Chronic symptoms after vestibular neuritis and the high velocity vestibulo-ocular reflex

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Abstract

Hypothesis: As the anterior and posterior semicircular canals are vital to the regulation of gaze stability, particularly during locomotion or vehicular travel, we tested whether the high velocity vestibulo-ocular reflex (VOR) of the three ipsilesional semicircular canals elicited by the modified Head Impulse Test would correlate with subjective dizziness or vertigo scores after vestibular neuritis (VN).

Background: Recovery following acute VN varies with around half reporting persistent symptoms long after the acute episode. However, an unanswered question is whether chronic symptoms are associated with impairment of the high velocity VOR of the anterior or posterior canals.

Methods: Twenty patients who had experienced an acute episode of VN at least three months earlier were included in this study. Participants were assessed with the video head impulse test (vHIT) of all six canals, bithermal caloric irrigation, the Dizziness Handicap Inventory (DHI) and the Vertigo Symptoms Scale short-form (VSS).

Results: Of these 20 patients, 12 felt that they had recovered from the initial episode whereas 8 did not and reported elevated DHI and VSS scores. However, we found no correlation between DHI or VSS scores and the ipsilesional single or combined vHIT gain, vHIT gain asymmetry or caloric paresis. The high velocity VOR was not different between patients who felt they had recovered and patients who felt they had not.

Conclusions: Our findings suggest that chronic symptoms of dizziness following VN are not associated with the high velocity VOR of the single or combined ipsilesional horizontal, anterior or posterior semicircular canals.

Keywords: Vestibular; vestibular neuritis; dizziness; vertigo, head-impulse test

Introduction

Vestibular neuritis (VN) is an acute disorder characterised by vertigo, nausea, vomiting and imbalance following sudden unilateral loss of peripheral vestibular function (1). Recovery is through peripheral and central vestibular compensation (2). Typically, symptoms last days or weeks but around 50% of patients experience chronic dizziness, unsteadiness and spatial disorientation (3,4).

It has been postulated that persistent peripheral vestibular loss could account for these chronic symptoms (5). The standard measure of peripheral vestibular loss is the gain of the vestibulo-ocular reflex (VOR) which is the ratio of the size of slow phase corrective eye movement to the size of head movement (peak slow phase eye velocity / peak head velocity). The VOR maintains gaze stability and preserves visual acuity during head movements. Impairment can cause visual blurring during head motion (6), which could be interpreted by the patient as dizziness, unsteadiness or spatial disorientation. Thus, a central question regarding the process of symptom recovery is whether this is related to a dysfunctional VOR.

Previous studies have shown that the low velocity VOR response from the caloric test does not predict chronic symptoms of dizziness or vertigo (3,7,8). However, recent advances have led to the development of a bedside clinical head thrust or impulse test (HIT) measuring the high velocity VOR of all six semicircular canals (9). The high velocity VOR elicited by the HIT recovers more slowly following acute VN compared to the low velocity VOR elicited by caloric irrigation (10-12), and may thus better reflect clinical outcome.

Interestingly, Palla and colleagues (13) have shown that there is no relationship between the high velocity horizontal canal VOR gain and chronic symptoms following VN. However, as the anterior and posterior semicircular canals are vital to the regulation of gaze stability (14), particularly during locomotion or vehicular travel, we posed the question of whether the high velocity VOR gain of the three ipsilesional semicircular canals (elicited by the modified HIT (9))

would predict subjective dizziness or vertigo scores after VN. The low velocity VOR of the horizontal canal (elicited by caloric irrigation) was measured for comparison.

Materials and Methods

Twenty patients (7 male, 31-87 years (mean 57.3 +/- 18) with clinical histories, physical examinations and function tests typical of acute VN were recruited i.e, horizontal nystagmus, clinically abnormal head-impulse test and a significant canal paresis. Of our patients, none had inferior vestibular neuritis. The exclusion criteria were patients with no current indications of overlapping vestibular migraine. For this study, all patients were tested in the chronic stage of VN (3-36 months after acute VN onset; mean 9.8 +/- 7.5), including a repeat caloric test. Informed consent was obtained from all subjects.

Vestibular assessment

Six-canal vHIT: Eye and head movements were simultaneously recorded using the ICS video Head Impulse system (vHIT, GN Otometrics, Denmark). The system consists of a pair of light-weight goggles containing 3-D gyroscopes to measure head velocity, and a small mounted video camera to record eye position. The video camera is mounted within the right eye-frame of the goggles, which were secured firmly to the subject's head with an adjustable elastic strap.

