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Original Article

# Can Video Head Impulse Testing Be Used to Estimate the Involved Canal in Benign Paroxysmal Positional Vertigo?

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**BACKGROUND:** There may be confusion about which canal is involved in patients with benign paroxysmal positional vertigo (BPPV), especially with those that have subtle findings. The study aimed to determine if video head impulse testing may be used in such patients as a diagnostic tool. Symptom scoring and treatment efficiency in BPPV are essential parts of the process. Therefore, inventories like "Dizziness Handicap Inventory" may be useful in this regard.

**METHODS:** Patients with posterior and lateral canal BPPV were included. Video head impulse testing was performed prior to treatment and 1 week after treatment. Vestibuloocular reflex (VOR) gains were noted and compared to the opposite side. The presence of correction saccades was noted as well. Also, pretreatment and posttreatment Dizziness Handicap Inventory scores were compared.

**RESULTS:** Fifty-seven patients were diagnosed with posterior canal BPPV, and sixteen were with horizontal canal BPPV. In patients with posterior canal BPPV, there was no difference between the involved canal VOR gains and the other canals on the same side ($P = .639$). The involved horizontal canal did not differ from the opposite horizontal canal. Patients with lateral canal BPPV show more significant improvement after treatment compared to patients with posterior canal BPPV.

**CONCLUSION:** Video head impulse testing may not be used to estimate the involved canal in BPPV; however, it may be used to evaluate the efficiency of the treatment, especially in the lateral canal.

**KEYWORDS:** Benign paroxysmal positional vertigo, otology, vertigo, vestibular diseases

## INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo.[1] The underlying cause is generally considered otolith-sticking within the semicircular canals. However, recent research and technological advances show that there may be associated semicircular canal pathology as well. Canalolithiasis and cupulolithiasis are the 2 theories attributed to the pathophysiology of BPPV.[2,3] The movement of otoconia originating from the utricular macula to semicircular canals forms the basic principle of canalolithiasis,[3] while cupulolithiasis explains the interference of the cupula involvement by basophilic debris.[2] Castellucci and colleagues[4] found significantly altered vestibuloocular reflex (VOR) gain in video head impulse testing (vHIT). The semicircular canal pathology may be related to an accumulation of otoconia within the cupula, which results in cupular deflection failure. Moreover, it is said that the involved semicircular canal can also be diagnosed with vHIT.[4] Therefore, vHIT would be an essential part of the diagnostic battery and would ease the diagnosis of some uncertain patients with BPPV.

Other than technological advances, classical methods such as surveys—Dizziness Handicap Inventory (DHI)—going through the course of diagnosis were investigated to predict the diagnosis and to estimate the prognosis. The DHI is widely used in assessing patients with dizziness.[5] Dizziness Handicap Inventory is mainly used before and after the maneuver to compare the effects of the treatment.[6,7] It consists of 3 main scores: physical, emotional, and functional.

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This paper primarily aimed to determine if VOR gain reduction occurred in BPPV patients and also if vHIT could be used as a diagnostic tool to define which canal is involved in BPPV. The secondary aim was to evaluate if DHI could be used to assess the effects of the treatment in BPPV patients.

## MATERIAL AND METHODS

All data used in the manuscript were obtained prospectively. An informed consent form was signed by all patients recruited in the study. Ethical committee of the Izmir University of Economics approval was obtained (Approval No: B.30. 2.İEÜ SB.0. 05.05 -20-2 42, Date: July 11, 2023).

### Patients

Seventy-three patients aged 16–75 were enrolled in this study. Forty-four were female, and 29 were male.The inclusion criteria were the following: patients should never have been diagnosed with vestibular pathology before, should not have spontaneous nystagmus at the time of testing, be diagnosed with BPPV, and be free of trauma before symptoms. All patients were diagnosed with posterior and lateral canal BPPV based on Dix–Hallpike and head roll testing, respectively, using video goggles. Dix–Hallpike testing was applied on all patients (in first examination and 1 week after diagnosis, even in patients with lateral canal BPPV). Head roll testing also was applied on all patients included in the study (even in patients with posterior canal BPPV and patients who had their first attack of BPPV). Apogeotropic nystagmus was not observed in any patient. On the first day (first examinatio n-diagnosis), only 1 correction maneuver was made on all patients. In seventh-day control, a maximum of 2 correction maneuvers were made (in 18 patients, 2 correction maneuvers were needed).

