollak et al (2002) study. The differences among these studies are likely to relate to different etiologies among the studies.

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). This is extremely important since treatment for BPPV, vestibulopathy, and dysequilibrium has been shown to be highly efficacious when properly administered (Gans & Harrington-Gans, 2002). An underlying vestibular disorder may be the cause of balance problems in these patients (Nadol & Schuknecht, 1989). A vestibulopathy 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). Knowledge of the presence of a vestibulopathy, in addition to BPPV, should lead to a more efficacious outcome for such patients. Consideration of vestibular rehabilitation therapy including canalith repositioning maneuvers, adaptation exercises, balance re-training, and fall prevention in the elderly population should improve, and may lead to more realistic, clinical outcomes in this population.

Conclusions

The purpose of this retrospective study was to determine the prevalence of vestibulopathy in BPPV patients, with and without a prior history of otologic disease. A greater prevalence of vestibulopathy, and on average, a larger average caloric asymmetry was found for the patients with a positive otologic history compared to 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 vestibulopathy, than patients with posterior canal BPPV and a negative otologic history, 2) prevalence rates of 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) prevalence rates of vestibulopathy reported in the literature for posterior canal BPPV, are also likely to be influenced by both the criterion used for clinically significant caloric asymmetry, and also the distribution of otologic diseases and co-morbid factors among the participants, and 4) knowledge about the presence of a co-existing vestibulopathy in addition to the BPPV may enhance clinical outcome even in cases of vestibulopathy contralateral to the ear with the BPPV. Further, knowledge of a vestibulopathy among patients with no prior history of otologic disease should also enhance clinical outcome, and is important to consider given the prevalence in those patients is only 20% less (though statistically significant) than observed for patients with a prior history of otologic disease.

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