Review Article

Perverted Compensatory Saccades After Head Impulses in Superior Vestibular Neuritis. Current Limits and New Perspectives For VHIT

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Abstract

Objectives: The Video Head Impulse Test (VHIT) was performed on patients with Superior Vestibular Neuritis (SVN) to detect refixation saccades that are not coplanar with the cephalic movement, known as Perverted Compensatory Saccades (PCS). Methods: We conducted a retrospective review of VHIT tests for SVN cases during the acute phase and in stabilized outcomes from 2021 to 2024. All video sequences were analysed in slow motion to verify the direction and size of compensatory saccades following cephalic impulses.

Results: Thirty-seven SVN cases, aged between 37 and 86 years, were enrolled. PCS were identified in 29 patients (78.4%). The results were analyzed using complex response models from the existing literature, accounting for both the simultaneous diffusion of kinetic energy and the static imbalance between the two labyrinths.

Conclusions: In SVN, a very high percentage of cases display saccades that are not coplanar with the cephalic impulse. It is suggested that PCS in the early stages of SVN mainly result from a static imbalance between the labyrinths. This imbalance is influenced by the activity of all semicircular canals involved in the kinetic stimulus that triggers eye movements during later stages of the disease. This study highlights that the VHIT software ignores non-coplanar eye movements and their gain, or miscalculates the reflex component, resulting in a pseudo-deficit that precludes a full evaluation of VHIT results.

Keywords: Video Head Impulse Test (VHIT), Superior Vestibular Neuritis, Vestibular-Ocular Reflex (VOR) anomalies, Perverted Compensatory Saccade

Abbreviations: IL-ASC: Ipsi-lateral Anterior Semicircular Canal; IL-PSC: Ipsi-lateral Posterior Semicircular Canal; IL-LSC: Ipsi-lateral Lateral Semicircular Canal; CL-ASC: Contra-lateral Anterior Semicircular Canal; CL- PSC: Contra-lateral Posterior Semicircular Canal; CL-LSC: Contra-lateral Lateral Semicircular Canal; VPCS: Vertical Perverted Compensatory Saccade; HPCS: Horizontal Perverted Compensatory Saccade; RPCS: Rotatory Perverted Compensatory Saccade VCS: Vertical Compensatory Saccade; HCS: Horizontal Compensatory Saccade; RCS: Rotatory Compensatory Saccade; AQ: AQEM (Anti-Compensatory Quick Eye Movement); U/D: up-beating (UB)/ down-beating (DB); Ipsi-lateral: (IL); Contra-lateral (CL); °: amplitude of saccade (1-3); A: Acute; SA: Sub-acute; EX: Outcomes; VHIT: Video Head Impulse Test; SVN: Superior Vestibular Neuritis; SC: Semicircular Canal; VOR: Vestibular-Ocular Reflex; ny: nystagmus; A: Acute; SA: Subacute; EX: Outcomes; PCS: Perverted Compensatory Saccades; IL: Ipsilesional; CL: Contralesional; LSC: Lateral Semicircular Canal; ASC: Anterior Semicircular Canal PSC: Posterior Semicircular Canal; VCS: Vertical Compensatory Saccades; HCS: Horizontal Compensatory Saccades; VPCS: Vertical Perverted Compensatory Saccades; HPCS: Horizontal Perverted Compensatory Saccades; RPCS: Rotatory Perverted Compensatory Saccades; UB: Up-Beating; DB: Down-Beating; AQ: AQEMs (Anticompensatory Quick Eye Movement); SI: Static Imbalance; NCDI: Non-Coplanar Dynamic Interference; CDI: Coplanar Dynamic Interference.

Introduction

Dysfunction of a semicircular canal is expressed during rapid head movements coplanar with it, with a decrease in reflexive eye-movement velocity and the appearance of refixation saccades. A positive head impulse test is a key diagnostic criterion in Superior Vestibular Neuritis (SVN), both in the acute and follow-up stages[1]. The Video Head Impulse Test (VHIT) is a diagnostic procedure that has become a routine part of daily clinical practice for assessing vestibular damage. It evaluates the impulsive Vestibular-Ocular Reflex (VOR) by measuring the relative velocities of the eye and head and recording catch-up saccadic movements [2].

SVN is a condition that causes targeted functional damage to the Anterior Semicircular Canal (ASC), Lateral Semicircular Canal (LSC), and part of the utricle. Its diagnosis depends on clinical evidence,

with VHIT proving particularly useful for confirming functional deficits in the ASC and LSC. The software of the VHIT systems calculates the relative velocities of the head and eyes only within the plane of the cephalic impulse-horizontal for the LSC, vertical for the ASC and the Posterior (PSC)-and does not assess eye movements outside the tested plane. Some VHIT systems, besides automatically generating numerical data, record the entire high-resolution video sequence of each cephalic impulse, including rejected ones. This footage can then be reviewed in slow motion by the operator. Such direct review is invaluable for removing artefacts and manually adjusting pupil position when automatic detection is inaccurate. Slow-motion observation also helps determine whether the reflex movement of the eyes occurs solely in the cephalic movement plane or includes components in other planes that the software may not recognize. According to current literature, all abnormal eye movements that do not align with the plane of the stimulated canal are termed perverted (from the Latin word «pervertere»: to flip, to overturn) and are regarded as abnormalities caused by brain lesions, especially in the cerebellum [3].

Perverted Compensatory Saccades (PCS) have so far been described only in the context of neurological pathologies [4-6]. It must be recognized that the SCs do not lie perfectly on a single plane but have portions inclined in different directions. Consequently, each cephalic movement simultaneously stimulates other SCs alongside the one in which the cephalic impulsive movement occurs. This multi-channel stimulus, negligible at low speeds, becomes significant in the VOR response at higher speeds. [7,8]. Based on anatomical (first) and radiological (second) studies, developed a mathematical model of the labyrinth's response, in which they calculated the relative involvement of each SC for each type of cephalic movement. These matrices explain how the vertical components of compensatory saccades can be observed after a horizontal cephalic impulse in the SVN and selective deficits of the PSC, as previously described in our articles (in which we did not define them as PCS) [7-10].

