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Suppression Head Impulse Paradigm (SHIMP) in evaluating the vestibulo-saccadic interaction in patients with Vestibular Neuritis.

Manzari Leonardo [1] , Tramontano Marco [2,3]

1 MSA ENT Academy Center, Cassino, Italy

2 Fondazione Santa Lucia IRCCS, Rome, Italy

3 Department of Public Health and Infectious Diseases, Sapienza University of Rome, Piazzale Aldo Moro 5, Rome 00185, Italy

Corresponding Author

Manzari Leonardo , M.D., **[ORCID 0000-0001-9746-5856]**

MSA ENT Academy Center, Cassino, Italy

email: lmanzari1962@gmail.com

Tel and Fax 0039 0776310745

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Suppression Head Impulse Paradigm (SHIMP) in evaluating the vestibulo-saccadic interaction in patients with Vestibular Neuritis.

Purpose

Evaluate the potential clinical application of the Suppression Head Impulse Paradigm (SHIMP) in evaluating the vestibulo-saccadic interaction in patients with Vestibular Neuritis (VN).

Methods

A retrospective study was performed. Fifteen patients diagnosed with unilateral VN were identified from a database of ENT vestibular clinic from January 2011 through February 2020. Medical records were reviewed to determine clinical presentation, vestibular testing results, treatment, and recovery.

Results

Fifteen patients (7 left ear, 8 right ear, mean age 58.73 ±10.73, six female) met the inclusion criteria and were enrolled in the study. Significant differences were found in the within-subjects analysis at T1 in DHI score ($p=0.001$), VOR gain ($p<0.005$) and in the percentages of impulses containing a SHIMPs saccade when the head is passively turned toward the affected side ($p=0.001$).

Conclusions

SHIMPs paradigm provides useful information about the value of vestibulo-saccadic interaction as new recovery strategies in patients with VN

Keywords: Vestibular Neuritis, Vestibulo-Ocular Reflex, SHIMP, DHI, Saccade, Vestibular compensation

Introduction

The functional state of the vestibular system can be assessed by measuring the corrective eye movement during an unpredictable head movement, the Vestibulo-Ocular reflex (VOR) [1].

It has been proven that the first 100 ms of the eye movement does not defend from other sources of vision control and the earliest part of the eye movement responds only to a vestibular stimulus [2,3]. Head Impulse Test (HIT or HIMP) assesses the functional state of each semicircular canal and the subject is instructed to maintain fixation on an earth-fixed target during small, abrupt, passive, unpredictable head turns (head impulses) in the plane of the canal under test [4,5]. After its clinical validation vs dual magnetic scleral search coil [6] video Head Impulse Test (vHIT) constitutes the diagnostic clinical gold standard of the horizontal and vertical VOR. Thus, during the HIMP the patient with reduced unilateral horizontal or vertical canal function fails to maintain fixation on an earth-fixed target and so makes corrective (overt or covert) saccades during or at the end of the head rotation in order to regain fixation [6]. These corrective saccades are a substitution sign of the reduced VOR dynamic function [6].

The vHIT test provides an objective measure of VOR gain and VOR gain asymmetry (analogous to caloric canal paresis) just like dual magnetic scleral search coil and it is well tolerated by all subjects even at a young age as well as in a senior age and it is a quick test (three to four minutes maximum) to measure dynamic vestibular functions.

Few years ago a new complementary test paradigm was proposed to be an indicator of vestibular function [7]. This paradigm has been named suppression head impulse test (SHIMP), where the subject is asked to look at a head-fixed target rather than at the earth-fixed target used in HIMP during the passive head turn. Healthy subjects make a corrective saccade (a "SHIMPs" saccade), whereas patients at the time of acute peripheral vestibular loss do not [7]. SHIMPs paradigm gives more precise information on the VOR gain compared to HIMPs because the evaluation of the gain is not affected by covert saccades [8].

