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Covert Saccades in Suppression Head Impulse Paradigm: Compensation Strategy in Unilateral Vestibulopathy

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억제 두부 충동검사에서의 은폐성 단속 운동: 일측성 전정신경병증의 보상 전략

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Background and Objectives In the suppression head impulse paradigm (SHIMP), an alternative protocol to the conventional head impulse paradigm (HIMP) in the video head impulse test (vHIT), covert saccades (CSs) are infrequent, unlike in HIMP. However, some patients with unilateral vestibular loss (UVL) reportedly manifest CSs in SHIMP. This study aimed to evaluate the characteristics of CSs and their role as a compensatory strategy for vestibulopathy in patients undergoing SHIMP.

Subjects and Method This retrospective study included 30 patients diagnosed with UVL based on vestibuloocular reflex (VOR) testing. vHIT was conducted using two paradigms, HIMP and SHIMP. Statistical analyses were performed to compare the characteristics of CSs between the two paradigms and assess the correlation between CSs and anticompensatory saccades (ACSs) in SHIMP.

Results CSs observed in SHIMP exhibited significantly reduced amplitudes and shorter latencies than those in HIMP, with a significant correlation between latency in the two paradigms. ACSs in SHIMP were more pronounced in traces exhibiting CSs, with a positive correlation between the amplitudes of CS and ACS.

Conclusion Our results suggest that CSs observed using SHIMP are associated with the augmentation of ACSs in patients with UVL. This implies that CSs in SHIMP may serve as a rehabilitation strategy, supporting VOR in maintaining forward gaze in patients with UVL. Clinically, the presence of CSs should be considered when interpreting ACSs in SHIMP.

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Introduction

The vestibuloocular reflex (VOR) helps stabilize vision by maintaining focus during angular or linear head acceleration, allowing adequate dynamic visual acuity. Patients with vestibular deficits cannot maintain a stable gaze during head movements, causing the target image to slip on the retina, resulting in low visual acuity and oscillopsia. As a conventional video head impulse test (vHIT), the head impulse paradigm

(HIMP) is widely used to quantify the VOR function of the semicircular canals in the high-frequency domain.[1] In HIMP, subjects are directed to keep their gaze fixed on an earth-fixed target during brief, sudden, and unpredictable head-turns toward the ear being tested. With a normal VOR, the eyes immediately shift in the opposite direction of the head impulse to keep the gaze steady on the target. In individuals with an impaired VOR, the eyes move slower than the head, causing off-target fixation, and requiring a compensatory saccade to regain the target. Therefore, compensatory saccades indicate VOR hypofunction in the semicircular canals.[2,3] These saccades can appear during (covert saccade [CS]) and/or after (overt saccade [OS]) head impulses. CS reduces the amplitude of the subsequent OS.[4] In addition, patients diagnosed with vestibular neuritis exhibit a transition in their saccadic patterns from overt to covert.[5] Moreover, CS accounts for most eye displacements, compensating for head movements and improving dynamic visual acuity.[6-8]

Recently, a new vHIT protocol called the suppression head impulse paradigm (SHIMP) was introduced.[9] The main difference between SHIMP and HIMP is that SHIMP uses head-fixed targets instead of earth-fixed ones. Herein, the target is a laser dot projected by lightweight goggles, which moves in sync with the head during the head impulse. In healthy individuals with normal VOR function, the eyes move in the opposite direction of the head turn, leading them away from the moving target. This necessitates an anticompensatory saccade (ACS) to refocus on the target at the end of the head turn. Patients with an absent VOR do not produce this saccade because their eyes already move along the head during the head impulse. Therefore, the presence and amplitude of this ACS represent the residual vestibular function. Saccades in SHIMP testing are known to occur mainly after the head impulse (OS) and not during it (CS).[9,10] Since CSs in HIMP might hamper the VOR gain calculation due to their interference with eye movement produced by VOR,[11] SHIMP is considered more reliable than HIMP in facilitating the VOR gain calculation.

During the SHIMP routinely conducted at the author's clinic, some patients with unilateral vestibular loss (UVL) reportedly manifest CSs, which is contrary to previous studies reporting that CS is rare in SHIMP.[9] Also, some recent studies have reported the occurrence of CS during SHIMP in patients with UVL.[12,13] Although the mechanism of CSs has not been fully established, the CS in patients with vestibulopathy is thought to be an adaptive mechanism and a strategy to minimize the blurring of vision during head movement, especially in bilateral vestibulopathy.[7,14,15] Therefore, we aimed to examine CSs in SHIMP of patients with UVL, and analyze their association with ACSs in SHIMP and CS in HIMP.