### Video Head Impulse Test

Video head impulse testing was performed before and after the repositioning maneuver (Epley and Barbeque roll) on all patients diagnosed with BPPV on the first visit. Subsequently, provocation maneuvers and vHIT procedures were performed on the seventh day after the first visit. Repositioning maneuvers were repeated if symptoms or positive signs were also observed in provocation maneuvers on the seventh day. If patients were symptom-free, ultimate vHIT was performed. The values of VOR gain and gain asymmetry and also saccades were measured before and after repositioning maneuvers on the first and seventh days (when patients were symptom-free and negative in provocations). The values of affected semicircular canals were compared to the other canals on the same side of the involved canal and each contralateral individual canal.

"Synapsys vHIT Ulmer" was used to perform vHIT and "Maestro" software was used to analyze the data received from hardware.

### MAIN POINTS

- Video head impulse testing is not useful in the diagnosis of posterior canal benign paroxysmal positional vertigo (BPPV).
- It may be more reliable in lateral canal BPPV, although it still shows insignificant results.
- It is not useful in evaluating treatment efficiency, as well. Dizziness Handicap Inventory, however, is useful in the evaluation of success of treatment.

### Dizziness Handicap Inventory

Dizziness Handicap Inventory (Turkish version)[8] was applied to all patients before the first repositioning and 1 week after the first visit, or if patients went further repositioning after the ultimate maneuver. Physical, emotional, functional, and total scores were calculated.

### Statistical Analysis

Statistical analysis was made using Statistical Package for the Social Sciences Statistics software version 22.0 (IBM SPSS Corp.; Armonk, NY, USA). Comparison of the canals as categorical data was made using chi-square test. Normality test was performed using Shapiro–Wilk test, and parametric variables were examined using paired *t*-test, while nonparametric variables were analyzed using Wilcoxon and Mann–Whitney *U* analysis. Pearson correlation was used for the linear relationship in noncategorical variables, and Spearman's ρ analysis in nonparametric data. Also, effect sizes were calculated according to Cohen's *d* analysis and the Wilcoxon *r* test. $P < .05$ was considered statistically significant.

## RESULTS

There were 73 patients aged between 16 and 75. The mean age was 48.76. Fifty-seven patients were diagnosed with posterior canal BPPV, and 16 patients with horizontal canal BPPV. Persistent apogeotropic nystagmus was not observed in any patient (considering that there were no patients with cupulolithiasis).

In patients with posterior canal BPPV, there was no difference between the involved canal VOR gains and the other canals on the same side before and after treatment [pretreatment posterior semicircular canal (post-SCC): 0.9119 (0.65-1.05), SD 0.081 vs. pretreatment ipsilateral semicircular canals (SCCs): 0.9117 (0.38-1.04), SD 0.11; posttreatment post-SCC: 0.9165 (0.6-1.03), SD 0.092 vs. posttreatment ipsilateral SCCs: 0.9114 (0.55-1.02), SD 0.094] ($P = .639$). Also, there was no difference between the involved and the opposite posterior canal in the context of VOR gain [pretreatment: 0.9512 (0.51-1.22), SD 0.1338; posttreatment: 0.9611 (0.37-1.21), SD 0.1233] ($P = .682$). Moreover, when the involved horizontal canal and other canals on the same side were compared, there was no difference in VOR gain [pretreatment lateral semicircular canal (latSCC): 0.8825 (0.76-1.05), SD 0.084 vs. pretreatment ipsilateral SCCs: 0.9048 (0.68-1.02), SD 0.088; posttreatment latSCC: 0.8932 (0.81-1.03), SD 0.073 vs. posttreatment ipsilateral SCCs: 0.9071 (0.66-1.03), SD 0.085] ($P = .715$). The involved horizontal canal did not differ from the opposite horizontal canal as well.