Two studies [9,10], have already suggested the use of vHIT and HIMP paradigm to detect vestibular patients in the acute phase.

Recently, saccades during SHIMPs has been related to the reduction of oscillopsia in patients with bilateral vestibular loss and would appear to be a compensatory strategy, and the anti-compensatory saccade could show how well patients are learning to generate them at the end of the head movement [10].

Our hypothesis is that SHIMPs anti-compensatory saccades and their relationship with the vestibular input could be a useful mechanism for minimizing subjective symptoms after vestibular loss through an efficient interaction and to date no studies investigated the application of SHIMPs paradigm in the acute stage, the time of the attack, in comparison with the subjective recovery stage after vestibular neuritis.

Thus the aim of this study is to investigate SHIMPs paradigm during follow-up in patients with VN.

Methods

Study design

This is a retrospective study aimed to investigate the recovery of vestibular and saccade system interaction after VN. All procedures contributing to this work comply with the ethical standards of the relevant national and institutional guidelines on human experimentation and with the Helsinki Declaration of 1975. The study was carried out according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. All patients gave written consent to publish the results obtained from their clinical examinations and instrumental tests.

Setting

Medical records of patients with a diagnosis of Vestibular Neuritis (VN) who were admitted to the ENT MSA Academy Center from November 2011 to January 2020 were reviewed.
SHIMP testing is in routine clinical use at MSA ENT Academy Center since 2011 and has been used on thousands of patients by an expert physician.

Participants

The inclusion criteria in this study were: 1) diagnosis of VN in acute phase (<72 hours since the acute vestibular syndrome [AVS]); 2) at least three vestibular function evaluation sessions in 1 month or later; 3) absence of hearing loss on pure tone audiometry that could be related to other type of vestibular pathology(i.e. Ménière's Disease) and abnormal findings on neurologic examination 4) patients who were not undergone pharmacological or rehabilitative treatment for VN.
We excluded the medical records of patients who showed one of the following exclusion criteria: 1) other vestibular diagnosis (e.g., VN > 72 hours since the acute vestibular syndrome, Meniere disease, bilateral vestibular loss, vestibular migraine, BPPV, etc.), somatic or psychiatric disorders; 2) presence of neurological diseases.
At the time of the first evaluation all patients were instructed to return to the normal daily activities as soon as possible.
All patients admitted to MSA ENT Academy Center, Cassino (Italy) with a diagnosis of VN were undergone to a vestibular assessment that included a self-assessment inventory with DHI, an assessment of horizontal and vertical semicircular canals with bed side HIT + vHIT, Air Conducted Sound and Bone Conducted Vibration Cervical and Ocular VEMPs.
Data on Horizontal Semicircular VOR gain and on eye velocity, head velocity and percentages of impulses containing a SHIMPs saccade during the SHIMPs procedure was collected.
All patients were evaluated in the first days and at least after 1 month or later after the AVS.
VN was diagnosed on the following criteria : a) a history of acute onset of severe, prolonged, rotatory vertigo, nausea, and postural imbalance; b) on clinical examination the presence of horizontal spontaneous nystagmus with a rotational component toward the unaffected ear (fast phase) without evidence of a central vestibular lesion; c) abnormal bed-side HIT showing an ipsilateral deficit of the horizontal semicircular canal [5]; d) alterations in the VEMPs results and absence of neurological signs; e) an MRI of the brain that showed no lesions that could account for any vestibular disturbance. Demographic characteristics are reported in Table 1.

Insert Table 1 around here

Dizziness Handicap Inventory (DHI)

The quality of life of all VN patients were assessed by the DHI.
The DHI is a self-assessment inventory, including 25 questions to evaluate self-perceived activity limitation and restriction resulting from dizziness [11].

