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Original article

Skull vibration induced nystagmus in patients with superior semicircular canal dehiscence

G. Dumas[a,d], H. Tan[b], L. Dumas[c], P. Perrin[d], A. Lion[e], S. Schmerber[a,f,*]

[a] Auditory Implants, Cochlear Implant Centre of the Alpes, Department of Oto-Rhino-Laryngology, Head and Neck Surgery, University Hospital Grenoble Alpes, 38700 La Tronche, France
[b] Department of Otolaryngology H&N surgery, University School of Medicine, Shanghai Ninth People's Hospital, Shanghaï Jiao Tong, 200011 Shanghaï, China
[c] Inserm S 1039 Bioclinic Radiopharmaceutics Laboratory, University Grenoble Alpes, 38700 La Tronche, France
[d] EA 3450 DevAH, Development, Adaptation and Handicap, Faculty of Medicine and UFR STAPS, University of Lorraine, 54600 Villers-lès-Nancy, France
[e] Sports Medicine Research Laboratory, Luxembourg Institute of Health, 1460 Luxembourg, Luxembourg
[f] Inserm BrainTec Lab UMR 1205, University Grenoble Alpes, CHU A. Michallon, BP 217, 38043 Grenoble cedex 09, France

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ABSTRACT

Objective: To establish optimum stimulus frequency and location of bone conducted vibration provoking a skull vibration induced nystagmus (SVIN) in superior semi-circular canal dehiscences.
Methods: SVIN 3D components in 40 patients with semi-circular canal dehiscence (27 unilateral and 13 bilateral) were compared with a group of 18 patients with severe unilateral vestibular loss and a control group of 11 volunteers.
Results: In unilateral semi-circular canal dehiscences, SVIN torsional and horizontal components observed on vertex location in 88% beat toward the lesion side in 95%, and can be obtained up to 800 Hz (around 500 Hz being optimal). SVIN slow-phase-velocity was significantly higher on vertex stimulation at 100 and 300 Hz ($P = 0.04$) than on mastoids. SVIN vertical component is more often upbeating than down-beating. A SVIN was significantly more often observed in unilateral than bilateral semi-circular-canal dehiscences ($P = 0.009$) and with a higher slow phase velocity ($P = 0.008$). In severe unilateral vestibular lesions the optimal frequency was 100 Hz and SVIN beat toward the intact side. The mastoid stimulation was significantly more efficient than vertex stimulation at 60 and 100 Hz ($P < 0.01$).
Conclusion: SVIN reveals instantaneously in unilateral semi-circular canal dehiscences a characteristic nystagmus beating, for the torsional and horizontal components, toward the lesion side and with a greater sensitivity toward high frequencies on vertex stimulation. SVIN three components analysis suggests a stimulation of both superior semi-circular canal and utricle. SVIN acts as a vestibular Weber test, assessing a vestibular asymmetrical function and is a useful indicator for unilateral semi-circular canal dehiscence.

1. Introduction

Bone conducted vibrations (BCV) applied to the cranium, a new modality of stimulation of vestibular receptors are now more clearly documented [1]. They can provoke otolith stimulations at frequencies around 200-500 Hz [ocular vestibular evoked myogenic potentials (oVEMP) or cervical vestibular evoked myogenic potentials (cVEMP)] [2] and induce at 100 Hz a Nystagmus named the Skull Vibration Induced Nystagmus (SVIN). This new global vestibular test meets the need to find its place (frequency range analysis) among other first line vestibular tests in the vestibulo-ocular reflex (VOR) exploration for guidance, orientation to further explorations or targeted imaging and to show its utility in the screening of common unilateral vestibular loss (UVL) or less common vestibular pathologies as a rapid not invasive test with an

Abbreviations: BCV, Bone Conducted Vibrations; SCC, semi-circular canal; SCD, Superior canal dehiscence; uSCD, Unilateral superior canal dehiscence; bSCD, Bilateral superior canal dehiscence; UVL, Unilateral vestibular loss; SUVL, Severe unilateral vestibular loss; SVIN, Skull Vibration Induced Nystagmus; SVINT, Skull Vibration Induced Nystagmus Test; SPV, Slow Phase Velocity of the Nystagmus; cVEMP, cervical Evoked Myogenic Potentials; CaT, Caloric Test; VHIT, VideoHead Impulse Test; HST, Head Shaking Test.

