The Journal of International Advanced Otology

J Int Adv Otol 2021; 17(2): 103–108 • DOI: 10.5152/JIAO.2021.8572

Original Article

Video Head Impulse Test (vHIT) Findings in Patients With Superior Semicircular Canal Dehiscence: A Case–Control Study

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Cite this article as: Tikka T, Mohd Slim MA, Gaggini M, Kontorinis G. Video head impulse test (vHIT) findings in patients with superior semicircular canal dehiscence: A case–control study. *J Int Adv Otol.* 2021; 17(2): 103-108.

OBJECTIVE: To explore the usefulness of the responses of video head impulse testing (vHIT) in assessing symptomatic patients with superior semicircular canal dehiscence (SSCD).

METHODS: This was a prospective case–control study performed in a tertiary skull base referral Centre in the UK. It included all patients diagnosed with SSCD from January 2015 to January 2019 and compared to a control group of age and gender-matched unaffected individuals. The main outcome of the study was semicircular canal vestibule-ocular reflex (VOR) gains during vHIT assessment and link to patients' symptoms.

RESULTS: A total of 28 patients were diagnosed with SSCD during the study period and completed the vHIT assessment. Reduced VOR gains (<0.8) were noted in 57% of patients ($n = 16$). Half of these ($n = 8$) were in canals other than the superior semicircular (posterior only: $n = 5$; lateral and posterior: $n = 3$). Three patients (10.7%) had abnormal responses in 2 canals. The canals in the contralateral side were affected in 56.5% of the cases. There was no correlation with the patients' symptoms. Results were directly comparable with the control group vHIT results with no identifiable statistically significant differences on comparison of the ipsilateral SSCD side with a randomly selected side from the control group (all comparisons: $P > .05$).

CONCLUSION: SSCD can affect the vestibular responses from all 3 semicircular canals; not necessarily the superior one. Similar responses were found in a control group of normal subjects. Although the use of vHIT in the assessment of SSCD is not diagnosis-specific, it can still help with identifying the impact of surgery on all canals prior to any intervention in order to avoid bilateral vestibular failure.

KEYWORDS: Superior semicircular canal dehiscence, head impulse test, vestibulo-ocular reflex

INTRODUCTION

The superior semicircular canal dehiscence (SSCD) was first described by Minor et al. in 1998. It is a condition constituted by the presence of vertigo and/or oscillopsia induced by middle ear pressure changes or exposure to loud noises, due to dehiscence in the superior semicircular canal (SSC).[1] Presence of chronic disequilibrium has also been described as well as intermittent tinnitus, low-frequency conductive hearing loss and autophony, hypersensitivity to own body sounds, and hyperacusis.[2,3]

The clinical diagnosis can be confirmed by cross-sectional imaging supplemented with vestibular-audiological testing. On imaging, a high-resolution coronal computed tomography scan (CT) of the temporal bone will identify the bony dehiscence in the SSC. Vestibular-audiological investigations will reveal the presence of low threshold, abnormally large, sound-induced cervical vestibular evoked myogenic potentials (c-VEMPs) and vertical or upwards torsional evoked eye movements induced by sound or pressure.[3-5] Vertical vestibulo-ocular reflex (VOR) eye movements are visible in patients exposed to loud clicks (click-evoked VOR) being at least 10 times greater than the response in normal individuals, suggesting superior canal receptor hypersensitivity to sound.[6,7] Six canal video head impulse testing (vHIT) can give information on VOR in response to head movements without the addition of other stimuli.[8] In SSCD patients, as well as in other patients with vestibulopathies, vHIT can provide an assessment of vestibular function in the natural range of daily motions and an appreciation of how the presence of SSCD affects SSC function, hence peripheral balance coordination in the absence of the external stimulus.

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Received: August 6, 2020 • **Accepted:** August 9, 2020 • Available Online Date: X XX 2021

Available online at www.advancedotology.org

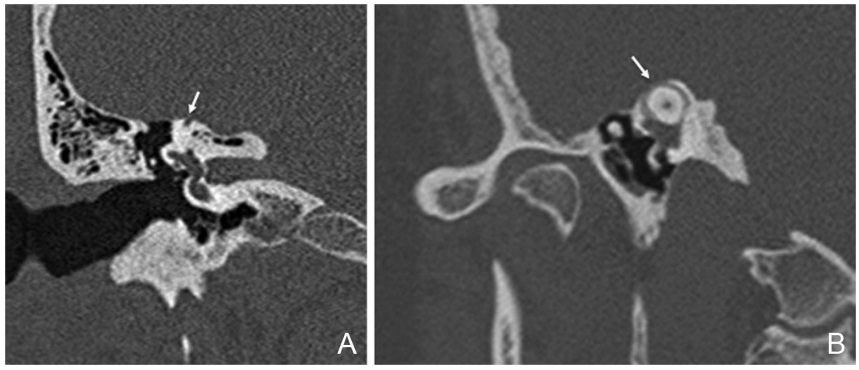

Figure 1. High-resolution temporal bone CT scan showing an SSCD (arrow) on the coronal plane (A) and sagittal oblique (B).