Video Head Impulse Test

The function of the horizontal semicircular canals was assessed by using horizontal video-HIT (OtosuiteV®, GN Otometrics, Denmark) as previously described [6]. Subjects were instructed to fixate an earth-fixed dot on the wall at 1m distance in front of them. Room lighting conditions were adjusted to ensure that the pupil was small and the pupil image was not affected by reflections in the pupil image at any point in the range of the head movement. At each testing

epoch the clinician (L.M.) applied about 20 brief, rapid, horizontal head turns (head impulses) to each side, always starting from centre, with unpredictable timing and direction with minimal bounce-back or overshoot at the end of the head impulse: each head impulse was “turn and stop”. The amplitude of the head rotation was about 10–15 deg, and the peak head velocity of the impulse was about 140–220deg/s, with angular accelerations of between about 3000 deg/s 2 and 5000 deg/s 2. Eye velocity and head velocity were recorded for each head turn.

SHIMPs testing procedure was exactly the same as for HIMPs with one exception. Participants were instructed to fixate a head-fixed target—a laser spot projected on the wall at 90 cm distance in front of them projected by a head-mounted laser [7]. This spot moved with the head, and during testing it appeared to subjects that they were looking at a dot which unexpectedly jumped around. At least ten impulses were delivered to left and right sides, respectively. To avoid anticipation, the head turn always started from the center. Eye velocity, head velocity and percentage of impulses containing saccades were recorded and evaluated in each head rotation.
VOR gain were calculated during HIMP and SHIMPs paradigm. HIMP gains (<0.76) and SHIMP gains (<0.66) identified the affected side of Unilateral VN with 100% sensitivity (48–100) and 100% specificity (74–100) and an AUC of 1.0 (0.81–1.0, $p < 0.0001$) [6,7].

Insert Figure 1 about here.

Statistical Analysis

The average horizontal slow phase eye velocity VOR gain for each side was calculated for each as the sum of the VOR gains for each trial. The percentages of impulses containing a SHIMPs saccade during the evaluation sessions were calculated in both affected and healthy vestibular side. Statistical analysis was performed with IBM SPSS Statistics software (v23, IBM Corp., Armonk, NY, U.S.A.). Data were reported in terms of means and standard deviations. The Wilcoxon signed ranks test were used for the within-subjects comparison. The Pearson's Correlation Coefficient was calculated at the baseline T0 and at T1, between DHI score and VOR gain and between DHI score and the percentages of impulses containing a SHIMPs saccade. To test the impact of confounding factors on our outcome, we performed a multivariate regression analysis with DHI score as dependent variable and percentages of impulses containing a SHIMPs saccade, VOR gain at T1 and the time of final evaluation as independent variables.
to evaluate the role of confounding factors on final results.

Results

We studied 15 patients suffering from VN (7 left ear, 8 right ear, mean age 58.73 ±10.73, six female), they met the inclusion criteria and were enrolled in the study. 13 out 15 patients had a superior VN and only 2 had VN *in toto*.
All clinical tests were performed at the MSA ENT Academy Center in Cassino by the same examiner (LM). Significant differences were found in the within-subjects analysis at T1 in DHI score (p=0.001), VOR gain (p<0.005) and in the percentages of impulses containing a SHIMPs saccade when the head is passively turned toward the affected side (p=0.001). In reverse when the head is passively turned toward the healthy side all patients had 100% of impulses containing a SHIMPs saccade at T0 and T1.
The correlation analysis showed significant results between DHI score and the percentages of impulses containing a SHIMPs saccade in the affected side at T1. Multivariate regression model showed that only the independent variable

percentages of impulses containing a SHIMPs saccade have an effect on DHI score (β=-0.666; t=-2.952; p=0.013) whereas the variables VOR gain at T1 and the time of final evaluation did not have any effect (all p>0.05).

All clinical data were reported in Table 2.

Insert Table 2 about here

~~Conclusion~~ Discussion

The aim of this study was to evaluate the recovery mechanisms in VN patients during SHIMPs paradigm.

During HIMPs paradigm the VOR acts to keep gaze on the earth-fixed target.