In this paper, we examined non-coplanar eye movements in response to stimulation of each semicircular canal. We categorized the observed saccadic responses into three spatial planes.

Slow-motion analysis of the VHIT recordings (showing both cephalic and ocular movements) enabled us to identify PCS in most cases (78.4%).

We compared our results with the labyrinthine response models to cephalic impulses developed by the Authors mentioned above, and we also considered the effect of tonic imbalance between the two labyrinths and the position of the eyes in the orbit.

Materials and Methods

All video sequences recorded during VHIT examinations of 37 patients with SVN, examined between 2021 and 2024, were reviewed in slow motion.

The diagnosis of SVN has been based on clinical assessment, observation of spontaneous nystagmus (ny) that does not change direction with different head positions, and the absence of new auditory or neurological symptoms, such as altered consciousness, headache, incoordination, dysarthria, or diplopia. Patients with a history of neurological disease were excluded.

All patients were assessed using the HINTS and STANDING protocols (searching for gaze nystagmus (Ny), a covert test for skew deviation, head impulse test, and standing evaluation) to exclude possible central vertigo. Eye movements (pursuit and saccade) were examined in both horizontal and vertical planes. Gaze and rebound Ny were observed under fixation. Spontaneous and positional Ny, as well as Head Shaking Ny, were evaluated with Frenzel glasses. All patients could stand upright (with assistance). Face mobility, upper limb strength, and coordination were also examined [11,12]. All patients underwent VHIT in a seated position on each SC plane using the Synapsys VHIT system (Inventis SRL, Padova, Italy). We selected only patients who presented with a unilateral deficit in both the anterior (gain < 0.7) and lateral (gain < 0.8) SCs 2,[13]. Antecedent SVN was also diagnosed in patients with a previous clinical diagnosis who later underwent VHIT and still showed a deficit in the ASC and LSC on one side, even though spontaneous nystagmus was no longer observed at the time of the instrumental test. The slow-motion review of all video sequences from each examination allowed us to verify the direction and extent of the compensatory saccades following the cephalic impulse, and to categorize the rapid movement observed into three planes. Only responses that were incongruent and confirmed in more than half of the sequences recorded for each stimulated channel were considered.

The horizontal direction of the saccade was reported with reference to the side of the lesioned labyrinth and therefore indicated as Ipsi-Lesioned (IL) or Contra-Lesioned (CL).

The direction of the torsional component was determined by observing the movement of the upper segment of the iris relative to the observer (clockwise if it beats towards the patient's left and vice versa). In this way, for a right deficit, a clockwise torsional movement is indicated as CL. The vertical component is explicitly described as up beating (UB) or down beating (DB).The amplitude of the saccadic movement was coded as 1 if it was less than 3°, 2 if it was between 3 and 6°, and 3 if it was greater than 6°. The reported evaluation is the average of the observations. Patients were examined at different times from symptom onset and were therefore divided into three groups: Acute (A), if seen within the first 7 days; Sub-Acute (SA), if seen between the 8th and 30th day; and Outcomes (EX), if seen later.

Results

Thirty-seven cases of SVN were enrolled, comprising 25 males and 12 females, aged 37-86 years (mean, 63.4). They were categorized as follows: 13 A, 8 SA, 16 EX.

All patients exhibited compensatory saccades consistent with the stimulation plan (HCS: Horizontal Compensatory Saccades; VCS: Vertical Compensatory Saccades).

In 29 patients (78.4%), saccadic refixation movements were also observed on planes other than the stimulated one (HPCS:

Horizontal Perverted Compensatory Saccades; VPCS: Vertical Perverted Compensatory Saccades; RPCS: Rotatory Perverted Compensatory Saccades).

Planar incongruences of reflex eye movement have been observed both in isolated form, that is, in place of the expected movement for that plane, and in conjunction with the expected movement. When combined, the movement may appear oblique (resulting from the superposition of linear components) or arched
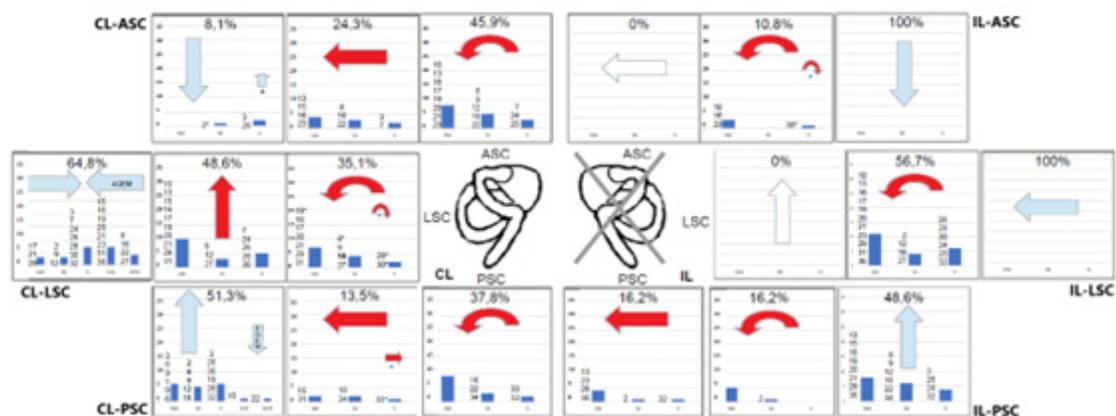
(due to the superposition of torsional components).

Table 1 presents, for each patient, the VOR gain values and an analysis of each directional component of the compensatory saccades (H: Horizontal; V: Vertical, R: Rotatory) for each semicircular canal examined.

The overall results are shown graphically in Figure 1, where, for each semicircular canal examined, the observed saccadic components are displayed in the three planes of space.