Subjects and Methods

Study population

This study included patients diagnosed with unilateral vestibulopathy at our center between January 2020 and August 2023. UVL diagnosis was based on low VOR gain (<0.8) measured using both HIMP and SHIMP in the affected horizontal plane and normal VOR gain (>0.8) in the unaffected horizontal plane. Among the included patients, those who demonstrated at least two gross CSs in SHIMP were included (Fig. 1). Additionally, to confirm the loss of unilateral vestibular function, the following test results were reviewed: 1) horizontal semicircular canal function by the caloric test, and 2) otolith function by cervical and ocular vestibular-evoked myogenic potentials (VEMPs). Participants were included when they showed at least one of the following results indicatives of weakness in unilateral vestibular function: 1) caloric canal paresis ≥20% in the caloric test, and/or 2) no response or interaural amplitude asymmetry ratio greater than 35% in VEMPs.[16] This study was approved by the Institutional Review Board of our center (IRB No. 3-2023-0411) and conducted in accordance with the Declaration of Helsinki. The same institution waived the requirement for written consent due to the retrospective design of this study.

vHIT

A vHIT device from GN Otometrics (ICS Impulse; GN Otometrics, Taastrup, Denmark) was used to record eye movements for both paradigms. For HIMP testing of the horizontal semicircular canals, the participants were seated in an upright position with their heads tilted forward by 30°, and instructed to gaze at a dot on a white screen (covering most of the visual field) at a distance of 90 cm in a dim light room. Head impulses were administered by the same right-handed examiner, with peak velocities ranging between 150°–250° per second, a rotational amplitude of 15°–20°, and duration of 150–200 ms. A minimum of 20 horizontal head impulses were delivered to each side with unpredictable timing and direction. Eye and head velocities were recorded for each head turn.

The SHIMP testing procedure was the same as that used for the HIMPs, with one exception. The patients were instructed

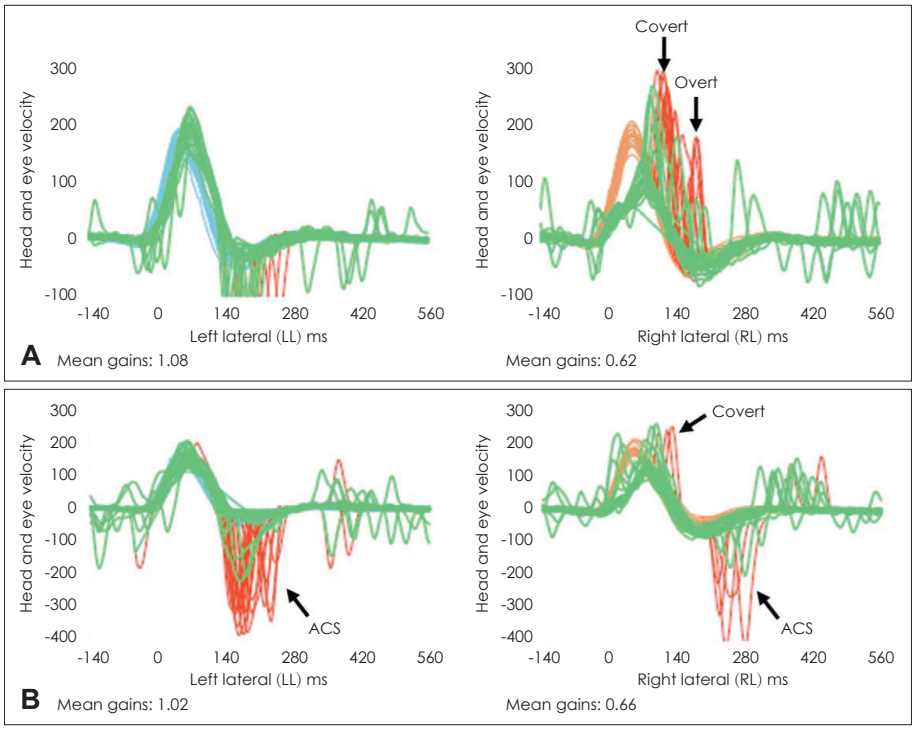

Fig. 1. An example of head impulse paradigm (HIMP) and suppression head impulse paradigm (SHIMP) traces in unilateral vestibulopathy. The figure shows superimposed eye (green) and head (red) velocity traces for many trials. The participant shows low vestibuloocular reflex slope gain in both HIMP and SHIMP on the affected side. A: During HIMP, mixed overt and covert catch-up saccades are present. B: In SHIMP, decreased anti-compensatory saccades (ACSs) accompanied by a few covert saccades are observed.