Conversely in the SHIMPs paradigm in the normal subjects initially the VOR drives the eyes opposite to the direction of head turn and so off the target, i.e. during head turn to the left, the head (and target) are pointed to the left, while the subjects gaze is directed to the right. So to regain the target as instructed at the end of the rotation, the healthy subject must make a large overt anti-compensatory saccade from right to left, but very important thing not during the head rotation. It is important to underline how this phenomenon occurs at the end of the rotation of the head.

In this way SHIMPs paradigm gives the clinician more precise information about VOR gain compared to HIMPs because, also as our cohort showed, since it is not affected by covert saccades.

Furthermore, as previously described by Shen et al.[8] performing HIMPs and SHIMPs in the same acute UVL patient revealed that compensatory catch-up saccades always occurred during HIMPs, while the anti-compensatory catch-up saccades were more inconsistent during SHIMPs.

However, there is a difference between the two patient cohorts. Shen et al.[8] tested their iatrogenic UVL patients within 6 weeks after the surgical procedure. In contrast, we tested for the first time our patients within 72 hours of the onset of acute vestibular syndrome due to vestibular neuritis

Anyway SHIMP paradigm revealed two important advantages vs HIMP in the clinical setting: i) It allows the clinician, through vHIT, to obtain the exact measurement of the VOR gain slow phase ; ii) the mechanism that returns the eyes to the target is that of saccadic suppression [13]. These results were relevant for several reasons.

Firstly, A patient with acute unilateral vestibular loss, i.e. operated from unilateral vestibular schwannoma[8], does not make a corrective saccade because, he has no VOR to drive his eyes off the target, so at the end of the SHIMPs impulse he is looking at the target [8].

Although our cohort study has a different diagnosis (Vestibular Neuritis vs iatrogenic UVL) from that studied by Shen et al. [8], in our group 11 out 15 (73,3%) patients did not show anti-compensatory SHIMPs saccades, while 4 out 15 a very small number compared to the number of head impulses when tested within 72 hours from the symtom onset.

From these considerations, in acute VN patients, unlike in acute surgical unilateral Vestibular Deafferented (uVD) patients, the recovery from AVS can be not only due to central compensation but also to recovery of peripheral function [12,13-15]. VOR gain recovery can be total [13,14], but also, as we have shown, partial, not reaching normal values and this cannot be expected in patients with iatrogenic UVL.

Secondly. In our study group, 13 out of 15 patients at T1 showed a “tiny” increase in VOR gain that correlates with DHI score and with an increase of percentages of impulses containing a SHIMPs saccade in the affected side.

We therefore want to further stress the concept that although the VOR gain can recover and return to normal values [15,16] in this cohort study it improves never reaching the normal cut off at T1.

It should also be noted that in 2 out of 15 patients there was no increase in the VOR gain at T1. Nevertheless they equally had an increasing percentage of impulse containing SHIMPs saccades. In these two patients we might hypothesize that another compensatory strategy is activated differently from the rest of the sample (i.e. somatosensory input).

Interestingly is that although the VOR gain is still deficient the subjective symptoms referred by the patients are significantly improved which we believe may be related to the reappearance of the SHIMPs.

Indeed we found a significant increase of percentages of impulses containing a SHIMPs saccade in the affected side at T1.

Thirdly. We could hypothesize that a tiny recovery of the VOR gain [seeTable 2], may be responsible for the reappearance of anti-compensatory SHIMPs saccades.

When a passive impulse is applied,VOR gain drives the eyes off the target thus reactivating the vestibulo-saccadic interaction with VOR suppression by the saccadic system [17], this correlates with a significant increase of the DHI score meaning that this mechanism can compensate for gaze stability during the activities of daily living. Furthermore a multivariate regression analysis showed a significant effect of increasing percentages of impulse containing SHIMPs saccades on DHI score at T1 suggesting a positive effect of this factor on patients' disability.