The patient was instructed to fixate on a target positioned approximately 1.5 metres in front of them. The examiner, while holding the patient's head from behind, then made a series of brisk head movements (10–20° amplitude) corresponding to the horizontal, left anterior-right posterior (LARP) and right anterior-left posterior (RALP) canal planes (15). In contrast to early papers measuring VOR responses along the LARP and RALP planes (16), with the vHIT

technique the head must be turned in yaw by approximately 40-45° so that the head impulse delivered only (or mostly) elicits vertical VOR movements.

Eye and head velocities were sampled at 250 Hz and the ratio of eye-to-head peak velocity (VOR gain) was calculated for each semicircular canal from an average of 20 head impulses performed over a range of velocities (50–300°/s) (17). Asymmetry between the ipsilesional and contralesional canals was also calculated and expressed as a percentage (18).

In addition to the single canal gain values and asymmetry values generated automatically by the vHIT program, we calculated a total gain for each side:

$\sqrt{(horizontal\ canal\ gain)^2 + (anterior\ canal\ gain)^2 + (posterior\ canal\ gain)^2}/3,$ and total right/left asymmetry (%). As previous studies have reported no correlation between horizontal canal vHIT gain or asymmetry with long-term recovery, we also focussed on the vertical canals and calculated a vertical canal gain $\sqrt{(anterior\ canal\ gain)^2 + (posterior\ canal\ gain)^2}/2$, and vertical canal right/left asymmetry (%). These formulae provide overall values for the contributions from each canal.

Caloric test: Bithermal caloric irrigations (30 & 44°C) were performed (ICS CHARTR, GN Otometrics, Denmark)and the degree of canal paresis was calculated using Jongkees formula and expressed as a percentage as previous studies (18).

Symptoms questionnaires

In parallel, symptoms during the past month were scored with the Dizziness Handicap Inventory (DHI) (19) and the Vertigo Symptoms Scale short form (VSS) (20). We also asked each patient whether they felt they had recovered from the acute episode or not.

-Table 1 about here-

Pearson Correlation Coefficient analyses were employed between all measures. Independent samples t-tests were used as confirmation. Linear regression was used to test whether vHIT gains predict DHI or VSS scores. P-values were corrected for multiple comparisons.

Results

As shown in Table 1, eight patients felt that they had not fully recovered from the acute episode. These patients also had the highest DHI and VSS scores (paired t-test $P<0.002$). There was a strong significant correlation between DHI and VSS across the group of 20 VN patients ($P<0.001$, Pearson Correlation Coefficient=0.857). There was no correlation between caloric paresis and DHI score (Pearson correlation coefficient = -0.134, P=0.57) or between caloric paresis and VSS score (Pearson correlation coefficient = -0.076, P=0.572). There was also no correlation between caloric paresis and horizontal canal vHIT gain asymmetry (Pearson correlation coefficient = 0.176, P=0.458).

With linear regression, the adjusted R-square was 0.02 for DHI scores and 0.084 for VSS scores. The regression was not significant for either DHI scores (F[0.47], P=0.82) or VSS scores (F[1.29], P=0.327). Similarly, stepwise linear regression identified no predicting independent variables in the analysis (no variables were entered into the analysis for either DHI or VSS).

As shown in Figure 1 A-F, there was no correlation between the ipsilesional vHIT gains for the horizontal, anterior and posterior canals and vHIT gain asymmetry for the horizontal, anterior and posterior canals versus DHI score.

As shown in Figure 2 A-F, there was also no correlation between the ipsilesional vHIT gains for the horizontal, anterior and posterior canals and vHIT gain asymmetry for the horizontal, anterior and posterior canals versus VSS score.

We also compared vHIT gains and vHIT gain asymmetries for the horizontal, anterior and posterior canals between the 8 patients who felt they had not recovered and the 12 patients who felt they had recovered. Independent samples t-tests showed no difference between these groups (P=0.26-0.92).

-Figure 1 about here-

-Figure 2 about here-

We also investigated the relationship between vHIT response and recovery by grouping the vHIT single canal gains into the mean sum of the canal vectors to give a single gain value for the ipsilesional and contralesional sides. We also grouped the ipsilesional semicircular canals into a single value for the anterior and posterior (vertical) canals gain and asymmetry, as described in Methods.