Only a few studies with a limited number of subjects have been identified looking specifically at vHIT responses in SSCD prior to surgical repair.[9] Moreover, despite the good number of studies reporting surgical outcomes following plugging of SSCD, there is no robust evidence to describe non-surgical rehabilitation of patients who are not good candidates for surgical repair or do not wish surgical intervention.[10] vHIT assessment could be critical in appreciating how SSCD affects vestibular function during daily activities hence inform rehabilitation strategies for the conservative management of SSCD.

The aim of this study was to prospectively record 6-canal vHIT findings on newly diagnosed symptomatic SSCD patients and compare the results with unaffected individuals in order to assess how the dehiscence affects the function of all SSCs.

MATERIALS AND METHODS

Study Settings and Patient Characteristics

This is a prospective case–control study conducted in a tertiary teaching hospital. The NHS health research online tool was used to sought UK research ethics committee advice. Ethics committee approval was not required as the data collected for this study were part of the routine assessment of individuals with SSCD and atypical dizziness that comprised the control group. Therefore the study was approved by the Local Audit Committee.

A total of 31 patients with SSCD were prospectively recruited from January 2015 to January 2019. Patients were symptomatic at the time of diagnosis with the presence of at least autophony or vertigo on exposure to loud noise or pressure changes that led to the clinical diagnosis of dehiscence of the SSC. All patients underwent a high-resolution computed tomography scan (CT; thickness 05-0.625 mm) of the temporal bone with confirmation of SSCD (Figure 1). Peripheral vestibular assessment as part of the investigation process was arranged for all patients at the time of diagnosis including cervical vestibular evoked myogenic potential (cVEMP) testing and vHIT assessment. Twenty-eight patients completed a 6-canal vHIT assessment during the study period and were included in the study.

The control group was compromised with 28 age- and gender-matched subjects seen in an otology clinic and assessed for generic otology symptoms. These patients described atypical dizziness during the consultation; thus, they underwent thorough clinical, audiological and vestibular assessment, which excluded a peripheral, vestibular cause of their dizziness. Additionally, they had no history of chronic ear disease or ear surgery or cervical spine/neck fixation hence they were considered healthy individuals.

Examined Factors

All patients underwent 6-canal-vHIT assessments in standardized settings. Six-canal vHIT were performed by experienced, subspecialised audiologists using the Otometrics© 6-canal vHIT device and software (Otometrics, Taastrup, Denmark) in a targeted velocity of >200 degrees/second to ensure accurate, reproducible results. A VOR gain between 0.8 and 1.2 was considered normal as per the previous study.[8] We additionally analyzed the raw data for each semicircular canal with normal gain documenting the presence of covert or/and overt saccades. Findings were correlated with patients' presenting symptoms and demographics.

Data Analysis

vHIT outputs were treated as both categorical ($\geq$0.8, <0.8) and continuous variables and analyzed using the chi-square and Student's *t*-test respectively, following assessment confirming normality. The SPSS 23 (IBM Corp, Armonk, NY) was used for the statistical analysis.

RESULTS

SSCD Demographics and Presentation

Of the 31 patients diagnosed with SSCD over the 4-year period of this study, 28 patients completed a 6-canal vHIT assessment (33 ears positive for SSCD). The commonest presentation was autophony (64%). A breakdown of symptoms is available in Table 1. The majority of patients were females (60.7%, $n = 17$). The mean age was 47 years, ranging from 28 to 78 years (SD = 12.8). Bilateral dehiscence was found in 5 subjects (17.9%), the right ear was affected in 14 patients

Table 1. SSCD Patients Presenting Complains

Presenting Symptom	Number (frequency)
Autophony	16 (64%)
Feeling of blocked ear	8 (28.6%)
Dizzy on pressure change	8 (28.6%)
Hypersensitivity to own body sounds	8 (28.6%)
Dizzy on loud sound	8 (28.6%)
Non-specific dizziness	7 (25%)
Tinnitus	6 (21.4%)
Hyperacusis/distorted sound	3 (10.7%)