We acknowledge some limitations of the present study. First, as a retrospective study, there are limitations in the interpretation of our results, secondly, the sample size is relatively small but the manuscript would be a preliminary scientific report to better design further SHIMPs saccades studies.

Conclusion

SHIMPs paradigm provides useful information about the value of vestibulo-saccadic interaction as new recovery strategies in patients with VN. Further SHIMPs saccades studies are clearly needed in VN patients especially between those without VOR gain recovery.

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Data availability statement: The data that support the findings of this study are available from the corresponding author [LM] upon reasonable request

Ethics approval: the study was approved by local ethics committee

Table 1: Demographic and clinical characteristics at baseline

Table 2: vHIT SHIMPs data for Vestibular Neuritis patients

Figure 1: SHIMP TEST in patient n°8

Caption

Figure 1:

The corresponding records for one patient who was diagnosed as having an acute (right) vestibular neuritis on occasion T0 (21-11-2011) and then tested on occasion T1 (10-1-2012).

In this patient the eye velocity is substantially less than head velocity on occasion 1(T0) when the passive impulses are toward the lesion side [B]. However the eye velocity response during the head impulse changes only slightly over the succeeding test and at the final test (T1) on 9-1-2012 the VOR gain increases at 0.23 [D] - not the same (slightly greater) than the original VOR gain of 0.16.

In the second test there is a clear reappearance of anti-compensatory SHIMPs saccades [D] at the end of the head turn after the head velocity has returned to zero and so are classified as overt saccades.

This is evidence that the slow phase eye velocity to high acceleration head impulses has changed over time albeit slightly, and so we conclude that the neuritis has resolved even if has caused probably permanent damage (as a matter of fact VOR gain remained below the lower normal limits, 0.23<0.66 in SHIMPs paradigm).

There is strong evidence of changes in the pattern of saccades.

At the last test, 9th january 2012, this patient reported good balance function and considered that they had subjectively recovered from Vestibular Neuritis .

Caption

Figure 2:

The corresponding records, both paradigms SHIMPs vs HIMPs, for the same patient who was diagnosed as having an acute (right) vestibular neuritis on occasion T0 (21-11-2011) [A-B] and then tested on occasion T1 (10-1-2012) [C-D].

In this patient the eye velocity is substantially less than head velocity on occasion 1(T0) for both paradigms when the passive impulses are toward the lesion side [A-B].

It is confirmed, however, that eye velocity response during the head impulse changes slightly over the succeeding test and at the final test (T1) on 9-1-2012 the VOR gain increases at 0.23 (SHIMPs) [C], 0.49 (HIMPs) [D] - not the same (slightly greater) than the original VOR gain of 0.16(SHIMPs) [A], 0.26 (HIMPs) [D].

At the time of the first tests the HIMPs paradigms clearly highlights a shower of “Covert” and “Overt” saccades (black arrows) [B].

In the second test there is a clear reappearance of anti-compensatory SHIMPs saccades [C] (SHIMPs paradigm, red arrows) at the end of the head turn after the head velocity has returned to zero and so are classified as overt saccades, while, for the HIMPs paradigm [D] when the passive impulses are toward the lesion side, the saccades pattern has clearly changed, in a couple of saccades clusters, "Covert" and "Overt(black arrows).

This is another evidence that the slow phase eye velocity to high acceleration head impulses has changed over time albeit slightly for both paradigms, SHIMPs and HIMPs, as a matter of fact VOR gain remained below the lower normal limits, 0.23<0.66 in SHIMPs paradigm – 0.49 <0.76 in HIMPs paradigm.

There is strong evidence of changes in the pattern of saccades for both paradigms: a) the SHIMPs saccades reappearance; b) the HIMPs saccade clustering.