Table 1: Table describes the VHIT gain and the perverted compensatory saccades found in all semicircular canals, both ipsilesional and contralesional. IL-ASC: Ipsilesional Anterior Semicircular Canal; IL-PSC: Ipsilesional Posterior Semicircular Canal; IL-LSC: Ipsilesional Lateral Semicircular Canal; CL-ASC: Contralesional Anterior Semicircular Canal; CL-PSC: Contralesional Posterior Semicircular Canal; CL-LSC: Contralesional Lateral Semicircular Canal; PCS: Perverted Compensatory Saccades; VPCS: Vertical Perverted Compensatory Saccades; HPCS: Horizontal Perverted Compensatory Saccades; RPCS: Rotatory Perverted Compensatory Saccades; VCS: Vertical Compensatory Saccades; HCS: Horizontal Compensatory Saccades; AQ: AQEM (Anti-Compensatory Quick Eye Movements); U/D: up-beating (UB)/ down-beating (DB); I/C: Ipsilesional (IL)/ Contralesional (CL); °: amplitude of saccade (1-3); A: Acute; SA: Sub-Acute; EX: Outcomes.

Figure 1: Representation of the data collected in (Table 1). CL-ASC: Contralesional Anterior Semicircular Canal, CL-PSC: Contralesional Posterior Semicircular Canal, CL-LSC: Contralesional Lateral Semicircular Canal, IL-ASC: Ipsilesional Anterior Semicircular Canal, IL-PSC: Ipsilesional Posterior Semicircular Canal, IL-LSC: Ipsilesional Lateral Semicircular Canal. The labyrinths are indicated as Ipsilesional (IL) (marked by a cross) or Contralesional (CL). The ASCs' results are displayed on the top row, those of LCSs in the middle row and those of the PSCs on the lower row. For each examined SC, the observed saccadic components are reported, divided into the three planes of space, with graphic indications of the direction. The coplanar responses are in light blue, the non-coplanar in red. The recurrence of each saccadic component is expressed as a percentage of the total number of patients and divided into subgroups (histograms) according to the phase of the disease: acute (A), subacute (SA), and outcomes (EX). Each subgroup is associated with a list of patients, identified by the number reported in the (Table 1). On the CL-LSC and CL-PSC, cases presenting an anti-compensatory direction are indicated as AQEM (Anticompensatory Quick Eye Movement). The histograms of the relative subgroups are reported correspondingly and are labelled as AcAQ for the acute AQEM and SubAQ for the subacute AQEM. On the other SCs the anticompensatory saccades are identified with an asterisk.

Healthy LSC

Planar incongruences were identified on the LSC of the healthy side in 20 patients (10A, 5SA, 5EX): 9 cases were isolated (7 VPCS and 2 RPCS), and 11 cases were associated (VPCS + RPCS).

18 VPCS, all directed upwards (10A, 3SA, 5EX); 7 isolated (3A, 1SA, 3EX), and 11 associated with RPCS (7A, 2SA, 2EX). Of these, eight are directed contralaterally (7A, 1SA), and three ipsilaterally (2EX, 1SA).

There are 13 RPCS (7A, 4SA, 2EX), with 9 directed towards the healthy side (6A, 3SA) and 4 towards the pathological side (1A, 1SA, 2EX). Additionally, 2 are isolated (2SA), with one directed contralaterally and one ipsilaterally; and 11 are associated with VPCS (7A, 2sa, 2ex).

24 HCS 13 with compensatory direction (3A, 3 SA, 7 EX) and 11 AQEM (Anticompensatory Quick Eye Movement), (7A; 4 SA) 14,15.

Healthy ASC

On the ASC of the healthy side, PCS were observed in 18 patients (A, 5SA, 4EX): 11 were isolated (2 HPCS and 9 RPCS), and seven were associated (HPCS + RPCS).

There are 16 RPCS, all directed towards the healthy side (8A, 5SA, 3EX), with 9 in isolated form (5A, 2SA, 2EX) and 7 associated with HPCS (3A, 3SA, 1EX).

9 HPCS: all directed towards the healthy side (4A, 3SA, 2EX), of which 2 are isolated (1A, 1EX) and 7 are associated with RPCS (3A, 3SA, 1EX).

3 VCS, 2 with compensatory direction (2EX), and 1 with anti-compensatory direction (1SA).

PSC

On the PSC of the healthy side, PCS were identified in 15 patients (9A, 3SA, 3EX): 11 were isolated (including 10 RPCS and 1 HPCS), and 4 were associated (HPCS + RPCS).

14 RPCS: all directed towards the healthy side (9A, 3SA, 2EX); of which 10 were isolated (7A, 1SA, 2EX) and 4 were associated with HPCS (2A, 2SA).

5 HPCS: 4 directed towards the healthy side (2A, 2SA), always associated with RPCS directed towards the healthy side; 1 isolated (1EX) directed towards the diseased side.

19 VCS: 17 with compensatory direction (6A, 5 SA, 6 EX) and 2 AQEM (1A, 1SA) [14,15].

Pathological PSC

On the PSC of the injured side, PCS were identified in 8 patients (6A, 1SA, 1EX), of which four were isolated (2 HPCS and 2 RPCS), and four were associated (HPCS + RPCS).

6 HPCS always directed towards the healthy side (4A, 1SA, 1EX) of which 2 were isolated (1A, 1EX) and 4 were associated with RPCS (3A, 1SA).

Six RPCS, always directed towards the healthy side (5A, 1SA); of these, two were isolated (2A), and four were associated with HPCS (3A, 1SA).

18 VCS (8A, 6SA, 4EX) all directed in a compensatory direction (UB).

Pathological LSC

On the LSC of the injured side, PCS were identified in 21 patients, all of whom had RCPS (11A, 4SA, 6EX), which were always directed towards the healthy side; directional components on the vertical plane were never observed. In all cases, as required by the study's inclusion criteria, an HCS was identified.

Pathological ASC

On the ASC of the affected side, PCS were identified in four patients, all having RPCS (3A, 1EX). Three of these pointed towards the healthy side (3A), and one towards the pathological side (1EX). Horizontal directional components were never observed. In all cases, as required by the study's inclusion criteria, VCS was present.