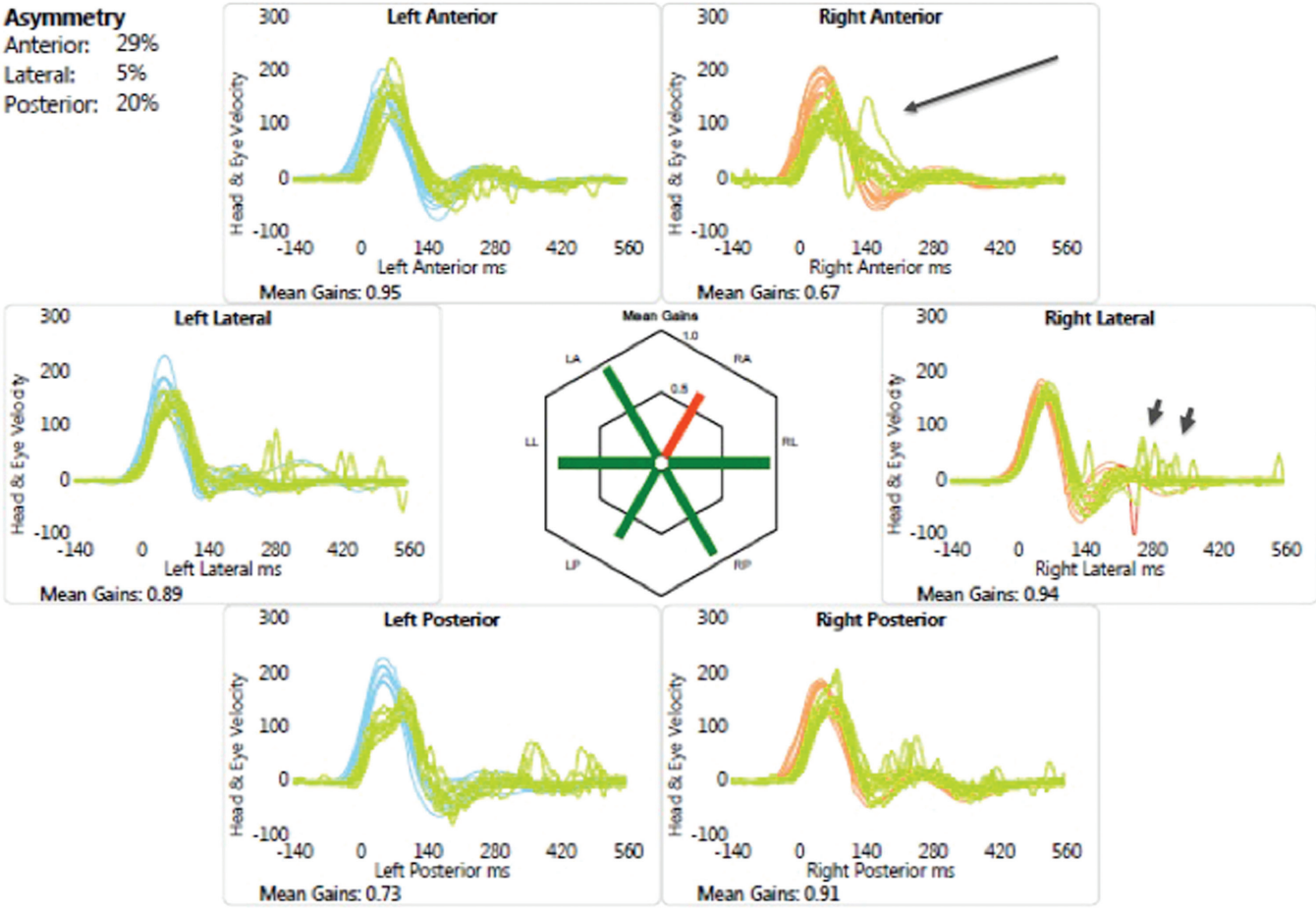

Figure 2. Six-canal vHIT in a symptomatic patient with right SSCD showing reduced VOR gain in the ipsilateral SSC (long arrow) and some overt saccades with normal gains in the ipsilateral lateral semicircular canal (arrow heads); similar responses were recorded in our control group.