When the involved canals (without dividing canals into lateral and posterior canal groups) were evaluated with vHIT before and after treatment (repositioning maneuver), significant differences between VOR gains were observed (pretreatment allSCC: 0.9024, posttreatment allSCC: 0.9121, $P = .022$), as posttreatment VOR gain means were significantly higher than pretreatment VOR gains. However, any difference between pretreatment and posttreatment VOR gains of the involved posterior canal was not observed (pretreatment: 0.912; posttreatment: 0.917, $P = .210$). In contrast, there were significant differences between horizontal canals (pretreatment: 0.883; posttreatment: 0.893, $P = .024$). Although there was no anterior canal BPPV among patients enrolled in this study, a significant difference was not determined when it was compared at the time points pretreatment and posttreatment. There were near-significant VOR gain

**Table 1.** Pretreatment and Posttreatment Vestibuloocular Reflex Gains

| | Pretreatment | Posttreatment | *P*/Effect Size/Correlation |
|---|---|---|---|
| VOR Gain | | | |
| Posterior canal vs. ipsilateral canals (mean) | 0.9119 (0.65-1.05; SD 0.081) vs. 0.9117 (0.38-1.04; SD 0.11) | 0.9165 (0.6-1.03; SD 0.092) vs. 0.9114 (0.55-1.02; SD 0.094) | .639/ $r=0.079$ |
| Lateral canal vs. ipsilateral canals (mean) | 0.8825 (0.76-1.05; SD 0.084) vs. 0.9048 (0.68-1.02; SD 0.088) | 0.8932 (0.81-1.03; SD 0.073) vs. 0.9071 (0.66-1.03; SD 0.085) | .715 $r=0.173$ |
| Contralateral posterior canal (mean) | 0.9512 (0.51-1.22; SD 0.1338) | 0.9611 (0.37-1.21; SD 0.1233) | .682/ $r=0.039/0.588$ ($P<.01$) |
| Contralateral lateral canal (mean) | 0.9044 (0.76-1.02; SD 0.081) | 0.9306 (0.77-1.01; SD 0.071) | .142/ $r=5\ 02/0.650$ ($P<.01$) |
| Posterior canal (mean) | 0.912 | 0.917 | 0.210/ $d=0.114/0.610$ ($P<.01$) |
| Lateral canal (mean) | 0.883 | 0.893 | 0.024/ $d=0.458/0.878$ ($P<.01$) |
| DHI score | | | |
| Posterior canal BPPV (mean) | 43.088 (8-80) | 18.320 (0-70) | $P<.001/d=-1.145/0.950$ ($P<.01$) |
| Lateral canal BPPV (mean) | 48.125 (14-74) | 19.625 (2-38) | $P<.001/d=-1.04/0.971$ ($P<.01$) |

Vestibuloocular reflex gains of the posterior and lateral canals and average of all canals on the same side and individual posterior and lateral canals are shown. Contralateral posterior and lateral canal vestibuloocular reflex gains are indicated as well. Also, Dizziness Handicap Inventory scores are shown. In the first and second rows vestibuloocular reflex gains of the involved canal compared to ipsilateral canal average, before and after treatment, are shown. The last two rows include Dizziness Handicap Inventory score changes before and after treatment. In comparison of the VOR gains of the involved posterior canal and average VOR gains of the ipsilateral canals Wilcoxon signed-rank test was used (first and second column). In comparison of the VOR gains of the posterior and lateral canal before and after treatment paired *t*-test was used. In addition, comparison of pre, post, DHI scores was made using paired *t*-test as well. Cohen's *d*-test (effect size) was also calculated and indicated under *P*-value as *d*; *d* value smaller than 0.1 was accepted as "small effect," 0.3 as "medium effect," and greater than 0.5 as "large effect." *r* is the effect size in Wilcoxon signed-rank test. Pearson's correlation test was used to analyze posterior and lateral canal changes as well as DHI score changes before and after treatment. Spearman's ρ correlation analysis was used to analyze other values. $P<.05$ was accepted as significant.
BPPV, benign paroxysmal positional vertigo; DHI, Dizziness Handicap Inventory; VOR, vestibuloocular reflex.

improvements before and after treatment (pretreatment vs. posttreatment: 0.9384 vs. 0.9559, $P=.066$) (Table 1).