to focus on a head-fixed target, a laser spot projected by a head-mounted laser on the wall at a distance of 90 cm. Twenty impulses were delivered each to the left and right sides. To avoid anticipation, the head turn always started from the center toward the side, with the gaze ending laterally (passive outward impulse).

Eye movement data

VOR gains were calculated by dividing the area under the head velocity curve by the area under the desaccaded eye velocity curve, using the manufacturer's algorithm.[2] Raw data were exported from the Otometrics system to determine the saccades; only traces accepted by the Otometrics system were exported. All traces were checked for artifacts, and two authors verified each saccade by visually inspecting the raw data. Traces were excluded from the analyses when the peak head velocity was <120°/s. CS is occasionally difficult to distinguish from the VOR response, and its onset is sometimes concealed. Therefore, saccades were included when they 1) occurred after the peak head velocity, 2) had a magnitude of >100°/s and exceeded the peak eye velocity,[17-19] and 3) were clearly distinguishable from the peak eye velocity produced by VOR. CSs were identified as saccades starting before the head velocity had returned to 0°/s, and OSs were identified as the ones beginning after the return to 0°/s head velocity within a maximum latency from the start of head rotation of 500 ms.[3] Head impulse onset was set at a head velocity exceeding 10°/s. The head impulse offset was defined as the head velocity crossing zero.[20] The amplitude (peak velocity) and latency of the saccades were extracted from the first ten artifact-free traces. The latency of the saccade was measured between the onset of the head impulse and the moment of the peak velocity of the saccade.

A total of ten head impulses toward the affected side in the vHIT testing per person were divided into two groups based on the presence and absence of CSs, CS(+) and CS(-), respectively. The average amplitude and latency of the CS were calculated only from CS(+) impulses. The average amplitude and latency of ACSs in SHIMP were analyzed separately in CS(+) and CS(-) groups. If there was no identifiable ACS, the ACS amplitude was defined as zero and the ACS latency was excluded from the calculation.

Statistical analysis

The normality of the dataset was assessed using the Shapiro–Wilk test. A paired-t test or Wilcoxon signed-rank test

was used to compare the results from the two paradigms and the mean ACS amplitudes according to the presence of CSs in SHIMP, depending on the outcome of the normality test. Pearson's or Spearman's correlation analysis was used to determine the correlation between CSs and ACSs in SHIMP. $p<0.05$ indicated statistical significance. Statistical analyses were conducted using the SPSS software ver. 26 (IBM Corp., Armonk, NY, USA)

Results

Demographic and vHIT data

The study population included 30 patients with UVL (12 male and 18 female) (Table 1). The mean age was 53.4±17.9 years. Definite and probable etiologies included vestibular neuritis (n=11), vestibular schwannoma (n=17), and labyrinthitis (n=2). The duration of dizziness ranged from 1 day to 4 years, with an average of 21.7±38.8 weeks. The mean canal paresis in the caloric test was 68.5%±27.3%. In VEMPs, 80.8% of patients in cervical VEMP and 76.0% in ocular VEMP showed either no response or an interaural amplitude asymmetry ratio greater than 35%. The average HIMP VOR gain in the lateral canal of the affected side was 0.46±0.14, which was significantly higher than the SHIMP VOR gain (0.41±0.15; paired t-test, p=0.004). The average peak head velocity was significantly lower during SHIMP compared to HIMP (187.68 ±16.26°/s and 200.85±21.17°/s, respectively; paired t-test, p=0.002). However, the values for both HIMP and SHIMP fell within the recommended peak head velocity range of 150−250°/s.