* Corresponding author at: Auditory Implants, Cochlear Implant Centre of the Alpes, Department of Oto-Rhino-Laryngology, Head and Neck Surgery, University Hospital, avenue Maquis du Grésivaudan, 38700 La Tronche, France.
E-mail addresses: gdumas@chu-grenoble.fr (G. Dumas), thycxf@126.com (H. Tan), dumaslauurent@gmail.com (L. Dumas), philippe.perrin@univ-lorraine.fr (P. Perrin), alexis.lion@crp-sante.lu (A. Lion), sschmerber@chu-grenoble.fr, schmerber.orl@free.fr (S. Schmerber).

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accurate cost-efficiency rate [3–9]. SVIN evaluates the upper part of vestibular frequency spectrum for the vestibule-ocular reflex (VOR) exploration (Caloric test occupies the lower part of VOR frequency spectrum at 0.003 Hz; otolith structures are explored at higher frequencies (200–700 Hz) with EMG analysis as described above). SVIN at 100 Hz acts as a vestibular Weber test and shows usually a nystagmus with a primarily horizontal component beating toward the intact side in different common peripheral UVL as an indicator of asymmetrical vestibular function [7,10]. SVIN may show various pattern of responses (horizontal, vertical, torsional components) at different locations and frequencies of stimulations following the pathologies and we hypothesize that SVIN addresses different structures, i.e. semicircular canals or otolith end organs involved in these pathologies. To our knowledge, this hypothesis has not been investigated in clinic for very high frequencies. In animals (with intact labyrinth), neural evidence shows that the bone conducted vibration stimulates both canal and otolith afferents at 100 Hz but only otoliths at 500 Hz [1,10–14]. In common vestibular explorations a nystagmus is classically observed when the VOR is stimulated by canal tests (calorics or rotatory test), but a nystagmus may also be observed when stimulating otoliths during the Off-Vertical Axis Rotation (OVAR) test. In clinical practice the stimulus frequency for Skull Vibration Induced Nystagmus test (SVINT) is commonly between 60 to 120 Hz [4]. One hundred Hz is the most commonly used frequency [3,6,15] and is accepted as the most efficient stimulation. A previous study of frequency tuning in UVL patients [4] used frequencies ranging from 20 to 150 Hz at constant amplitude of 0.2 mm vibration and optimal frequencies were observed between 60 to 120 Hz.

SVIN Test (SVINT) shows usually at 100 Hz in severe unilateral vestibular lesions (SUVL) with labyrinths normally encased in bone, a SVIN beating toward the intact side [10]. In superior canal dehiscence (SCD), the nystagmus induced by pressure or sound (Hennebert sign, Tullio phenomenon) is ipsilateral torsional and down beating corresponding to the stimulation of the dehiscent superior semi-circular canal (SCC) [16,17]. Similar observation with cranial vibrations have been reported describing a nystagmus with vertical and torsional components [18–20]. However, in SCD a horizontal significant component can be observed [21,22] and suggests a concomitant stimulation of other vestibular structures than the sole Superior SCC following Flourens and Ewald laws and according to canal nerves [23] or utricle nerve stimulations [24]. It has been hypothesized a possible contribution of the utricle in animals by Tullio [25], and in humans [26]. Vestibule bony hyper sensitivity to high frequencies has been described in animals with artificially created SCD [13] but to our knowledge no systematic clinical trials in SCD patients has so far been reported with SVIN.

The first aim of this study was to design a practical midline stimulation reference (front, vertex, occipital) in order to standardize the results. The second objective was to analyze SVIN responses (Slow phase velocity of the horizontal, vertical and torsional components of the nystagmus) at higher frequencies (up to 800 Hz) to establish the optimal frequency stimulation in SCD patients. The last aim was to establish SVIN sensitivity when using other vestibular tests such as cVEMP, caloric test (CaT), Head Shaking Test (HST) and video head impulse test (VHIT).

2. Participants and methods

2.1. Participants

Participants had no history of psychiatric or neurological disorders and neither patients nor control subjects had taken medications during at least one month before recordings. All the participants gave written informed consent before data collection, in accordance with the current ethical laws in France and the declaration of Helsinki. The different procedures stimulations were performed with our CE-marked Skull vibration material, which is approved by our local ethical committee.