Corrective saccades were observed in 3 patients (4.1%) before treatment, in whom only 1 patient had posterior canal BPPV, and 2 had lateral canal BPPV. After treatment in 3 patients, covert saccades were still observed. The mean latency of the covert saccades was 103.79 milliseconds, and the mean velocity of the saccades was 164.75 °/s before treatment. After treatment, the mean latency was 104.32 milliseconds, and the mean velocity was 164.89 °/s ($P=.914$). In the posterior canal BPPV, the latency was 104.68 milliseconds and the velocity was 170.04 before treatment, and after treatment the latency was 105.12 milliseconds and the mean was 166.02. In the lateral canal BPPV, mean latency was 103.35 milliseconds and the mean velocity was 159.46 (153.74 -165.18) before treatment, and after treatment the mean latency was 103.93 ($P=.705$) and the mean velocity was 163.75 (150.16-177.34) after treatment ($P<.01$) (Table 2).

The DHI total scores of the patients with posterior canal BPPV pretreatment and posttreatment were also noted. There were significant differences between the scores [pre: 43.09 (8-80), post: 18.32 (0-70), $P<.001$)]. The scores of the patients with lateral canal BPPV also differed before and after [pre: 48.13 (14-74), post: 19.63 (2-38), $P<.001$]. Patients with lateral canal BPPV showed more significant improvement after treatment compared to posterior canal BPPV (Table 1). The correlation between DHI scores and VOR gains before and after treatment was investigated as well. All patients included in the investigation had a significant negative correlation when a comparison was made between pretreatment and posttreatment. The patients with lateral canal BPPV had total scores of correlation smaller than –0.5 (Pearson's correlation coefficient value: –0.73). The patients

**Table 2.** Covert Saccades and Their Latency and Velocity Changes Before and After Treatment

| | | Latency (ms) | *P*/Cohen's *d* | Velocity (°/s) | *P*/Cohen's *d* | VOR gain (mean) |
|---|---|---|---|---|---|---|
| Posterior canal (n = 1) (56 years old) | Pretreatment | 104.68 | – | 170.04 | – | 0.927 |
| | Posttreatment | 105.12 | | 166.02 | | 0.910 |
| Lateral canal (n = 2) (44-61 years old) | Pretreatment | 103.35 (98.45-108.25) | .705/0.091 | 159.46 (153.74- 165.18) | <.01/1.351 | 0.962 (1.01-0.914) |
| | Posttreatment | 103.93 (98.26-109.60) | | 163.75 (150.16- 177.34) | | 0.985 (1.018-0.951) |
| Total (n = 3) | Pretreatment | 103.79 | .352/0.169 | 164.75 | .914/0.072 | 0.945 |
| | Posttreatment | 104.32 | | 164.89 | | 0.959 |

All values are analyzed by *t*-test. Cohen's d test (effect size) was also calculated and indicated under *P*-value as *d*; *d* value smaller than 0.1 was accepted as "small effect," 0.3 as "medium effect," and greater than 0.5 as "large effect." $P<.05$ was accepted as significant.
ms, milliseconds; VOR, vestibuloocular reflex.

with posterior canal BPPV also had scores smaller than −0.5 (−0.61); however, the correlation was weaker compared to lateral canal BPPV patients.

## DISCUSSION

Since BPPV is the most common cause of vertigo, numerous investigations have been conducted to explain its pathophysiology and ease the diagnostic processes. Mistakenly, it seems very easy to be diagnosed. Most of the patients with BPPV were diagnosed with classical maneuvers such as Dix–Hallpike. However, remainings make a significant part of the stringent diagnostic process. In such patients, use of classical maneuvers could not get the clinician to any point, moreover, inaccurate diagnostic guidance could be the reason for the overtreatment and overdiagnosis. Dix and Hallpike and other maneuvers are essential tools on the way to the diagnosis, however, more objective and reliable diagnostic tools, such as vHIT, may overcome diagnostic difficulty, especially in patients with resistant BPPV. Also, follow-up is the other part of the BPPV treatment, as the need for recurrent maneuvers is a reality in this group of patients. Moreover, in Dix–Hallpike maneuver there should be evident nystagmus to diagnose BPPV. However, numerous patients have mild vertigo and hardly visible nystagmus on the examination, especially without frenzel goggles. Video head impulse testing seems to be a more accurate and standardized method in the diagnosis of BPPV.

It has been considered that there is no canal pathology in BPPV. Otoliths are stuck within the canals, although they should be present in the utricle and saccule. In contrast, cupulolithiasis is the other theory explaining BPPV that otoliths are stuck within the cupula rather than the canals.[2] The long-lasting and treatment-resistant BPPV are considered that they may have cupulolithiasis pathology.[4] In such patients with BPPV, it may be tough to determine which canal was involved due to cupulolithiasis. Therefore, objective, reliable diagnostic tools that are easily performed are necessary. tool to be easily performed and accurate.