Figure 1

Click here to access/download;Figure;Figure 1.tif

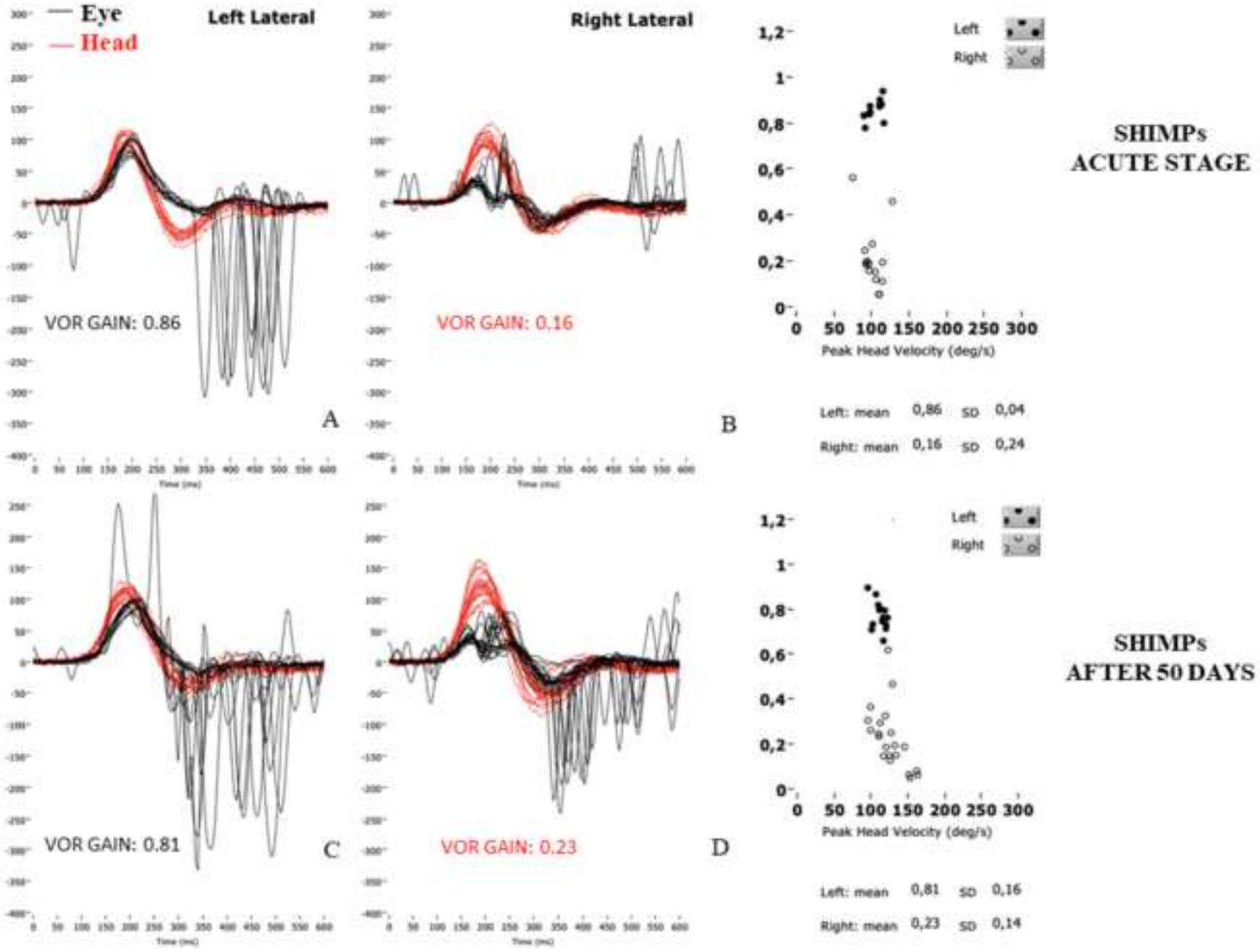
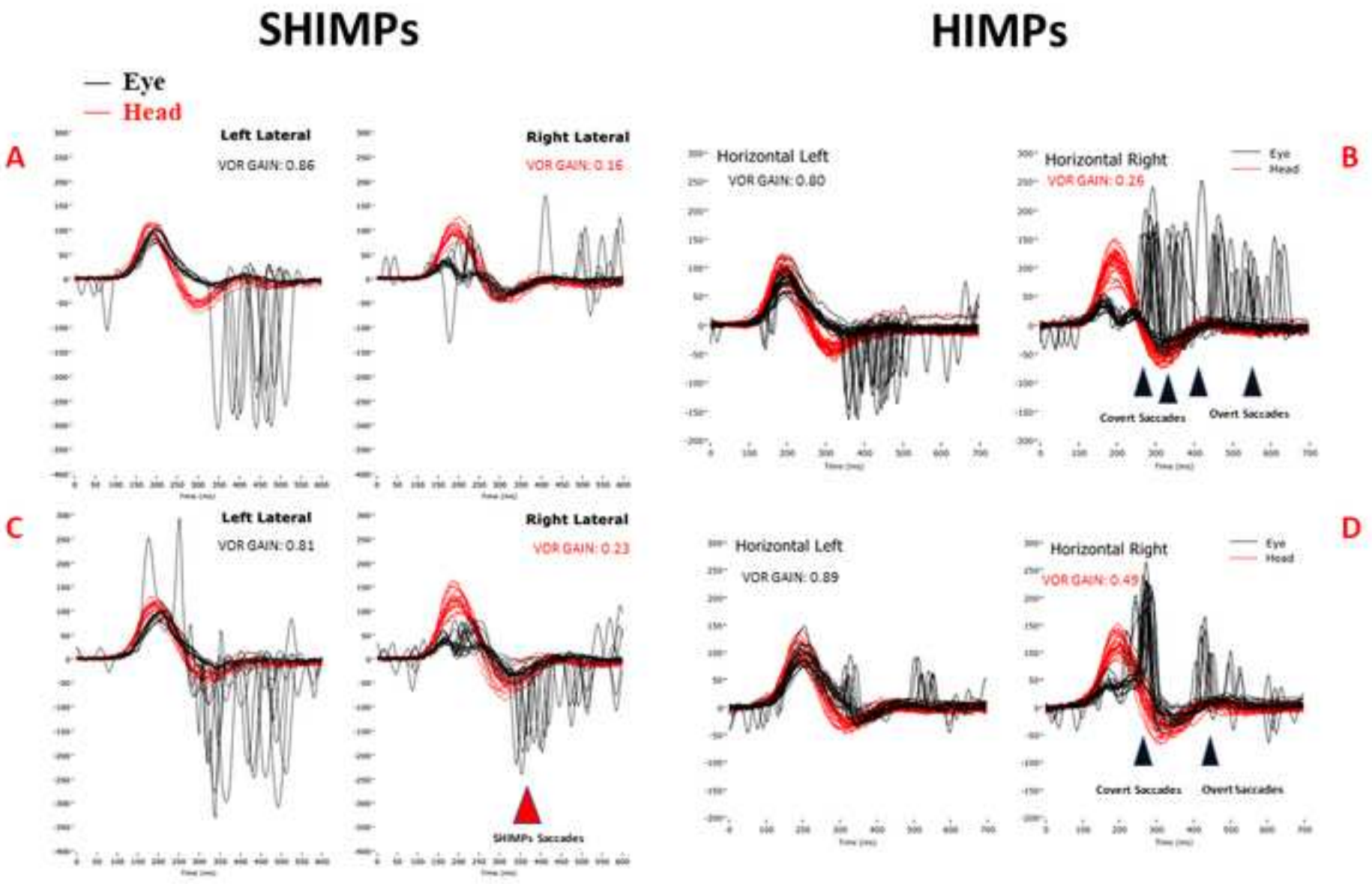