Table 1. Information of enrolled patients and video head impulse test data (n=30)

	Values	*p*-value
Age (yr)	53.4±17.9	N/A
Male:female	12:18	N/A
Duration of dizziness (week)	21.7±38.8	N/A
Etiology		N/A
Vestibular neuritis	11 (36.67)	
Vestibular schwannoma	17 (56.67)	
Labyrinthitis	2 (6.67)	
Gain of affected LSCC		0.004*
HIMP	0.46±0.14	
SHIMP	0.41±0.15	
Peak head velocity (deg/s)		0.002*
HIMP	200.85±21.17	
SHIMP	187.68±16.26	
Average CS occurrence rate (%)		0.104
HIMP	44.00±0.35	
SHIMP	33.00±0.21	

Values are presented as mean±standard deviation or n (%). *$p < 0.05$. N/A, not applicable; LSCC, lateral semicircular canal; HIMP, head impulse paradigm; SHIMP, suppression head impulse paradigm; CS occurrence rate, covert saccade presence impulses divided by total impulses

CS

CS amplitude and latency was compared between HIMP and SHIMP. The average amplitude of HIMP CSs was 250.12 ±55.80°/s, and that of SHIMP CSs was 206.75±51.40°/s, significantly lower than HIMP (paired t-test, p=0.021) (Fig. 2). The average CS latency in SHIMP was significantly shorter (110.98±13.05 ms) than in HIMP, (120.85±13.17 ms; paired t-test, p=0.002). The CSs of both tests showed a significant correlation with the latency (Spearman's correlation, correlation coefficient=0.469, p=0.018) but not with the amplitude.

ACS

In SHIMP, the average ACS amplitude was 236.59±78.84°/s, and its average latency was 271.22±56.72 ms. The SHIMP impulses of each patient were categorized into two groups based on the presence or absence of SHIMP CSs: CS(+) and CS(-) (Fig. 3A and B). The average ACS amplitude in the CS(+) impulses was higher than in the CS(-) impulses, measuring 172.78±104.77°/s and 122.66±103.38°/s, respectively (Wilcoxon signed rank test, p=0.009) (Fig. 4A). However, the average SHIMP gain did not differ between the two trace groups, 0.43±0.18 in covert-present traces and 0.41±0.17 in covert-absent traces (Wilcoxon signed rank test, p=0.179) (Fig. 4B). The average ACS latency also showed no difference between the two trace groups, 279.54±65.61 ms in covert-present traces and 268.26±61.27 ms in covert-absent traces (Wilcoxon signed rank test, p=0.848) (Fig. 4C).

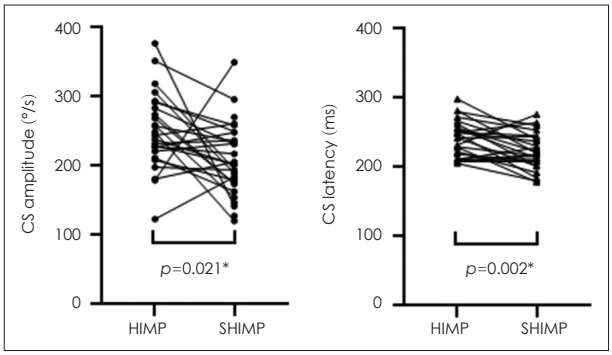

Fig. 2. Comparing the features of covert saccades (CSs) in head impulse paradigm (HIMP) and suppression head impulse paradigm (SHIMP). The average amplitude and latency of the SHIMP CSs were lower than those of the HIMP CSs. *p<0.05.

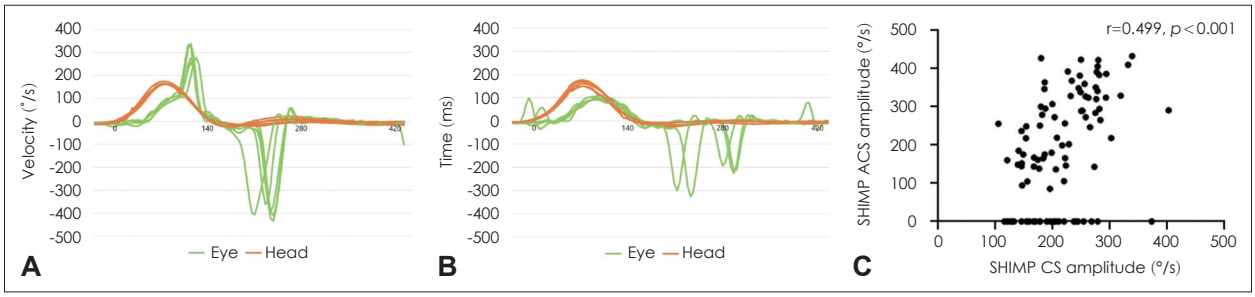

Fig. 3. The suppression head impulse paradigm (SHIMP) traces from each patient were divided into two groups depending on the presence of covert saccades. Representative data from one of the enrolled patients. A: Traces with a covert saccade (CS), followed by a large anticompensatory saccade (ACS). B: Traces without a CS, followed by a smaller ACS. C: Covert-present SHIMP traces from all subjects are collected. A significant correlation was observed between the amplitudes of CSs and ACSs.