There are a large number of trials to find the best tool to overcome confusion in BPPV diagnosis. Elsherif and colleagues[9] conducted a meta-analysis. Although it was demonstrated that vHIT might be used as a diagnostic tool in determining which canal was involved, the number of trials and the number of patients included in the study (5 trials and 168 patients were included in this study) were unsatisfactory. In contrast, Salturk and Yetiser[10] investigated 60 patients and found no altered VOR gain in vHIT in the involved side. Furthermore, Qiongfeng and colleagues[11] compared patients with the control group with vestibular neuritis and BPPV. They found that patients with BPPV had lower gains than the control group, but the difference was insignificant. In our study, VOR gain in the involved semicircular canal was compared to the contralateral normal paired semicircular canal, as well as the ipsilateral average of all canals. When the VOR gains were calculated and compared to the average of all canals on the involved side, we did not observe any difference when lateral and posterior canals were evaluated individually. When involved canals were compared to the contralateral same canal, there was also no significant difference between VOR gains; in the posterior canal BPPV, involved posterior canal VOR gains were 0.9119 (0.65-1.05, SD 0.081), and the contralateral posterior canal VOR gains were 0.9512 (0.51-1.22, SD 0.1338) ($P = .152$), whereas involved lateral canal VOR gains were 0.8825 (0.76-1.05, SD 0.084), and the contralateral lateral canal VOR gains were 0.9044 (0.76-1.02, SD 0.081) ($P = .407$). In contrast to Castellucci,[4] we did not find any alteration in the involved canal in BPPV patients, Also, VOR gains of the canals other than the pathologic canal on the involved side were also compared before and after treatment, however, their study included patients with persistent vertigo that indicated cupulolithiasis. Although it is considered that canalolithiasis does not lead to canal pathology, cupulolithiasis may impact the vHIT VOR gains due to high-velocity cupula movement alteration.[12] In our study, we did not observe any persistent apogeotropic nystagmus, so-called cupulolithiasis; therefore, we could assess if there was canal pathology in involved canals other than cupulolithiasis. Our findings suggest vHIT may be utilized to determine the involved canal in persistent BPPV (may be in only cupulolithiasis) that does not resolve despite recurrent repositioning maneuvers.

Although vHIT may not be used as a diagnostic tool to elicit which canal is involved, the impact of the repositioning maneuver (Epley and Barbeque roll maneuver) may be evaluated using vHIT. Cinar and colleagues[13] investigated 44 patients with 24 having isolated posterior semicircular canal BPPV. Their study compared pretreatment and posttreatment VOR gains and found insignificant vHIT VOR gains. Califano and colleagues[6] also evaluated 150 patients before and after treatment with first-time BPPV. No significant VOR gains were observed on vHIT in the subject of the posterior canal. They also found no difference between lateral and anterior canals. In their study, Karababa et al[14] stated similar results that vHIT VOR gains did not show any improvement; therefore, vHIT would not be used in determining the effectiveness of the repositioning maneuver. However, in our study, the average of the VOR gains in all canals on the involved side significantly improved after the repositioning maneuver. Nevertheless, there was no significant improvement when posterior canals were taken into the subject solely. Namely, patients with lateral canal BPPV had a marked increase in VOR gain. Significant improvement of the VOR gains after treatment in lateral canal BPPV is an interesting finding because 2 patients had lateral canal BPPV (12.5% of patients with lateral canal BPPV and 1.7% in posterior canal BPPV) with corrective saccades that indicated vestibular/canal pathology. It might indicate that canal pathology is the possible cause of BPPV, at least in lateral canal BPPV and treatment-resistant patients. According to our knowledge, there are no studies in the literature evaluating BPPV from this perspective. However, more investigations with larger and more specific groups of patients should be made to clearly define this situation. Although there were no patients with anterior canal BPPV in our study, anterior canal VOR gains also showed improvement, although insignificant as the *P*-value indicates ($P = .066$, pretreatment gain: 0.9384, posttreatment gain: 0.9559, $d = 0.837$). Anterior canal gain recovery may be due to having the common crus with the posterior canal. Also, significant improvement in the DHI scores may partially be the result of this finding. There is 1 study[6] that evaluates VOR gain changes before and after treatment. Insignificant change was found in that paper. Although in our study anterior canal has insignificant improvement according to the *P*-value, effect size shows there is clinically significant change is present.