(50%), and the left in 9 (32.1%). This is totaling in 33 ears with SSCD in which VOR was assessed in the ipsilateral and contralateral side.

vHIT Analysis

Ipsilateral vHIT output was abnormal (<0.8) in at least 1 canal in 48.5% of ears (n = 16), with the SSC most commonly affected (n = 8, 50%) (Figure 2). In the rest of the cases with abnormal gain, the posterior canal was affected (n = 8, 50%). In addition, 3 ears had abnormal values in 2 canals that were a combined reduced gain in the posterior and lateral canal. A reduced gain in the SSC was an isolated finding. One patient with bilateral dehiscence had normal vHITs. The remaining 4 patients with bilateral SSCD had reduced gain in 1 canal only (posterior: n = 3; superior: n = 1). Table 2 describes in detail the vHIT findings in all patients.

Contralateral vHITs were abnormal in a total of 13 patients (56.5%): 6 posterior canals; 6 SSC; 1 lateral canal. Two-canal reduced gain was found in 4 cases, being a combination of the superior and posterior canals. The presence of contralateral VOR abnormalities did not correlate with the patients' symptoms.

One subject had 2-canal reduced gains in both the ipsilateral (lateral–posterior) and contralateral side (superior–posterior). A specific pattern in the ipsilateral–contralateral side combination of the affected canals was not identified. Seven subjects had abnormal vHITs on both sides at the same time.

There was no difference in the distribution and combination of affected canals and gender or age (P > .05). We also did not identify any link between individual symptoms and abnormal individual canals or 2-canal abnormal gains (P > .05 in all comparisons). Finally, 4 patients had additional abnormalities from other canals other than the ones with abnormal gain (all 4 not affecting the SSC), indicating that abnormal responses can be recorded for every semicircular canal, not only the superior one (Table 2).

The control group comprised 28 patients (F: 17; M: 11) with a mean age of 46.7 (SD: 12.7, range 28-78). Following the assessment of the mean readings from the right and left-sided paired canals, no statistically significant differences were identified for either the control or SSCD group, neither on direct comparison of the ipsilateral SSCD side with a randomly selected side from the control group (all comparisons: P > .05). Eight patients (28.6%) in the control group had abnormal SSC readings (<0.8), 7 in the posterior canals (25%) and 1 patient in the lateral canal (3.6%); this was also confirmed by the raw data and the presence of covert/ overt saccades. Again all cross-tabulation between the ipsilateral SSCD affected side and either of the ears of the unaffected subjects did not show any statistically significant difference in the number of canals having reading below the 0.8 cut-off point. Four patients (14.3%) had abnormal cut-offs bilaterally and 1 patient in more than 1 canal on the same side (superior–posterior). Abnormal saccades were found for additional 4 patients with normal VOR gain.

Table 2. Individual Ipsilateral and Contralateral Responses (Gains and Saccades)

Patient		SSCD		Ipsilateral Superior	Ipsilateral Lateral	Ipsilateral Posterior	Contralateral Superior	Contralateral Lateral	Contralateral Posterior	Raw Data Saccades*
1	Right			0.67	0.94	0.91	0.95	0.89	0.73	Overt R SCC
2	Right			0.93	0.65	1	0.81	0.95	0.91	Overt R LSC and R PSC
3	Left			1.15	0.81	0.93	1.01	0.91	1.2	WNL
4	Left			0.63	1	0.92	0.88	0.85	0.69	Overt L LSC
5	Left			1.33	0.85	0.99	1.07	0.96	1.34	WNL
6	Right			1.02	0.77	0.77	1.02	0.73	0.95	WNL
7	Left			0.98	0.87	1.04	1.03	0.87	0.96	WNL
8	Right			1.06	1.09	0.72	1.14	0.91	0.7	WNL
9	Bilateral	Right		0.96	0.91	0.77				WNL
		Left		0.89	0.81	0.86				WNL
10	Right			0.91	0.91	0.72	0.72	0.88	0.77	WNL
11	Right			0.63	0.98	0.89	1.04	0.9	0.86	WNL
12	Bilateral	Right		0.95	1.01	0.89				WNL
		Left		0.83	0.88	1.09				WNL
13	Left			0.92	0.95	0.85	0.76	1.03	0.88	WNL
14	Left			0.67	0.98	0.83	0.74	1.13	0.77	WNL
15	Right			0.59	1.09	0.93	0.98	1.05	1.01	WNL
16	Right			0.95	1.09	0.88	0.88	0.87	0.89	WNL
17	Bilateral		Right	0.92	1.12	1				WNL
			Left	0.66	1.08	1.04				WNL
18	Bilateral		Left	0.94	0.89	0.89				WNL
			Right	0.92	1.04	0.78				WNL
19	Right			0.8	1.08	0.92	0.92	1.18	1.03	WNL
20	Right			0.8	1.09	0.95	0.9	0.97	0.92	WNL
21	Right			0.7	0.93	0.81	0.85	0.94	0.8	WNL
22	Right			0.93	1	0.84	1	0.87	0.91	Overt R LSC
23	Bilateral		Left	0.93	0.85	0.76				WNL
			Right	0.85	1.01	0.83				WNL
24	Left			0.42	0.93	0.81	0.76	1.04	0.68	WNL
25	Right			1.04	1.03	0.93	0.7	0.96	1	WNL
26	Left			0.86	0.75	0.59	0.5	0.88	0.75	WNL
27	Right			1.04	1.13	1.02	0.97	1	1.06	WNL
28	Left			0.9	0.86	0.89	0.88	1	0.86	WNL