Fourty patients with a SCD without previous surgery were enrolled between 2001 and 2016 (mean age 54 ± 16 years min-max: 32–82; 26 women and 14 men). Among them twenty seven had a unilateral SCD (uSCD) (the mean age was 59 ± 17 years, min-max: 39–82, with 16 women and 11 men) (they were used in experiment 3–4) A Sub class of 14 patients (mean age 53 ± 16 years, min-max: 32–82; 8 women, 6 men) with uSCD were studied in 3D and was used in exp 2.

Thirteen had a bilateral SCD (bSCD) (mean age was 54 ± 16 years, min-max: 32–79, with 10 women and 3 men; experiment 3 & 4).

Eighteen SUVL patients and 11 subjects for the control group were enrolled and studied in 3D.

SCD patients were defined by conductive hearing loss on low frequencies (under 1000 Hz) with a preserved stapedial reflex and the audiological Weber lateralized toward the lesion. All patients had a temporal bone CT scan reformatted in the Pöschl plane of the superior SCC confirming a dehiscence wider than 3 mm (19 had a Cone Beam and 21 a HRCT). The symptoms included hyperacusis, autophony, pulsatile tinnitus and sound or pressure-induced vertigo at different degrees or positional vertigo. The presence of increased amplitude and lower threshold for cVEMP was a favourable argument but not mandatory. Similarly, the observation of a Hennebert sign or a Tullio phenomenon (observed in 3 and 5 of our patients respectively) was characteristic but seldom observed and not mandatory. These inclusion criteria are in accordance with those enacted by Ward et al. [27].

SUVL were defined with a severe unilateral vestibular loss (UVL): caloric hypofunction higher than 80% (Jongkees formula) and a vestibular lesion dating more than 6 months. (mean age 60 ± 11 years, min-max: 42–80, 10 women and 8 men). Eleven were treated by intratympanic gentamicin, 3 had a vestibular neuritis and 4 a total UVL (trans labyrinthine surgery or vestibular neurectomy). Eleven among them were used in experiment No. 1, and the totality of them in experiment 2.

The subjects (control population) were selected among normal volunteers (mean age 55 ± 15 years, min-max: 24–70, 7 women and 4 men). The volunteers were collaborators working at the hospital without any medical history of vertigo, with normal hearing, normal caloric test and normal VHIT.

2.2. Stimulation and vestibular explorations: vestibular evaluation

The characteristics of SVIN stimulation were similar in the different experiments exepted for the topography (exp. 1) and frequency (exp. 2) and yielded differences adapted to the procedure of each experiment.

The other vestibular tests were used for the systematic inclusion of patients.

2.2.1. SVIN

The vibratory stimulation was applied in experiment No. 1 in different locations on the midline (front, bregma, vertex, occipital, sub-occipital) to determine optimal midline location for the following experiments. In experiments 2–4 all participants were stimulated to the vertex and successively on the left and right mastoid during 10 s for each location (each stimulation being separated by 3 seconds) using the handheld vibrator Minishaker 4810 (Bruel and Kjaer, Naerum Denmark) with the dominant hand. Participants were in an upright sitting position. On vertex and bregma, the force is given by the weight of the vibrator (1 kg) with no additional pressure; the vibrator being simply maintained in place (Force is

1 kg × 9.81 = 9.81 N). For the mastoid location the static force of application evaluated with force sensors in a previous work was approximately 10 N when applied laterally on a manikin simulating mastoid location [28]; on the patients mastoids the stimulation was retro-auricular level to the external auditory meatus on the left and then right side; the recordings were performed under 2D and/or 3D VNG, [i.e. eyes open (direct medial gaze) behind a videoscopic helmet, to identify the possible SVIN occurrence and its slow-phase eye velocity (SPV, in°/s)].

The nystagmus axis can be horizontal, vertical or torsional and its direction can be right or left, upward or downward, rotatory clockwise or counter-clockwise. This procedure has already been described in detail previously [5,10].

In experiments 1, 3 and 4 frequency was 100 Hz and for experiment 2 frequency varied from 10 to 800 Hz.

2.2.2. Caloric test (CaT)

This test was performed using VNG according to the bithermal (30° and 44 °C) caloric test protocol, with water infusion of the ear canal for 30 s. A hypofunction > 20% was considered significant.

2.2.3. Head-shaking test (HST)

The examiner shook the patient's head in the horizontal plane for 20 s at a frequency of 2 Hz, and rotation amplitude of 45 on both sides and VNG measured the response. The test was positive when at least one eye flick or nystagmus was observed.