We found no significant correlation between the vector sum of the three ipsilesional canal gains (horizontal + anterior + posterior) and DHI scores (Pearson correlation coefficient =-0.124, P=0.60) or VSS scores (Pearson correlation coefficient =-0.302, P=0.196). Asymmetry did not correlate to DHI (P=0.55) or VSS scores (P=0.13) as shown in Figures 3A and 3B.

In addition, there was no significant correlation between the vector sum of the vertical canals (anterior + posterior) and DHI scores (Pearson correlation coefficient = -0.125, P=0.60) or VSS scores (Pearson correlation coefficient=-0.152, P=0.15). Asymmetry did not correlate with DHI (P=0.77) or VSS scores (P=0.10) as shown in Figures 3C and 3D.

Neither total nor vertical canal gain and asymmetry values were significant predictors of DHI or VSS scores with multiple regression analysis, i.e., no values were entered into the analysis during stepwise regression.

-Figure 3 about here-

Discussion

Here, we find no evidence to support the hypothesis that chronic symptoms of dizziness or vertigo following acute VN are associated with the high velocity VOR of the three ipsilesional semicircular canals. There was no correlation between ipsilesional high velocity VOR gain or gain asymmetry of the single or combined horizontal, anterior and posterior canals measured with the vHIT and DHI or VSS scores. Patient 4 is a representative example: this individual was asymptomatic (DHI=0) but had an ipsilesional posterior canal gain of 0.33. In contrast, patient 20 who was the most symptomatic individual (DHI=70) had normal vHIT gains for each of the canals (above 0.78).

Also, as in previous studies, there was no correlation between caloric paresis and chronic symptoms after VN (21,22) or between caloric paresis and horizontal canal HIT asymmetry (11) probably reflecting the different frequency ranges of these tests.

There is little doubt that acute VN triggered the patients' chronic symptoms, however residual semicircular canal deficits might not be a crucial factor. As the otoliths are involved in the translational VOR (tVOR) (23), it is possible that impaired otolith function could explain chronic symptoms in some patients. Utricular function is typically affected in VN as measured with ocular VEMP (oVEMP) (24). In a one-year follow-up study in VN patients, Magliulo and colleagues (25) found that four out of five patients with chronic symptoms, had absent ipsilesional oVEMP responses. Saccular function is impaired when the inferior branch of the vestibular nerve is affected. However, it is unlikely that otolith damage would be the critical variable predicting long term outcome in VN given that even patients with vestibular neurectomy recover well (26).

Another explanation is that the relative weightings of vestibular, visual and somatosensory signals change following unilateral vestibular loss. Indeed, we have found that chronic symptoms after VN may relate to increased visual dependence (3). Psychological (22,27) and spatial orientation factors (28), also have a strong influence on long term outcome.

The sample size used in this study (n=20) is also a potential limitation, but if this were the case it would imply that the relationship between clinical outcome and vHIT gains is very weak and therefore unlikely to be sensitive enough to be of practical use in a clinical environment. Using mean and standard deviation data from our strongest correlation coefficient (Figure 2E, anterior canal gain vs VSS) we calculated that subjects recruited would need to equal n=58 to achieve $P<0.05$ (Power= 0.8) before correction for multiple comparisons.

To conclude, chronic symptoms of dizziness or vertigo following acute VN were not related to the high velocity VOR of the horizontal, anterior or posterior semicircular canals. It is likely that clinical recovery and outcome depends mostly on central compensation, including higher level processing in the brain.