Total ipsilateral ears: 33 (including 5 bilateral cases); Total contralateral ears: 23; R, right; L, left; SCC, superior semicircular canal; LSC, lateral semicircular canal; PSC, posterior semicircular canal; WNL, within normal limits (refers to the canals with normal gain only).

*The raw data reported referring to the findings from the semicircular canals with normal gains for each individuals, as canals with abnormal gains typically gave abnormal saccades.

DISCUSSION

Summary of Key Findings

We report the vHIT findings in a cohort of symptomatic patients with SSCD, showing abnormal responses not necessarily coming from the SSC, when examining both gains and the raw data. Additionally, we demonstrate normal responses in a few symptomatic subjects. When these findings were compared to normal individuals we identified no statistically significant differences in the vHIT gains despite multilevel comparisons. To our knowledge, this is the largest reported series of vHIT responses in SSCD, a relatively rare and under-examined entity, and the first time these responses are compared with a control group.

Despite the increasing use of vHITs in the initial assessment of patients presented with SSCD and the trend to compare pre- with postoperative gains, we did not find any disease-specific role of the use of vHIT in the identification of symptomatic patients compared to normal subjects. However, as vHIT can show responses from all 6 canals, it can be a useful tool in comparing pre- and postoperative responses and identifying the impact of surgery on the VOR. Additionally, vHIT should be used whenever surgical intervention for SSCD is considered to determine the functional level (responses) of the contralateral ear, either in cases with bilateral dehiscence or even in unilateral cases with a possible pre-existing contralateral vestibular deficit. Such information can be crucial in avoiding bilateral vestibular failure. Finally, vHIT can add more information for the patient and facilitate a holistic approach and an informed consent process.

Six-Canal vHIT Results in SSCD in the Literature

vHIT has been recently used for the assessment of SSC function without the need for expensive oculography equipment.[8] The first mention of vHIT assessment in SSCD patients was reported by Schubert et al., in 2006.[9] Assessment of all 6 canals was performed in 5 SSCD subjects and 6 patients post-surgical repair and compared to these of normal subjects. Prior to surgical plugging, a mean reduced gain was noted on the affected side on the superior and posterior canals but the mean horizontal canal mean gain was normal. In the post-intervention cohort, a significantly reduced gain was still evident in the SSC when compared to the rest of the canals.[9] Similarly, Carey et al. (2007) reported gains less than 0.7 for the dehiscent SSC pre-operatively ($n = 19$), with normal values of the rest of the canals. Post-operatively VOR was 40% reduced on the SSC in the operated site but a decrease in the mean VOR values was also noted for the ipsilateral and contralateral posterior canals, which was suggested to be as a result of the plug effect in the common crus for the former and due to loss of inhibitory contribution of the plugged canal, exciting the contralateral posterior canal.[10]

Although our results demonstrate the absence of any disease-specific findings, vHITs can still help in accurately determining changes in all 3 semicircular canal responses when comparing pre- with postoperative VOR gains and raw data.[9,10] Additionally, knowing the precise functional levels of the contralateral labyrinth is important when surgical intervention is considered. This could prevent irreversible bilateral vestibular failure in the presence of a pre-existing contralateral deficit.