2.2.4. Video Head impulse test (VHIT)

This test was performed on a sitting upright subject with the VHIT Ulmer device [Synapsys Inc., France] at 100 Hz image sampling. This device records eye position and shows possible covert saccades. It also analyses the vestibulo-ocular reflex (VOR) gain. The examiner exerted head impulses (10–20 of amplitude) at velocities higher than 200°/s in the horizontal, anterior, and posterior SCC planes. The results were considered abnormal for the horizontal canal when the ocular VOR response gain was < 80% and for the vertical canals when the gain was < 70% [29].

2.2.5. Cervical Vestibular Evoked Myogenic Potentials (cVEMP)

Patients were positioned supine on a stretcher and were asked to turn their head contralaterally to the stimulated ear. The protocol has previously been described in detail in a previous work [21]. The stimuli delivered with a headphone (TDH 39) were short-tone bursts at a frequency of 500 Hz (from 105 dB to 70 dB nHL, rise/fall time 1 ms, plateau time 2 ms). cVEMPs were recorded with a Racia Centor (Racia Biomedical Inc., France). One hundred to 200 stimulations were averaged on each side. The percentage of cVEMP asymmetry in patients with unilateral lesions was measured by calculating the evoked potential ratio (EPR) as follows EPR = AL-AS/AL + AS × 100, where AS is the smaller P13-N23 peak-to-peak amplitude and AL is the larger P13-N23 peak-to-peak amplitude. In unilateral cases, the test was considered positive in our clinic when the difference in amplitude between both sides was greater than 50%. In bilateral cases, the test was considered positive when the cVEMP thresholds were lower than 80 dB nHL.

The four experiments were performed by the same examiner (GD) and were conducted in the following order:

2.3. Experiment 1: midline location stimulus optimization at 100 Hz

2.3.1. Participants

Eleven severe unilateral vestibular lesion (SUVL) out of 18 patients were included: 7 had been treated by intratympanic gentamicin, 3 had a vestibular neuritis, one underwent vestibular neurectomy. The mean age was 62 ± 16 years, min-max: 43-80, with 6 women and 5 men.

2.3.2. Procedure

To identify the optimum site for midline stimulation, experiment No. 1 was conducted at 100 Hz in SUVL: stimulation during 10 seconds was applied without additional pressure else that the vibrator own weight on the cranium midline at frontal (Fz), Bregma (Br), vertex (Vx), and with the usual hand held pressure for occipital (Occ) and sub occipital (SOcc) regions. SVIN SPV was recorded on a 3D video nystagmograph (VNG) (Synapsys, Marseille, Inc, France).

This experiment was performed before the following experiments to give optimal midline location stimulation.

2.4. Experiment 2: comparison between uSCD, SUVL patients and normal subjects stimulated at different frequencies and locations.

In this experiment a 3D analysis was used to verify the influence of stimulation frequency and location on horizontal torsional or vertical components.

2.4.1. Participants

Twenty seven had a unilateral SCD (uSCD): 14 cases (mean age 53 ± 1.6 years, min-max: 32–82; 8 women, 6 men) were studied in 3D recording (Table 1) at different frequencies (10–800 Hz).

They were compared to eighteen SUVL patients and to eleven normal volunteers analyzed in 3D recording.

2.4.2. Procedure

The 18 SUVL patients, 14 uSCD patients and 11 controls (volunteers) were stimulated with 10–800 Hz vibration (Minishaker 4810, Bruel & Kjaer; Naerum; Denmark) on each mastoid process successively and on the cranium midline vertex. SVIN Horizontal, Vertical and Torsional components were recorded with a 3D VNG (Synapsys Inc., France) and SVIN-SPV was analyzed.

2.5. Experiment 3: SVIN Sensitivity in unilateral SCD (uSCD) vs. bilateral SCD (bSCD)

2.5.1. Participants

Twenty seven uSCD and 13 bSCD patients were included.

2.5.2. Procedure

The stimulation was performed at 100 Hz on the vertex and each mastoid. SVIN was recorded on 2D VNG and the SVIN SPV was analysed (left and right side averaged value). The SVIN horizontal response for both populations was compared. Among the 27 uSCD, 14 were studied in experiment No. 2. Results were expressed in mean ± SD.

2.6. Experiment 4: SVIN sensitivity in uSCD and bSCD vs other vestibular tests

SVINT, CaT, HST, VHIT and cVEMP were performed in uSCD ($n = 27$) and bSCD ($n = 13$). The percentages of positive results (abnormal results) for those vestibular tests were compared in both groups of patients.