Overall Results

Out of the 74 identified RPCS (44A, 16SA, 14EX), 69 were directed towards the healthy side (43A, 15 SA, 11 EX); only in 5 cases where they directed towards the injured side: 4 on the CL-LSC (1A, 1SA, 2EX) and one on the IL-ASC (1EX). 48 RPCS (29A, 8SA, 11EX) were isolated and 26 (15A, 8SA, 3EX) were associated with other PCS.

The 18 VPCS (10A, 3SA, 5EX) were found only on the CL-LSC (see the related paragraph).

The 20 HPCS (10A, 6SA, 4EX) were observed on all vertical SCs, except on the IL-ASC. Nineteen were directed contralesionally; one was directed ipsilesionally (1EX on CL-PSC, not associated with RPCS). Fifteen (8A, 6SA, 1EX) were linked to RPCS that consistently beat contralesionally, and five were isolated (2A, 3EX).

Discussion

In patients with SVN, during an impulsive cephalic movement, the eyes are influenced by multiple intercurrent factors: the orbital tensile-elastic mechanisms, the tonic deviation caused by the static imbalance between the two vestibules (slow phase of spontaneous ny), and the action of all the stimulated SCs.

The resulting eye movement is therefore complex and arises from the interaction of the mechanisms mentioned earlier, which differ with the specific direction of cephalic movement and vary across different phases of the disease. The final motor effect of these factors must also consider the position of the examined eye in the orbit; this is especially important when assessing vertical SCs, as the examined eye is always in an abducted position in these cases.

On the abducted eye, the action of the superior and inferior rectus muscles becomes purely vertical. Here, the effects of the ipsilateral ASC and the PSC on the opposite side— which have direct projections onto the ipsilateral superior rectus muscle and the

contralateral inferior rectus, respectively— are better evaluated; conversely, the rotational components of the ASC on the opposite side and the ipsilateral PSC are more observable [16].

1)   Orbital tensile-elastic mechanisms

The initial eye movement in response to a cephalic impulsive stimulus is Anticompensatory: the eyes move in the same direction as the head. This movement occurs without delay and is completed in approximately 10 milliseconds; the absence of delay indicates a mechanical origin of the movement, not a reflex, related to orbital tensile-elastic forces (due to the eyeballs' eccentric position relative to the head's center of rotation) [17].

This action diminishes the efficiency of the VOR in cephalic impulses towards the healthy side; conversely, on the affected side, the initial anti-compensatory movement combines with the slow phase of the spontaneous nystagmus. For the evaluation that follows, this component will be excluded, as it is constant across all responses.

2)   Static Imbalance (SI)

During SVN, the relative tonic imbalance of the labyrinths causes an ipsilateral horizontal and downward vertical eye deviation, along with an ipsilateral torsional eye rotation.

SI occurs on all tested SCs and is greater with more recent

damage; therefore, its reduced effect can be expected in the outcomes of SVN when functional compensation is more developed.

Because of the abducted position of the tested eye, SI-related rotational components are more apparent when testing the CL-ASC and IL-PSC, and less so when testing the IL-ASC and CL-PSC.

SI-related horizontal components, in accordance with Alexander's law (peripheral nystagmus appears wider when gaze is directed towards the healthy side), will be more noticeable when assessing the CL-ASC and IL-PSC; conversely, they will be less evident on the IL-ASC and CL-PSC, as previously observed for rotational components.

3)   Action of SCs non-coplanar to cephalic movement (Non-Coplanar Dynamic Interference: NCDI).
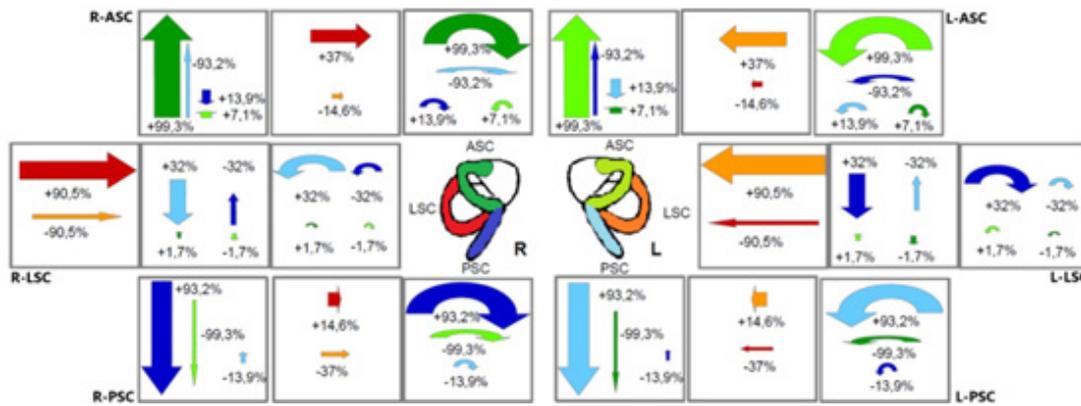
For any given head rotation, the acceleration detected by a specific SC is determined by the geometric projection of that SC onto the plane of rotation of the head [18].