The other interesting point of this investigation is that there are 3 patients with covert saccades. It may be an indication of canal

pathology. After treatment covert saccades continued to occur on vHIT in all patients. When we look at the latency and velocity changes before and after treatment including all canals, there were some changes. The mean latency in the posterior and lateral canal BPPV seems to be increased after treatment. In vestibular pathology, we expect latency duration to be decreased after treatment; however, in our study, latency period got longer compared to the pretreatment values. Although the presence of pretreatment saccades indicates canal pathology or vestibular pathology other than canal pathology in BPPV patients, insignificant changes after treatment might show an association with other vestibular pathologies rather than canal pathology. However, all VOR gains were in the normal range, though Fernandez and colleagues[15] suggested VOR gains might be normal in patients with unilateral vestibulopathy. Therefore our findings may still suggest canal pathology. The number of patients is very low to indicate accurate results. Moreover, 1 week may be insufficient for canal pathology to be resolved in BPPV. There is a lack of data investigating saccades in BPPV. Therefore, more investigations should be conducted to clearly define saccades in BPPV.

Unfortunately, it seems vHIT may not be used as an "involved canal detector"; instead, it may be used in the follow-up period to assess the impact of the repositioning maneuver, especially in lateral canal BPPV.

It is tough to determine the medical, surgical, and rehabilitative management effects of the patient with vertigo.[16] Researchers try to find the best tool in this way. Vestibular Disorders of Daily Living Scale, Vertigo Handicap Questionnaire, and DHI are all used for this purpose. Dizziness Handicap Inventory has been investigated pretty much more than the rest. It is widely used in assessing patients with dizziness.[5] It consists of 25 items to evaluate the perceived handicapping effects imposed by vestibular system disorders. The BPPV symptom scoring with DHI has also been investigated in numerous studies. Zamyslowska-Szmytke and colleagues[5] subcategorized the DHI into subscales based on the original DHI. F3 (positional) subscale has been found sensitive and specific to BPPV in their research. In another study, Wang[17] found that DHI total scores decreased after repositioning. Therefore, the author indicated that DHI total scores could be used in assessing the effectiveness of the treatment. In our trial, we used DHI adapted to the Turkish language to show the reliability of VOR gain changes before and after treatment. Improvement in DHI scores was more prominent in the lateral canal. Although DHI scores showed evident improvement in the posterior canal, there were insignificant changes in VOR gains. As indicated before, it may be the result of improvement in uninvolved canals on the same side or central adaptation.

The bigger part of the investigations indicates vHIT may be used to estimate the involved canal in BPPV patients; however, our research found that there is limited use of the vHIT in the context of prediction of the involved canal. The presence of the saccades in BPPV may show the associating vestibular pathology, rather than canal pathology. Even so, in the lateral canal, BPPV vHIT may indicate the involved canal. The VOR gain improvements in uninvolved canals on the same side are interesting and worth conducting more research. Moreover, DHI is a useful tool to assess treatment success as found in other investigations.

Consequently, deciding which canal to be involved in BPPV may be difficult. Objective evaluations like vHIT would be of great importance in diagnosis. Unfortunately, there is no reliable assessment to find the involved canal, even using vHIT. Nonetheless, as in our research, VOR scores obtained from vHIT may be used in lateral canal and resistant BPPV to assess the effect of the treatment, although they cannot be used in posterior canal BPPV. Dizziness Handicap Inventory total scores can be used to evaluate the impact of the treatment as well.

### Limitations of the Study

The main limitation of the study is the lack of a control group, although the involved canal could be compared with the opposite and same-side uninvolved canals. There were correction saccades in only 3 patients. The results are interesting. However, a small patient group with saccades limited the results. Also, there were no patients with cupulolithiasis.

**Ethics Committee Approval:** This study was approved by the Ethics Committee of IzmirUniversity of Economics (Approval No: B.30.2.İEÜSB.0.05.05-20-242, Date: July 11, 2023).

**Informed Consent:** Informed consent was obtained from the patients who agreed to take part in the study.

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