Statistical analysis

Statistical analysis was performed using a ANOVA (Experiment 1–3) and Cochrane Q test (Experiment 4) with GraphPad Prism software (GraphPad, Inc., La Jolla, CA, USA). In experiment 2, Anova test followed by running post-hoc Tukey's test was performed to assess optimal frequency and location in the 3 populations. The results are presented as the mean ± s.e.m.

Table 1
SVIN in SUVL and uSCD at 100 Hz (Experiment 2).

SUVL	Population				Vestibular Tests			SVIN									
n = 18	Age	Gender	Pathology	Side	CaTHypo %	HST	VHIT	Mastoid				Vertex				Vertical	
								Ipsilat		Contralat		Ipsilat		Contralat			
								H	T	H	T	H	T	H	T	Mastoid	Vertex
1	58	F	ItG	R	100	L	. . ..	-	-	+	+	-	-	-	-	Dw	0
2	64	F	VN	L	80	-	. . ..	-	-	+	+	-	-	+	+	Up	Up
3	43	M	ItG	L	82	R	LH	-	-	+	+	-	-	+	+	Up	Up
4	63	M	ItG	L	81	-	LH,LP	-	-	+	-	-	-	+	-	0	0
5	62	M	VN	R	81	-	. . ..	-	-	+	-	-	-	+	-	0	0
6	74	F	ItG	R	100	L	. . ..	-	-	+	+	-	-	+	-	Up	Up
7	54	F	ItG	L	82	R	LH,LP	-	-	+	+	-	-	-	-	Up	Dw
8	60	F	ItG	R	99	L	. . ..	-	-	+	+	-	-	+	+	0	0
9	42	M	ItG	L	80	R	. . ..	-	-	+	+	-	-	+	-	Up	0
10	64	F	ItG	R	84	L	. . ..	-	-	+	+	-	-	+	-	0	0
11	55	M	VNect	R	98	L	RH,RA,RP	-	-	+	+	-	-	+	+	Up	Up
12	45	F	ItG	R	83	-	RH,RA	-	-	+	-	-	-	-	-	0	0
13	56	F	ItG	R	100	L	RH,RA,RP	-	-	+	+	-	-	+	-	Up	0
14	62	M	VN	R	80	-	. . ..	-	-	+	-	-	-	+	-	0	0
15	77	F	ItG	R	98	L	RH,RP	-	-	+	+	-	-	+	+	0	Dw
16	55	M	VNect	R	100	L	RH,RP	-	-	+	+	-	-	+	-	0	0
17	80	M	TLA	R	100	L	RH,RA,RP	-	-	+	+	-	-	+	+	Up	0
18	62	F	TLA	L	100	R	RH,RA,RP	-	-	+	+	-	-	+	+	0	0

uSCD	Population				Vestibular Tests			SVIN									
n = 14	Age	Gender	Estimate size in mm	Side	CaTHypo %	HST	VHIT	Mastoid				Vertex				Vertical	
								Ipsilat		Contralat		Ipsilat		Contralat			
								H	T	H	T	H	T	H	T	Mastoid	Vertex
1	57	M	5	L	. . ..	-	N	+	+	-	-	++	++	-	-	Up	Up
2	39	F	3	L	67	R	LH	-	-	+	+	-	-	-	-	Dw	Dw
3	80	M	7	L	. . ..	. . ..	. . ..	-	-	-	-	+	+	-	-	0	Up
4	82	F	4	R	N	-	RA	+	-	-	-	++	+	-	-	Up	Dw
5	66	M	3	L	. . ..	-	N	-	-	-	-	+	+	-	-	Up	Up
6	46	M	5	L	N	-	N	+	+	-	-	++	++	-	-	Dw	Up
7	61	F	5	L	N	-	N	+	+	-	-	+	+	-	-	0	Up
8	43	F	8	L	N	L	N	+	+	-	-	+	++	-	-	0	0
9	52	F	4	L	80	-	N	+	-	-	-	+	-	-	-	Up	Up
10	33	F	4	R	N	-	N	-	+	-	-	+	+	-	-	Dw	Dw
11	72	M	4	L	30	-	N	-	-	+	+	+	+	-	-	Dw	0
12	57	F	4	L	N	-	N	-	+	+	-	++	+	-	-	Up	Up
13	68	M	3	R	N	-	. . ..	-	-	+	-	++	++	-	-	Up	Up
14	40	F	6	R	N	-	N	-	-	-	-	+	-	-	-	Dw	Dw