Figures 2 and 3 visually present the response matrices of the labyrinths under normal conditions, as predicted by the models mentioned above, which differ in certain vector components. [7,8]. When comparing these matrices and the results of this study shown in Figure 1, it is essential to remember that the vectors in Figures 2-5 represent the reflex eye movement before the compensatory saccade, which occurs in the opposite direction.
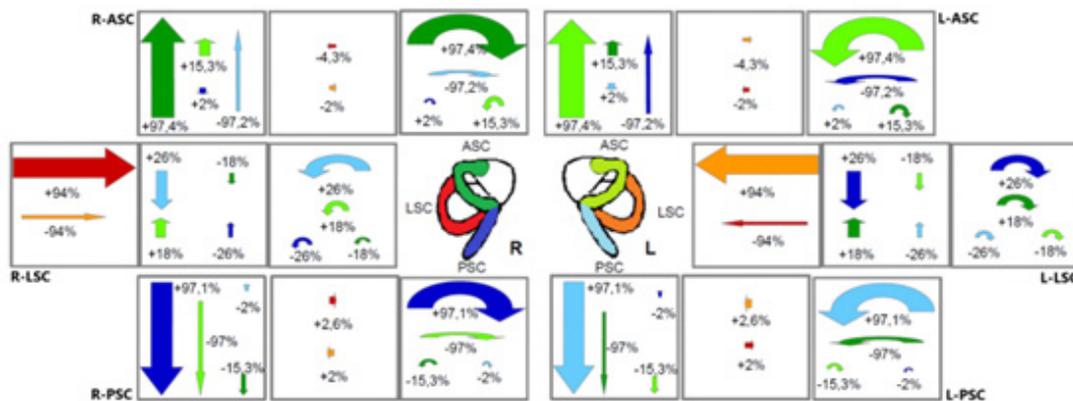
Figure 2: Representation of the response matrix of all semicircular canals in the normal subject, derived from *Della Santina*, et al.

The ocular response vectors obtained during the ASC tests are displayed on the top row, the LCS ones on the middle row, and the PSC ones on the bottom row. The vectors indicate the direction of the eye movement caused by the excitatory (wider arrows, +) and inhibitory (thinner arrows, -) stimulus of each semicircular canal, differentiated by colour. The length of the vectors expresses the intensity of the action as a function of the percentage (reported alongside) of kinetic energy transduced by each canal for a cephalic impulse imparted on the horizontal plane (for testing the LSC) and on the oblique planes (for testing the ASC and PSC). R: right; L: left; R-ASC: Right Anterior Semicircular Canal (dark green); R-PSC: Right Posterior Semicircular Canal (dark blue); R-LSC: Right Lateral Semicircular Canal (red); L-ASC: Left Anterior Semicircular Canal (light green); L-PSC: Left Posterior Semicircular Canal.
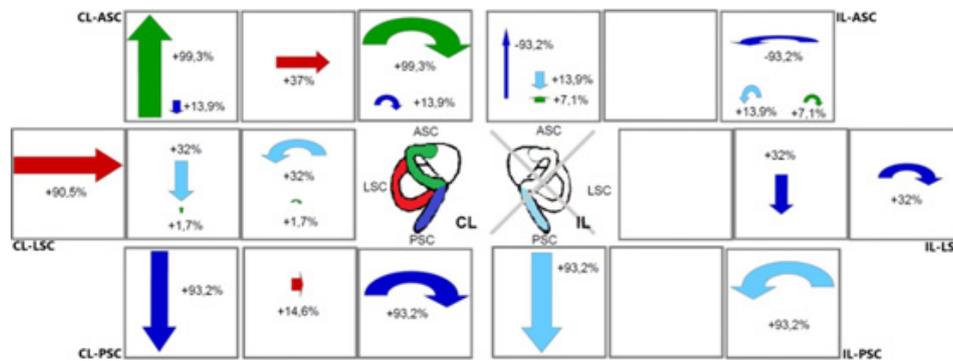
Figure 3: Representation of the response matrix of all semicircular canals in the normal subject, derived from *Cremer,* et al.

The ocular response vectors obtained during the ASC tests are displayed on the top row, the LCS ones on the middle row, and the PSC ones on the bottom row. The vectors indicate the direction of the eye movement caused by the excitatory (wider arrows, +) and inhibitory (thinner arrows, -) stimulus of each semicircular canal, differentiated by colour. The length of the vectors expresses the intensity of the action as a function of the percentage (reported alongside) of kinetic energy transduced by each canal for a cephalic impulse imparted on the horizontal plane (for testing the LSC) and on the oblique planes (for testing the ASC and PSC). R: right; L: left; R-ASC: Right Anterior Semicircular Canal (dark green); R-PSC: Right Posterior Semicircular Canal (dark blue); R-LSC: Right Lateral Semicircular Canal (red); L-ASC: Left Anterior Semicircular C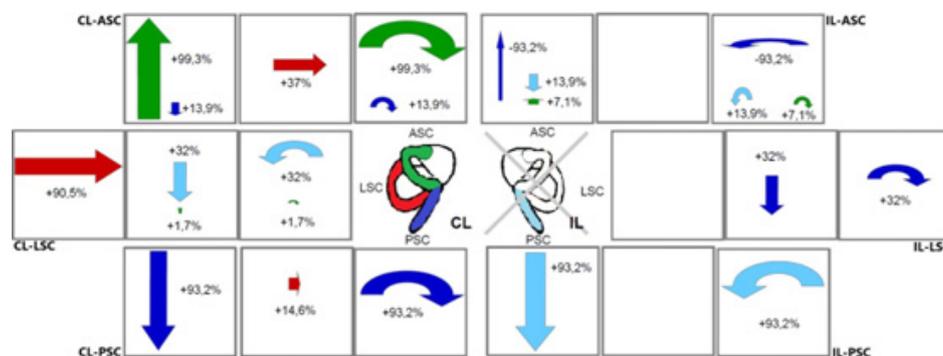anal (light green); L-PSC: Left Posterior Semicircular Canal (light blue); L-LSC: Left Lateral Semicircular Canal (orange).

Figure 4: Representation of the response matrix of all semicircular canals in the Superior Vestibular Neuritis, derived from Della Santina et al. The ocular response vectors obtained during the ASC tests are displayed on the top row, the LCS ones on the middle row, and the PSC ones on the bottom row. The vectors generated by the damaged CS are absent. The labyrinths are indicated as Ipsilesional (IL) or Contralesional (CL). Only the excitatory vectors are reported, except for the IL-ASC (see text). The vectors indicate the direction of the eye movement caused by the excitatory (wider arrows, +) and inhibitory (thinner arrows, -) stimulus of each semicircular canal, differentiated by colour. The length of the vectors expresses the intensity of the action as a percentage (reported alongside) of kinetic energy transduced by each channel for a cephalic impulse imparted on the horizontal plane (for testing the LSC) and on the oblique planes (for testing the ASC and PSC). CL-ASC: Contralesional Anterior Semicircular Canal (dark green); CL-PSC: Contralesional Posterior Semicircular Canal (dark blue); CL-LSC: Contralesional Lateral Semicircular Canal (red); IL-PSC: Ipsilesional Posterior Semicircular Canal (light blue).