The Importance of the Present Findings—Comparison With Previous Studies

The present findings are important in understanding vHIT responses in SSCD and enlighten the vestibular symptoms in patients with such conditions. Half of our tested patients had reduced gains in at least 1 canal and within these, in half of the cases, the SSC was not affected. We also identified abnormal gains on the contralateral side. The hypothesis of overstimulation of the contralateral SCCs in an attempt to compensate for the abnormal stimuli from the affected side is a reasonable explanation for this finding; contralateral abnormalities were found in more than half of our cohort. However, similar findings were found in our control group. This questions the use of vHIT in the battery of specific tests used in the initial assessment of patients with SSCD as well as their value in recording vHIT changes following a surgical intervention that is the current trend in the literature.[9,10] We also failed to identify an association with the canal responses and patients' gender, age, or presenting symptoms. In our study, 25% of patients had non-specific dizziness but no direct association was identified.

The abnormal responses from the affected SSC can be explained by the impact of the third-window on the inner ear homeostasis and the inner ear impedances. These result in the clinical presentation of SSCD with sensitivity to bone-conducted sounds and sound/pressure-induced vertigo and nystagmus. The abnormal responses from other canals than the SSC can potentially be explained by the same mechanisms, given the direct communication of all 3 canals and the ease of transmission of pressure changes through the canals.[10-12] Some studies have tried to explain the abnormality in more than 1 canal found in SSCD. Studies in afferent vestibular nerve responses to pressure changes in chinchillas following SSC fenestration have shown a positive response in all SSC but also a positive response to 33 % of lateral semicircular canal afferents. It has been suggested that increased compliance of the membranous labyrinth due to the dehiscence could result in deflections of the horizontal canal ampulla. Transferable dilational pressure in the ampulla could be also resulting from gradient pressure changes between the endolymph and perilymph due to the fenestration, despite being remote to the horizontal canal.[11] This could have been a reasonable hypothesis for the additional abnormal responses identified primarily in the posterior canal in some of our patients but the fact that similar abnormal findings were seen in our control group makes the above hypothesis questionable.

A previous study on 10 individuals showed weak gains in the ipsilateral horizontal and posterior canals in addition to the affected SSC in 7 out of 10 subjects in vibration-induced VOR testing.[12] Horizontal canal responses were also present during mastoid vibration with the axis of VOR being between the superior and horizontal canal planes, while posterior canal co-stimulation was variable.[12] Despite the different way of testing, the smallest number of patients with SSCD, and the lack of a control group in that previous study, the findings are in agreement with ours.

Finally, it is worth commenting on the abnormal responses that we identified in some of the healthy individuals from the control group. The exact cause of such responses is not entirely understood but one could hypothesize that such responses can be physiological or the

result of a previous subclinical event, severe enough to affect the VOR gains but not the clinical presentation. Additionally, the test–retest reliability can also be hypothesized, despite our tests being performed by very experienced audiologists.

As our knowledge on SSCD improves, identifying whether the vestibular and auditory symptoms might be more or less likely to improve in SSCD patients with normal superior canal VOR gains versus those with superior canal VOR impairment is a further avenue of future research.

Strengths and Weaknesses

SSCD is a relatively rare entity; thus the number of enrolled patients is the main weakness of our study. However, so far, this is the only case-control study of 6-canal vHIT findings in SSCD adding to the existing literature. Additionally, this is a prospective study, eliminating any bias of retrospective data collection, while the vHITs were performed by subspecialized audiologists ensuring reproducible data of high quality, interpreting gains, and raw data. Performance bias of reliability and interpersonal variability of the vHIT testing was eliminated by use of the same test settings and audiologists across the 2 study groups, which were age- and gender-matched.

CONCLUSION

Our study has shown an increased likelihood of abnormal vHIT gain in canals other than the superior with no difference in the gains when compared to an age- and gender-matched control group of unaffected subjects. Despite the recent trends and enthusiasm, the use of vHIT in the assessment of SSCD is therefore unlikely to add disease-specific information in understanding symptom severity. However, it can still facilitate decision-making with identifying abnormal contralateral responses prior to any intervention (potentially avoid bilateral vestibular failure) and provides additional information during the informed consent process.

Ethics Committee Approval: Ethics Committee approval was not required based on the assessment of the study design using the Health Research Authority (HRA) tool. Caldicott guardian approval was obtained instead from the Hospital's Review Board.

Informed Consent: N/A.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – T.T., GK. ; Design - T.T., G.K., MA.MS., M.G.; Supervision - T.T., G.K.; Resource - T.T., MA.MS., M.G.; Materials - T.T., M.A., M.G.; Data Collection and/or Processing - T.T., MA.MS., M.G.; Analysis and/or Interpretation - T.T., MA.MS., M.G. G.K.; Literature Search - T.T., MA.MS.; Writing - T.T., MA.MS., M.G., G.K.; Critical Reviews - T.T., MA.MS., M.G., G.K.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

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