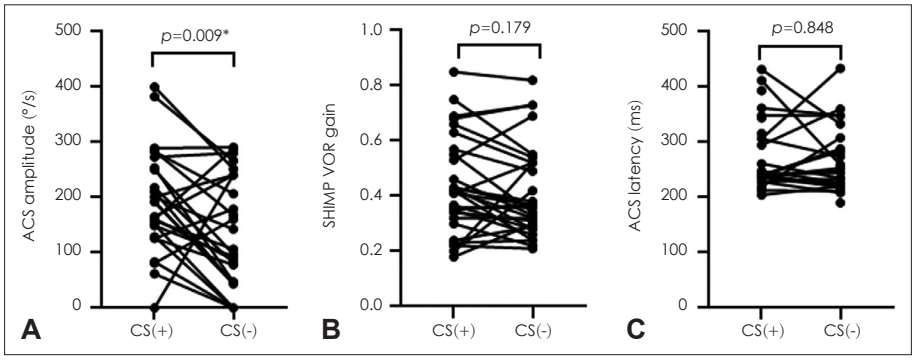

Fig. 4. Comparison of anticompensatory saccades (ACSs) based on the presence of covert saccades (CSs). A: CS-present traces showed significantly larger average amplitude of ACS compared to CS-absent traces. B: The average suppression head impulse paradigm (SHIMP) vestibuloocular reflex (VOR) gain showed no difference between CS-present traces and CS-absent traces. C: The average latency of ACS showed no difference between CS-present traces and CS-absent traces. *p<0.05.

A total of 101 traces were obtained by collecting all the SHIMP traces that exhibited CSs from all subjects. The correlation between the amplitudes of CSs and ACSs was confirmed (Pearson's correlation, correlation coefficient 0.499, p<0.001) (Fig. 3C).

Discussion

Although CSs in SHIMP reportedly occur infrequently, they can be present in some patients, playing a role in compensating for insufficient VOR. The results of this study support the notion that the presence of CSs significantly increases ACS amplitude. Furthermore, CS and ACS amplitudes were significantly correlated. In addition, the latency and amplitude of CSs in SHIMP were significantly lower than those in HIMP.

Previous studies have indicated that CSs are rarely elicited during SHIMP compared with during HIMP.[9,10] During SHIMP, when a target moves with head rotation, VOR becomes counterproductive for tracking the target. It has been suggested that VOR cancellation during passive head impulses occurs approximately 80−90 ms after the onset of head rotation, preceding the availability of visual feedback.[21-23] Moreover, the latency of VOR cancellation decreases as head acceleration increases. Therefore, participants were expected to be able to suppress their VOR to some extent during head turns in SHIMP. This is one hypothesis for the slightly lower VOR gains during SHIMP than during HIMP.[9,24] Since CSs appears to be generated to compensate for the deficient VOR, they may be suppressed by the same mechanism involved in VOR cancellation during SHIMP. This could be the underlying cause of the lower incidence of covert saccades in SHIMP than HIMP.

Nevertheless, studies have reported CSs during SHIMP in patients with bilateral vestibular loss (BVL).[20,25] During SHIMP, fewer CSs were produced compared to HIMP, with occurrence rates of 5%−6% and 34%−35%, respectively.[20] However, some patients with BVL who consistently generated a CS during SHIMP required a corresponding large ACS at the end of the head impulse to maintain their gaze on the head-fixed target. As these patients did not report oscillopsia despite impaired bilateral VOR gain, the authors suggest that

CSs in these patients during SHIMP may be a rehabilitation strategy.[25] The occurrence rates of CSs during HIMP and SHIMP in patients with UVL in this study were 44% and 33%, respectively, which are higher than those reported in previous study. This discrepancy can be attributed to the study's inclusion criteria, which specifically selected patients who demonstrated at least two gross CSs during SHIMP. As a result, the rates reported here reflect the selection of a specific subset of UVL patients with more prominent CSs and should not be interpreted as representative of the general occurrence rate of CSs in all UVL patients.