Population and SVIN 3D results at 100 Hz stimulation for different SVIN components [H: horizontal component; V: vertical; T: torsional] and directions (Up: up beating; Dw: down beating). SUVL: severe unilateral vestibular lesion; uSCD: unilateral superior semicircular canal dehiscence. ItG: intratympanic gentamicin; VN: vestibular neuritis; VNrct: vestibular neurectomy. R: right side; L: left side; . . .: no data. CaT: Caloric Test; HST: Head Shaking Test (L: HSN beating toward the left; R: HSN toward the right); VHIT: Video Head Impulse Test (RA: Right anterior canal dysfunction; RH: right Horizontal canal dysfunction; RP = right posterior canal dysfunction; LA, LH, LP: same signification for the left side). The estimated dimension of the dehiscence is given in mm. SVIN [horizontal and torsional components] always beats toward the intact side in SUVL and most often toward the lesion side in uSCD. An up beating VIN is commonly observed in uSCD. The SVIN results on mastoid and vertex marked+ or ++ correspond to a semi-quantitative evaluation of the nystagmus intensity.

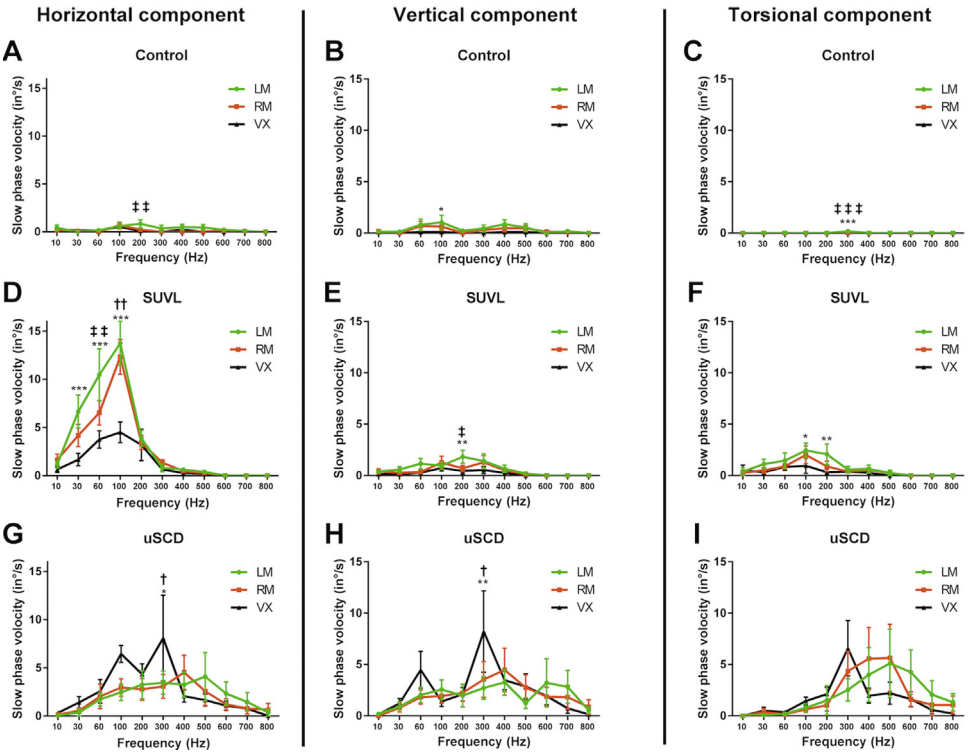

Fig. 1. SVIN horizontal, vertical and torsional components Slow phase velocity (SPV) at different frequencies and locations (Exp. 2). The stimulator delivered frequencies from 10 to 800 Hz with the Mini Shaker 4810 [Bruel &Kjaer; Naerum; Denmark]. SUVL: Severe Unilateral Vestibular Lesions ($n = 18$); uSCD: unilateral superior canal dehiscence ($n = 14$); Controls ($n = 11$). A, D, G. Horizontal component for the left mastoid [LM], right mastoid [RM] and vertex [VX] in the 3 populations: Controls (A); SUVL (D); uSCD (G). In SUVL (patients with lesion associated with an encased labyrinth), the optimal frequency was 100 Hz [$P < 0.001$]. No responses were observed beyond 300 Hz. Mean values were: LM [$13.75 \pm 2.28°$/s] and RM [$12,33 \pm 1.81°$/s]. Significantly lower responses are observed on vertex [$4.5 \pm 1.06°$/s] ($P < 0.01$). No responses were observed at 10 Hz and 500 Hz. In uSCD higher responses were observed at 500 Hz without significance (tendency) and there was a much wider range of sensitivity spread from 60 Hz up to 800 Hz. The average mean value at 500 Hz for the LM was $4.10 \pm 2.51°$/s. Significantly higher responses are obtained on vertex than on mastoids at 300 Hz ($P < 0.05$). In controls, no or few significant responses [VIN inconsistent direction] with a mean value lower than 1 or 2°/s at 100 or 200 Hz were observed. B, E, H. Vertical component observed on LM, RM and VX in the 3 populations. In SUVL, the SVIN-SPV at 200 Hz was small on LM [$1.81 \pm 0.65°$/s]. In uSCD higher values were observed at 400 Hz on RM [$4.46 \pm 2.12°$/s]. C, F, I. Torsional component on LM, RM, and Vx in the 3 populations. In SUVL SVIN-SPV at100 Hz on LM was very low [$2.44 \pm 0.72°$/s]. In uSCD, the SVIN-SPV on RM at 500 Hz was $6.64 \pm 3.27°$/s. *; **; *** indicates significance at $P < 0.05$, < 0.01, and < 0.001 respectively, when comparing LM/Vx. †; ††; ††† indicates significance at $P < 0.05$, < 0.01, and < 0.001 respectively, when comparing RM/Vx. ‡; ‡‡; ‡‡‡ indicates significance at $P < 0.05$, < 0.01, and < 0.001 respectively, when comparing LM/RM.