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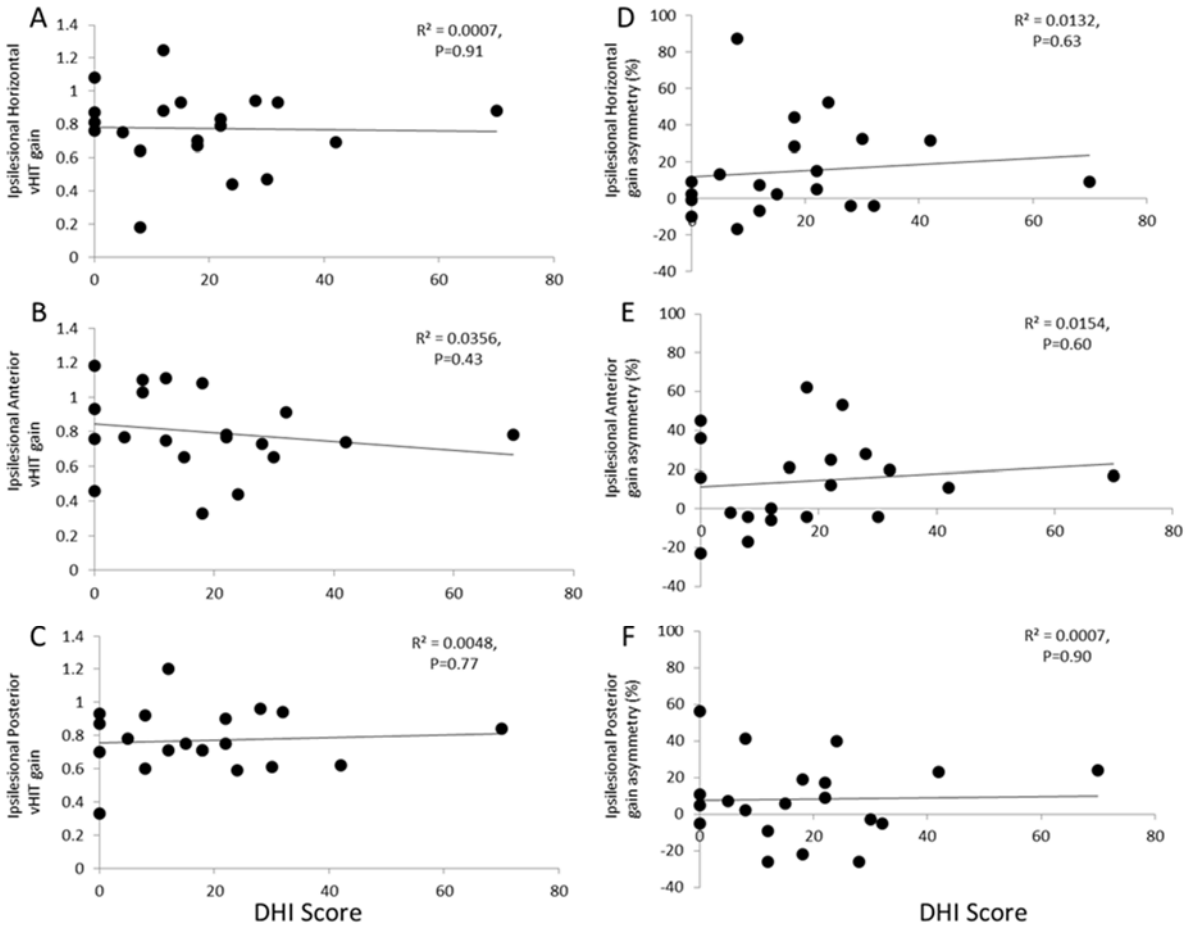

Figure 1. vHIT VOR gains for the ipsilesional A). Horizontal, B). Anterior and C). Posterior canals and vHIT gain asymmetry for the A). Horizontal, B). Anterior and C). Posterior canals versus DHI score. vHIT assessment did not correlate with DHI score.