Figure 2

Click here to access/download;Figure;Fig.2 Manzari_Tramontano.tif



Tables

Table 1. Demographic and clinical characteristics at baseline

Patients	Gender	Side	Initial signs and symptoms		Investigations		Length of time between symptom onset				Evaluations
			Spont Ny	hHIT	CT	MRI	Symptom Onset	First vHIT evaluation	Therapy	Recommendation	Number
1	F	LEFT	Yes	Yes	-	Negative	< 24 hours	< 24 hours	none	early mobilization	3
2	F	RIGHT	Yes	Yes	-	Negative	< 48 hours	< 48 hours	none	early mobilization	4
3	M	LEFT	Yes	Yes	-	Negative	< 48 hours	< 48 hours	none	early mobilization	3
4	M	LEFT	Yes	Yes	-	Negative	< 24 hours	< 24 hours	none	early mobilization	3
5	F	RIGHT	Yes	Yes	-	Negative	< 48 hours	< 48 hours	none	early mobilization	5
6	F	LEFT	Yes	Yes	-	Negative	< 24 hours	< 24 hours	none	early mobilization	4
7	M	RIGHT	Yes	Yes	-	Negative	< 48 hours	< 48 hours	none	early mobilization	4
8	M	RIGHT	Yes	Yes	-	Negative	< 24 hours	< 24 hours	none	early mobilization	5
9	F	RIGHT	Yes	Yes	-	Negative	< 24 hours	< 24 hours	none	early mobilization	4
10	F	RIGHT	Yes	Yes	-	Negative	< 48 hours	< 48 hours	none	early mobilization	3
11	M	LEFT	Yes	Yes	-	Negative	< 72 hours	< 72 hours	none	early mobilization	4
12	M	RIGHT	Yes	Yes	-	Negative	< 72 hours	< 72 hours	none	early mobilization	3
13	M	RIGHT	Yes	Yes	-	Negative	< 24 hours	< 24 hours	none	early mobilization	5
14	M	LEFT	Yes	Yes	-	Negative	< 72 hours	< 72 hours	none	early mobilization	3
15	M	LEFT	Yes	Yes	-	Negative	< 72 hours	< 72 hours	none	early mobilization	3

Table 2 vHIT SHIMPs data for Vestibular Neuritis patients

Patient	Ipsilesional mean VOR gain	Contralesional VOR gain	Proportion of head impulses with covert saccades, Ipsi	Proportion of head impulses with covert saccades, Contra	time of final evaluation (days)	DHI
1						
T0	0.31	0.58	2	100		62
T1	0.32	0.64	100	100	30	16
2						
T0	0.31	0.87	0	100		84
T1	0.50	0.85	92	100	60	12
3						
T0	0.55	0.81	0	100		58
T1	0.50	0.85	74	100	30	20
4						
T0	0.16	0.87	0	100		56
T1	0.25	0.91	67	100	41	20
5						
T0	0.17	0.91	12	100		78
T1	0.35	0.85	100	100	280	18
6						
T0	0.32	0.75	0	100		74
T1	0.54	0.63	100	100	40	16
7						
T0	0.12	0.81	10	100		38
T1	0.28	0.76	95	100	50	14
8						
T0	0.24	0.86	0	100		68
T1	0.36	0.81	100	100	50	14
9						
T0	0.32	0.70	22	100		58
T1	0.49	0.77	100	100	50	18
10						
T0	0.40	0.86	0	100		32
T1	0.48	0.98	65	100	30	28
11						
T0	0.37	0.85	0	100		72
T1	0.38	0.99	98	199	60	10
12						
T0	0.28	0.61	15	100		84
T1	0.32	0.59	80	100	60	28
13						
T0	0.26	0.81	0	100		72
T1	0.32	0.87	84	100	180	12
14						
T0	0.32	0.90	2	100		82
T1	0.26	0.94	53	100	150	28
15						
T0	0.51	0.71	0	100		74
T1	0.56	0.72	37	100	60	22