Figure 5: Representation of the response matrix of all semicircular canals in the Superior Vestibular Neuritis, derived from Cremer et al. The ocular response vectors obtained during the ASC tests are displayed on the top row, the LCS ones on the middle row, and the PSC ones on the bottom row. The vectors generated by the damaged CS are absent. The labyrinths are indicated as Ipsilesional (IL) or Contralesional (CL). Only the excitatory vectors are reported, except for the IL-ASC (see text). The vectors indicate the direction of the eye movement caused by the excitatory (wider arrows, +) and inhibitory (thinner arrows, -) stimulus of each semicircular canal, differentiated by colour. The length of the vectors expresses the intensity of the action as a percentage (reported alongside) of kinetic energy transduced by each channel for a cephalic impulse imparted on the horizontal plane (for testing the LSC) and on the oblique planes (for testing the ASC and PSC). CL-ASC: Contralesional Anterior Semicircular Canal (dark green); CL-PSC: Contralesional Posterior Semicircular Canal (dark blue); CL-LSC: Contralesional Lateral Semicircular Canal (red); IL-PSC: Ipsilesional Posterior Semicircular Canal (light blue).

Based on the model derived from [8]. There are no significant stimulations of the LSC in the cephalic impulses on the oblique planes when testing the ASC and PSC. At the same time, both vertical SCs participate in horizontal cephalic impulses. According to the model of [7]. During impulses on the oblique planes, there would be significant stimulation of the ipsilateral LSC, especially in the "nose down" movements (testing the ASC); additionally, on horizontal cephalic movements, only the contralateral PSC would be stimulated, without significant contributions from the ASC.

In a cephalic impulsive test, in addition to the effect of the SC on which the cephalic stimulus occurs, the effect of non-coplanar stimulation of the other SCs must also be considered, according to the response matrices indicated above.

In healthy subjects, head movement on the horizontal plane, which is not exactly coplanar with the LSCs (tilted 30° relative to them), not only involves the lateral SCs but also stimulates the vertical SCs on the opposite side of the head movement. A reflex occurs with opposing vertical components (according to the Della Santina model only), stabilizing the eyes on the vertical plane. Torsional components in the same direction are also generated, which combine (according to both models) and cause the eyes to torsion by about 5° towards the stimulated side. The effect of this ocular torsion is negligible because it does not produce significant foveal retinal slip [19,20].

If the horizontal head movement were to occur on a plane inclined 30° downwards (nose down), no further planar ocular response anomalies would be observed because the vertical SCs would no longer be stimulated [4].

In cases where one or more SCs have a functional deficit, head movements produce an unbalanced effect resulting from the combined influence of all the active semicircular canals.

In slow cephalic movements, NCDI has no distorting effect on the plane in which the eye movement occurs, and even in cases of lesion of one or more SCs, there are no significant consequences; on the contrary, during impulsive cephalic movements, NCDI becomes important and produces reflex eye movements on the planes of the "improperly" stimulated SCs.

To highlight the effects of NCDI in patients with SVN, Figures 4-5 show the same matrices mentioned earlier, in which the vectors related to the damaged SCs are no longer present.

The impulsive movement triggers a reflex that is the main expression of excitation alone; therefore, only this vector is reported for the functioning channels, not the inhibitory ones.

On the horizontal plane, the operator can easily control cephalic impulses at speeds exceeding 150°/s, ensuring complete saturation of the inhibitory components; therefore, these are not displayed in Figures 4 and 5 on the IL-LSC.

On oblique planes, in clinical practice, most cephalic impulses have velocities below 150°/s; this does not ensure complete saturation of the inhibited channel. On the plane of a damaged vertical SC (lacking excitatory components), the contralateral functioning coplanar canal can significantly contribute to the VOR; therefore, in Figures 4 and 5, on the IL-ASC, the vector caused by the inhibition of its coplanar channel (CL-PSC) is shown [21].

4) Action of SCs coplanar to cephalic movement (Coplanar Dynamic Interference: CDI)

When testing vertical SCs, it must be recognised that their stimulation physiologically causes a reflex eye movement with both vertical and rotational components. Since the eye is abducted, the torsional component of the reflex is suppressed; however, because the exact eye position is not always perfectly achieved in clinical practice, this rotational effect should be considered to some extent.

It follows that a torsional CDI vector must be considered, as it adds to the SI on the CL-ASC and CL-PSC and opposes it in the IL-ASC and IL-PSC.

From the total of dynamic and static interferences, summarised in Figure 6, we can attempt to interpret the results of our study. We shall now analyse for each SC the balance of the above factors.
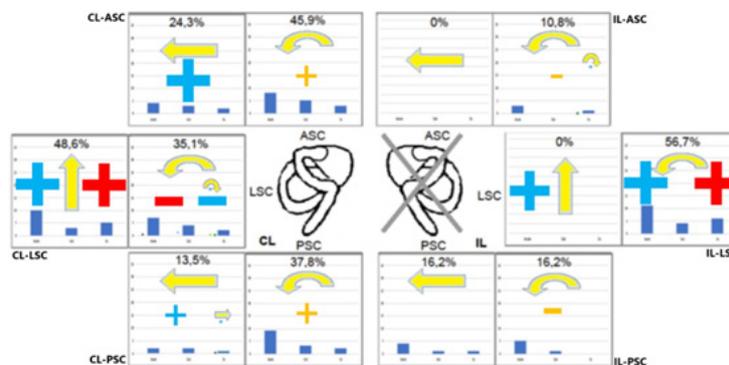
Figure 6: The figure illustrates the interactions between the pathophysiological conditions that cause PCS. The labyrinths are indicated as Ipsilesional (IL) or Contralesional (CL). The perverted saccades (yellow arrows) found testing the ASCs are displayed on the top row, those of the LCSs on the middle row and those of the PSCs on the lower row. The number of perverted compensatory saccades found is expressed as a percentage of the total number of patients. The plus sign (+) indicates that the static and dynamic interference vectors are added, and the minus sign (-) indicates that the static and dynamic interference vectors are subtracted. Non-Coplanar Dynamic Interaction is shown in blue according to Cremer et al., in red according to (8) and the Coplanar Dynamic Interaction in orange. The thickness of the signs indicates the intensity of the expected effect.