Unlike the relatively simple VOR arc, the mechanism through which corrective saccades are generated remains unclear. Volitional saccades are suggested to be the main triggers of corrective saccades, including OSs and ACSs.[6,26] However, the latency of the CS is <150 ms and can be as low as 70 ms, which is much less than volitional saccades to new visual targets (approximately 200 ms).[3,15] This suggests that CS can be triggered by nonvisual cues.[6] The remaining vestibular function and somatosensory input from the neck have been proposed as possible triggers for corrective saccades.[5] Other causes of activation might be the sensory cues that the head is about to or has just begun to rotate[27] or through the generation of internal models by the central nervous system.[28] Collectively, the mechanism of CSs is less likely to be related to volitional saccades, but rather to the generation of VOR, as hypothesized by the CS being inhibited by VOR cancellation (less frequent in SHIMP than in HIMP). However, the exact mechanisms and pathways involved must be investigated in future studies.

A previous comparative study of HIMP and SHIMP found no correlation between the CSs observed in the two paradigms.[29] In this study, the average amplitude and latency of the SHIMP CSs were lower than those of the HIMP CSs. In addition, the CS latency was significantly correlated between the two test paradigms. The only difference between the two paradigms is the position of the visual target. However, vision has not been considered as a trigger for CSs because visually induced saccades usually have longer latencies.[30,31] In addition, CS in patients with UVL show minimal change when performed in the dark without visual input, whereas OS is more visually sensitive.[32] Therefore, we suggest that the CS mechanisms in these two paradigms are identical. The differences in the CS amplitude and latency are possibly explained by several factors. First, the average peak head velocity differed between the two paradigms, although it was within the recommended range.[19] Second, patients might have become accustomed to the test protocol and anticipated upcoming impulses during SHIMP testing (despite randomly directed impulses), as the SHIMP tests were always conducted after HIMP tests. CS latency is shorter during active head turns than during passive ones, and predictable passive head movements trigger CSs earlier than unpredictable movements.[28,33-35] Lastly, VOR cancellation in SHIMP possibly affects the reduction of CS amplitude and latency.

The average ACS amplitude in the CS(+) traces was higher than in the CS(-) traces. Additionally, there was a significant correlation between the ACS and CS amplitudes of patients with UVL in SHIMP. This implies that CSs, operating in a manner supportive of VOR in maintaining a forward gaze, lead to an augmentation of ACSs, facilitating the reorientation of the eyes toward the target. Therefore, CSs in SHIMP appear to be a rehabilitation strategy in patients with UVL. This finding is consistent with previous studies involving patients with BVL.[25]

Nevertheless, this study had some limitations. First, as we focused on patients displaying CSs in SHIMP, the assessment of the incidence of SHIMP CSs in patients with UVL was not feasible. Second, the patient group was heterogeneous, encompassing both acute and chronic UVL of diverse etiologies. Given that the rehabilitation strategies may vary depending on the stage and etiology of UVL, the occurrence of saccades is expected to differ according to these factors. However, as the primary objective of this study was to examine the association of SHIMP CSs, ACSs, and HIMP CSs in each individual using pairwise analysis, heterogeneity in disease presentation did not significantly diminish the relevance of the study.

In conclusion, this study suggests that CSs observed in SHIMP are associated with the augmentation of ACSs in patients with UVL. This implies that CSs in SHIMP may serve as a rehabilitation strategy, supporting VOR in maintaining a forward gaze in patients with UVL. Clinically, the presence of CSs should be considered when interpreting ACSs in SHIMP.

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Author Contribution

Conceptualization: Seong Hoon Bae. Data curation: Ji Min Yun, Yujin Lee. Formal analysis: Ji Min Yun. Investigation: Ji Min Yun, Seong Hoon Bae. Methodology: Ji Min Yun, Seong Hoon Bae. Project administration: Ji Min Yun. Resources: Seong Hoon Bae, Sung Huhn Kim. Software: Ji Min Yun. Supervision: Seong Hoon Bae.

Validation: Ji Min Yun, Yujin Lee. Visualization: Ji Min Yun. Writing—original draft: Ji Min Yun, Yujin Lee. Writing—review & editing: Seong Hoon Bae, Sung Huhn Kim.

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