Statistically significant differences were accepted for a probability level of $P < 0.05$.

3. Results

3.1. Experiment 1: Optimal location on the midline (SUVL, n = 11)

Horizontal slow phase velocities of the SVIN were normally distributed. No significant difference was observed whatever the midline locations at 100 Hz (Repeated measures ANOVA: F = 0.52, $P = 0.87$). Vertical slow phase velocities of the SVIN were abnormally distributed and log-transformation of the data was not possible (several 0 values). No significant difference was observed whatever the midline locations at 100 Hz (Friedman test: $\chi^2 = 3.54$, $P = 0.47$). Similar results were observed for the torsional component, which showed low SPV responses.

Therefore, for midline stimulation, the vertex location, which is in clinical practice the most often used midline location, was arbitrary chosen as the midline stimulation reference for experiments No. 2–4.

3.2. Experiment 2

3.2.1. Optimal frequency SUVL/uSCD/Controls

*3.2.1.1. In SUVL (*n = *18).* The quick phases of SVIN always beat away from the affected ear (Fig. 1, Table 1).

For the "horizontal component", different frequencies caused different SPV values ($P < 0.0001$), $n = 18$ (Anova). Comparisons showed that 100 Hz was the optimal frequency to reveal the greatest slow-phase velocity of the horizontal SVIN component: for the right mastoid location, the SVIN SPV was higher at 100 Hz ($12.33 \pm 1.81°$/s) than all the other frequencies ($P < 0.001$). SVIN-SPV 60Hz = $6.52 \pm 1.05°$/s (60 Hz vs 100 Hz $P < 0.01$); 200 Hz SVIN-SPV = $3.43 \pm 0.83°$/s (200 Hz vs 100 Hz $P < 0.0001$); 500 Hz SVIN SPV = $0.28 \pm 0.05°$/s (500 Hz vs 100 Hz $P < 0.0001$). Similar results were obtained for the left mastoid at 100 Hz ($13.76 \pm 2.28°$/s), 30 Hz ($6.65 \pm 1.84°$/s), 200 Hz ($3.87 \pm 0.86°$/s), 500 Hz ($0.39 \pm 0.06°$/s) and with less velocity for the vertex location ($4.5 \pm 1.06°$/s) at 100 Hz (Fig. 1).

For the "vertical component", the different frequencies caused differences in vertical SVIN SPV ($P = 0.0001$). There was a tendency to show higher responses (as for the horizontal component) at 100 Hz than at lower (60 Hz) or higher frequencies (200–400 Hz) (Fig. 1), however, the comparisons (ANOVA test) did not show significant differences (SVIN SPV values in this condition were small and the population was small). At 100 Hz for the right mastoid SVIN–SPV was $1.25 \pm 0.63°$/s. Similar values were observed for LM ($0.94 \pm 0.37°$/s) and smaller for VX ($0.72 \pm 0.27°$/s).