A. Ipsilesional-Anterior Semicircular Canal

On this channel, the vertical DB saccade, expected due to the canal deficit, is observed almost exclusively; no horizontal components are seen, and rotational components are infrequently observed (in 4 patients).

The ASC is tested by examining the eye on the same side as the damaged labyrinth. In this eye, the ASC connects only to the superior rectus muscle, with no projections to the oblique muscles; therefore, rotational components caused by the SI are not expected [16].

Furthermore, when observing through the examined eye in the abducted position, the excursion of the tonic imbalance's torsional and horizontal components is also diminished. These considerations explain why incongruent saccades are rarely observed during examination of the damaged ASC.

Finally, it is important to consider the effect of the CDI, arising from the inhibitory action of the healthy PSC (which is not negligible in this case), which has a torsional effect opposite to that of the SI. In a patient with late-stage SVN, a torsional saccade directed towards the lesion was observed; this can be explained by CDI.

B. Ipsilesional - Posterior Semicircular Canal

In this case, NCDI vectors are absent, and PCS are scarce (8 patients), with all but one recorded in the acute and subacute phases. This suggests these are caused solely by the SI. The rotatory SI component is diminished by the CDI, whose vector here points in the opposite direction.

On this channel, PCS tend to occur only when the tonic imbalance is very severe: in 5 out of 8 cases, a significant deficit of the damaged ASC (responsible for the rotary component of the SI) is observed, with negative gain (-0.04).

The effect of the CDI is clear, as the number of RPCS is much lower than on the CL-PSC, where the SI and the CDI are combined. Despite the tested eye being positioned abducted towards the injured side, which makes the rotational components more noticeable on this side than on the contralesional one.

HPCS are an expression of SI only; no significant dynamic interaction vectors are expected in this case.

The high number of VCS on the PSCs of both sides is linked to the relatively advanced average age of the sample (63.4), suggesting a physiological deficit in these channels [22,23].

C. Ipsilesional - Lateral Semicircular Canal

On the LSC of the damaged side, no VPCS were observed. In addition to the expected large HCS, a very common RPCS (56.7%) was detected, consistently directed toward the healthy side.

As shown in Figure 4, on the IL-LSC, the NCDI involves the action of both contralateral vertical SCs, which stabilise the eyes in the vertical plane (no VPCS were found), while producing a rotation of the eyes towards the injured side Figures 4 and 5 that adds to the torsional vector of the SI.

The combined total of these two components (SI + NCDI) accounts for the high number of RPCS directed toward the healthy side observed in the IL-LSC.

Here, RPCS often occur very early, usually before the end of the cephalic movement (covert); later saccades (overt), isolated or following a first RPCS, are predominantly horizontal (HCS).

D. Contralateral - Anterior Semicircular Canal

NCDI, according to the *Cremer, et al*. model Figure 5, predicts that "nose down" movements on oblique planes would produce an excitatory effect on the ipsilateral LSC equal to 37% of the kinetic energy applied on that plane. This would result in a reflex eye movement with a consistent horizontal component towards the injured side.

The relatively high prevalence of HPCS (24.3%), always directed contralaterally to the lesion, may result from the combined effect of the SI and NCDI vectors.

RPCS are quite common (45.9%), which is supported by the sum of the static vector and the CDI.

It must also be considered that this SC is tested by evaluating the eye abducted towards the healthy side, where both the rotary and horizontal saccadic components are more readily observed.

E. Contralesional - Posterior Semicircular Canal

The relatively high proportion of RPCS (37.8%) results from the combined total of the SI and the CDI; however, their percentage is lower than that observed on the CL-ASC because the position of the examined eye (abducted towards the injured side) renders them less visible.

Regarding the NCDI, according to [7]. Figure 5, there is also a slight (+14.6%) stimulation of the healthy LSC during nose-up movements; this component is added to the SI. However, the number of HPCS observed here is slightly lower than that seen for the IL-PSC, where this vector is not expected. When evaluating these data, it is also important to consider the abducted position of the tested eye, which reduces the visibility of the horizontal saccades.

F. Contralesional - Lateral Semicircular Canals

VPCS were observed only on the CL-LSC, and all are directed upwards; they are linked with RPCS in 11 cases. The rotational components are oriented towards both the healthy and the damaged sides; in 7 cases, the VPCS are isolated.

VPCS tend to persist more frequently in the late stages of the disease (5/15) than RPCS (2/15, both directed towards the injured side, therefore not attributable to SI). The constant UB direction of VPCS contrasts with the different direction of the RPCS associated with them, suggesting that the vertical and rotational movements imply different pathophysiological mechanisms.

The vertical UB component on the CL-LSC can be explained as a result of the unbalanced input from the vertical SCs on the sick side, which are stimulated by an impulse on the horizontal plane directed towards the healthy side Figure 4 [9].

The same mechanism, operating in the opposite direction to that described here, has also been observed in certain cerebellar pathologies (which involve reduced selective inhibition of the ASC) and in selective deficits of the PSC; in both cases, there is a functional dominance of the ASC over the PSC. During impulsive horizontal movements towards the opposite side, the eyes move upward, followed by a vertical DB saccade [4,5,6,10].

G.   Regarding RPCS

On the healthy LSC, the NCDI vector is opposite in direction to the SI, but it has a limited effect because it is expressed only by the PSC of the pathological side, lacking the contribution of the damaged ASC. However, this is sufficient to reduce the expression of the SI; in fact, the RPCS appear in 35.1% of cases, compared to 56.7% observed on the IL-LSC, where both opposite vertical SCs are active and combine their effects on the SI. Supporting this hypothesis, in the later stages of the disease, when the SI is no longer active, the RPCS found (2EX) are both directed towards the injured side due to the effect of NCDI alone.