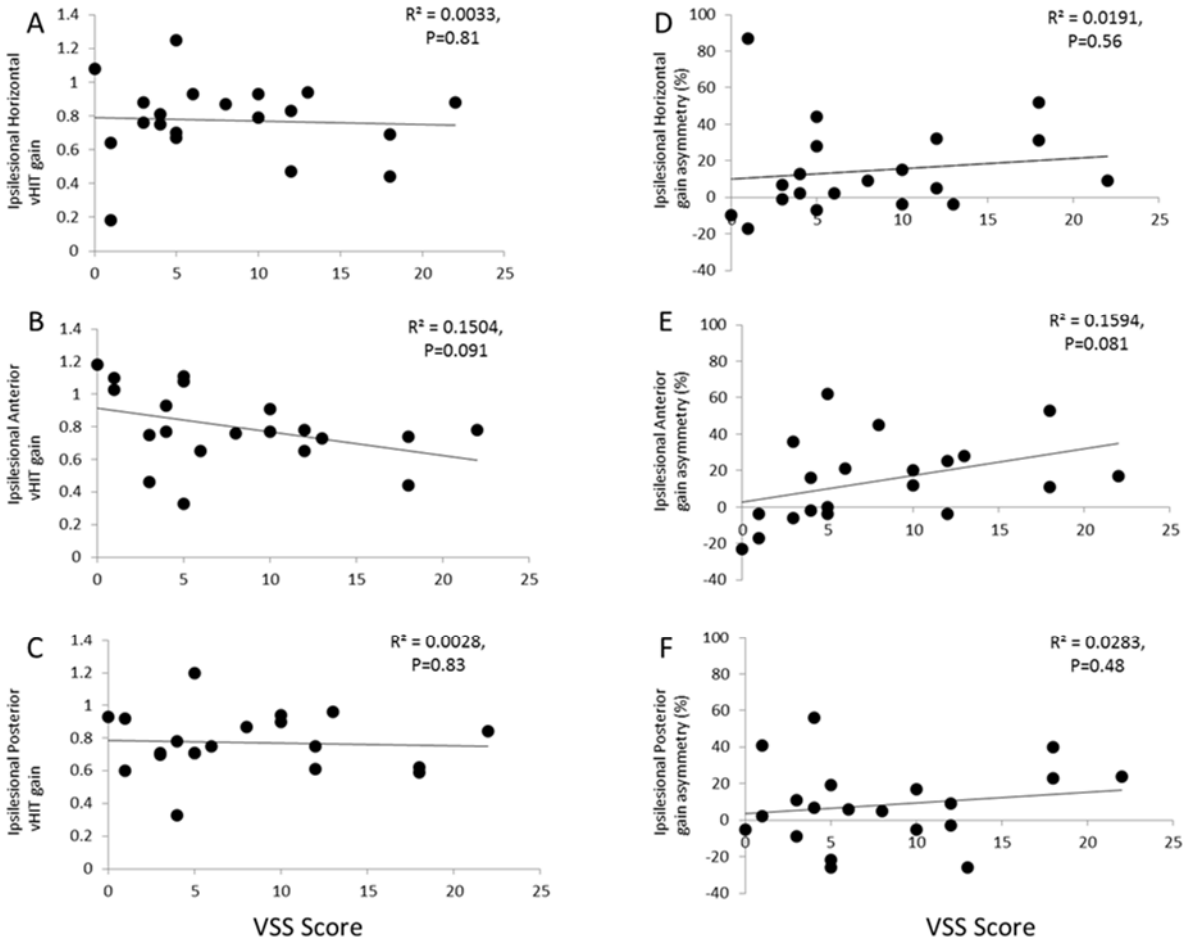

Figure 2. vHIT VOR gains for the ipsilesional A). Horizontal, B). Anterior and C). Posterior canals and vHIT gain asymmetry for the A). Horizontal, B). Anterior and C). Posterior canals versus VSS score. vHIT assessment did not correlate with VSS score.

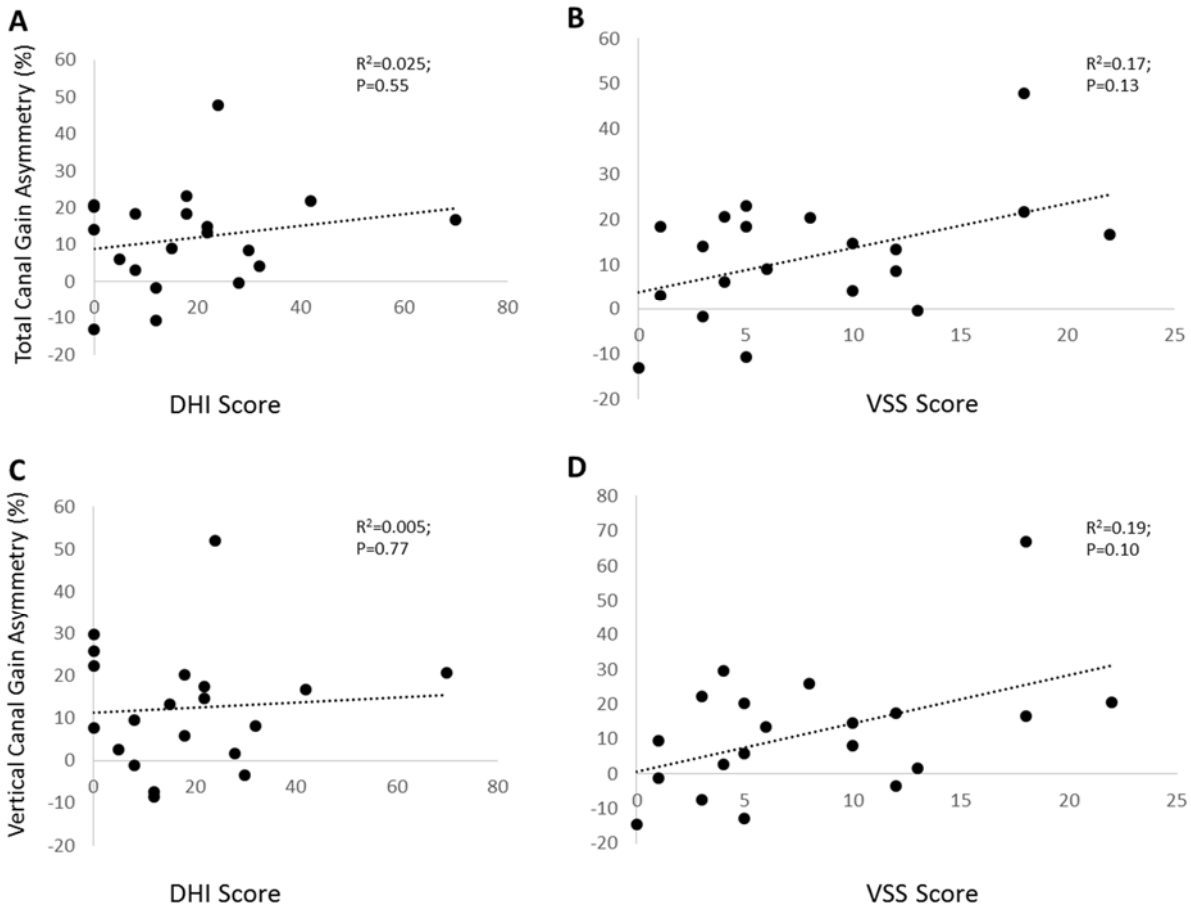

Figure 3: vHIT VOR gain for the total response versus DHI score (A) and VSS score (B) & vHIT VOR gain for the vertical canal (anterior + posterior canals) response versus DHI score (C) and VSS score (D).

Tables

Patient	Duration (months)	Age	Caloric Paresis (%)	Ipsilesional horizontal canal vHIT gain	Horizontal vHIT gain Asymmetry (%)	Ipsilesional anterior canal vHIT gain	Anterior vHIT gain Asymmetry (%)	Ipsilesional posterior canal vHIT gain	Posterior vHIT gain Asymmetry (%)	DHI score (/100)	VSS score (/60)	Subjectively Recovered (YES/NO)
1	12	44	62	0.44	52	0.44	53	0.61	40	24	18	No
2	4	75	-12	0.79	15	0.77	12	0.9	17	22	10	No
3	12	61	75	0.69	31	0.74	11	0.62	23	42	18	No
4	6	47	10	0.81	2	0.93	16	0.33	56	0	4	
5	36	75	28	0.75	13	0.77	-2	0.78	7	5	4	
6	6	54	0	1.25	-7	1.11	0	1.2	-1	12	5	
7	6	48	64	0.83	5	0.78	25	0.75	9	22	12	No
8	4	54	41	0.47	32	0.65	-4	0.61	-3	30	12	No
9	6	52	22	0.76	-1	0.46	36	0.7	11	0	3	
10	8	32	17	0.93	-4	0.91	20	0.94	-5	32	10	No
11	12	33	5	0.94	-4	0.73	28	0.96	-14	28	13	No
12	8	71	100	0.64	-12	1.03	-10	0.6	41	8	1	
13	7	78	100	0.67	28	0.33	62	0.71	-1	18	5	
14	3	70	62	0.88	7	0.75	-6	0.73	-9	12	3	
15	15	87	0	0.7	44	1.08	-4	0.71	19	18	5	
16	5	31	51	0.18	87	1.1	-4	0.92	2	8	1	
17	12	68	18	0.93	2	0.65	21	0.75	6	15	6	
18	18	78	26	1.08	-6	1.18	-8	0.93	-5	0	0	
19	7	31	68	0.87	9	0.76	45	0.87	5	0	8	
20	24	62	3	0.88	9	0.78	17	0.84	24	70	22	No

Table 1. Vestibular testing data and symptom scores from the patients who participated in this study (n=20).