H.   Rotatory Perverted Compensatory Saccades

Overall, the torsional components of saccades are observed in more SCs during the acute phase (mean 3.6) than they decrease over time (2.1 SA; 1.3 EX), indicating a direct relationship between the appearance of RPCS and the level of tonic imbalance between the labyrinths.

The number of RPCS detected increases when static and dynamic vectors are combined, especially in the IL-LSC (56.7%), the CL-ASC (45.9%), and the CL-PSC (37.8%).

In the late phase of the disease, RPCS continues to be directed toward the healthy side of the IL-LSC, CL-ASC, and CL-PSC, where both NCDI and CDI move the eyes in that direction. In contrast, they go toward the injured side on the CL-LSC and IL-ASC, where both NCDI and CDI have the opposite direction Figures 4 and 5.

I.   Horizontal Perverted Compensatory Saccades

According to the response matrix of [7]. Figure 4, a significant dynamic interference vector is expected in the horizontal plane, which, during CL-ASC testing, combines with the static vector, resulting in a higher detection of HPCS.

The close relationship between HPCS and RPCS, their agreement in direction, and their common findings, mainly in the early stage of the disease, suggest a shared pathophysiological mechanism, primarily related to the effect of SI; the influence of NCDI appears to be significant only for CL-ASC.

J.   Overall results

Patients suffering from acute SVN display four or more incongruent saccades simultaneously (average 5.25), whereas in the sub-acute stage most cases show three alterations (average 3.25), and in the outcomes, they are observed in a variable manner (from 1 to 5, average: 2.5). From this data, a close temporal correlation with the finding of PCS emerges, highlighting the importance of the SI in their development.

SI has a consistent effect across all test conditions, whereas dynamic interferences generate vectors that vary in direction and intensity for each semicircular canal tested.

The direction of the SI vector and those of dynamic interference (NCDI and CDI) can either add or contrast (Figure 6); specifically, they contribute to the CL-ASC for the horizontal and rotary components, to the CL-LSC for the vertical component, to the IL-LSC for the torsional component, and to the CL-PSC for both horizontal and torsional components. They contrast on the CL-LSC, on the IL-PSC, and on the IL-ASC for the torsional component.

In summary, when static and dynamic interferences conflict, the SI prevails. During the acute and subacute phases, out of a total of 35 PCS observed under contrast conditions, only 2 saccades with reverse direction are seen.

In late phases, when only dynamic interferences are present, significant effects of the inversion of the saccade direction are observed; in fact, in the contrast conditions, 4 out of 6 PCS beat towards the healthy side, and, in particular, all the RPCS found have an inverse direction.

In conclusion, it is suggested that perverted saccades in the acute stage of SVN mainly result from static imbalance between the labyrinths, with their expression being modulated by the action of all involved semicircular canals in response to the kinetic stimulus; this dynamic interference predominantly influences eye movements in the later stages of the disease.

Current VHIT systems assess eye movements only within the same plane as cephalic stimulation; therefore, they do not provide information about abnormal responses.

Perverted eye movements can be so pronounced that they disrupt accurate gain calculation, a condition regarded as pathological (pseudo-deficit) despite the tested SC functioning normally.

Sometimes, PCS are so severe that they cause the acquired sequence to be rejected from the gain calculations, making it difficult or impossible to complete the VHIT exam.

Current VHIT systems offer only a partial assessment of the impulsive VOR, which does not encompass all components of the complex ocular response. We lack information that may have clinical relevance and can currently be studied only through direct observation.

Conclusions

During the VHI test, in a very high percentage of SVN cases (78.4%), saccades are not coplanar with the cephalic impulse.

The ocular response to impulsive stimuli in SVN involves complex movements in all planes, not only in the plane of the cephalic stimulus; these movements result from the convergence of several factors: static imbalance, dynamic interferences (coplanar and non-coplanar), and the initial position of the eyes within the orbits.

When dynamic and static interferences are in opposition during the acute phase of SVN, the effects of static imbalance dominate, with their expression being modulated by dynamic canal interferences. In the later stages of the disease, PCS are solely attributable to dynamic canal interference. Sometimes, the perverted responses significantly disrupt the execution of the VHIT exam because the software rejects this data by default, making it challenging to complete the exam. Additionally, an error may occur in the calculation of the gain of the studied SC, which is incorrectly shown as deficient (pseudo-deficit).

The slow-motion visual examination of saccadic eye movements after a cephalic impulse enables a qualitative assessment of the complex oculomotor response. This method provides additional data to that generated by the VHIT software alone, which is solely dedicated to detecting the component of eye movement in the stimulated plane. The operator needs to know whether the reflex movement of the eye he is examining also occurs in other planes, so he can take this into account when assessing VHIT results. It is hoped that, in the future, VHIT software will automatically analyse eye movements across all planes to deliver a more comprehensive report of reflected eye movements.

Conflicts of Interest

The authors declare no conflicts of interest related to this study and no personal or financial relationships that could influence their work.

Authors' Contributions

All authors contributed equally to this work. F.D.O. wrote the main paper, L.N. reviewed the English, and G.N. reviewed the paper. All authors discussed the results and implications and commented on the manuscript at all stages.

Ethical Consideration

Generated Statement: Ethical approval was not required for the studies involving humans because patients underwent routine vestibular testing. The studies were conducted in accordance with local legislation and institutional requirements. Written informed consent for participation was not required from the participants or their legal guardians/next of kin in accordance with national legislation and institutional requirements, as patients underwent routine vestibular testing. The authors state that they have followed the Declaration of Helsinki, including obtaining informed consent from research participants. All the patients authorised us to use the data and video recordings for scientific purposes. The research was conducted ethically, with all study procedures being performed in accordance with the requirements of the World Medical Association's Declaration of Helsinki. Written informed consent was obtained from each participant/patient for study participation and data publication.

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