At 60 Hz,for the right mastoid, SVIN-SPV values were $0.34 \pm 0.06°$/s, at 200 Hz $0.69 \pm 0.23°$/s, at 500 Hz $0.12 \pm 0.01°$/s.

For the "torsional component", at 100 Hz on the left mastoid, SVIN-SPV was $2.44 \pm 0.72°$/s. It was $1.44 \pm 0.24°$/s at 60 Hz; $2.06 \pm 0.42°$/s at 200 Hz and $0.25 \pm 0.07°$/s at 500 Hz.

The SVIN-SPV at 100HZ was significantly greater for the horizontal component than for the vertical and torsional components, which are small (13.76 ± 2.28°/s; 0.94 ± 0.37°/s; 2.44 ± 0.72°/s respectively, $P < 0.01$).

SUVL patients signal usually, when stimulated, a sensation of latero-pulsion toward the safe side (16 patients among 18).

3.2.1.2. In uSCD (n = 14). The SVIN direction beats toward the lesion side in 92% of cases (Table 1 and Fig. 1).

A wider and higher range of frequency responses up to 700 Hz or 800 Hz was observed without a clear cut significant optimal frequency for the horizontal and vertical components of SVIN SPV (Anova was not significant) (Fig. 1).

For the "horizontal component", a tendency to higher SVIN SPV was observed at 400 and 500 Hz but was not significantly different from responses to other frequencies. The average mean value at 500 Hz for the LM was 4.09 ± 2.51°/s. The average value for RM at 400 Hz was 4.52 ± 1.78°/s. The average mean value at 100 Hz for the LM was 2.46 ± 0.67°/s and for RM was 2.96 ± 0.87°/s. For vertex stimulations, greater responses were observed at 300 Hz [8.09 ± 4.46°/s].

For the "vertical component", the SVIN SPV on the RM at 400 Hz was 4.46 ± 2.12°/s and on the vertex at 300 Hz was 8.21 ± 3.97°/s. It was on the RM at 100 Hz 1.93 ± 0.21°/s and on the vertex at 100 Hz 1.40 ± 0.27°/s. In 4 patients, the down beating nystagmus observed under videoscopy was enhanced when the gaze was directed toward the plane of the dehiscent canal and when the record was performed on the eye ipsilateral to the lesion.

For the "torsional component", a tendency to higher SVIN SPV was observed at 400 and 500 Hz but was not significantly different from responses to other frequencies. The average mean value at 500 Hz for the LM was 5.64 ± 3.27°/s. The average value for RM at 400 Hz was 5.57 ± 3.03°/s. For vertex stimulations, the average mean value at 300 Hz was 6.60 ± 2.70°/s. There was no significant difference whether the stimulation or the record were performed ipsilaterally or contralaterally.

There was no significant difference between horizontal, vertical, and torsional components for SVIN-SPV. Fig. 2 shows a 3D recording (direct tract) in an uSCD patient. In uSCD a tendency of sensitivity extension toward higher frequencies than 100 Hz is observed in 65% of cases (9 patients among 14). Frequencies lower than 100 Hz show a tendency to lower SPV.

Patients with uSCD (16 patients out of 27) signal usually after repeated stimulations a sensation of dizziness with nausea and symptoms commonly attributed to otolith signs.

3.2.1.3. In control subjects (n = 11). No significant difference for SVIN SPV was observed when comparing results at different frequency stimulation (Fig. 1). No nystagmus was observed in 62% of cases. A low-intensity SVIN, seldom observed in 38% of cases, was not reproducible, changed direction following the side of stimulation (inconsistency) or was observed in only one location. Its SPV (only observed for the horizontal and vertical components) was < 2°/s in 30% and < 3°/s in the other 8% of normal subjects.

The criteria of a significant SVIN and a positive test are as follow:

- consistency: the SVIN must have the same direction in at least 2 on 3 cranial location (left mastoid, right mastoid, vertex) e.g. a SVIN beating rightward after right mastoid stimulation, and leftward after left mastoid stimulation is not valid: negative test;
- reproducibility: a SVIN direction changing (rightward and then leftward on the same location) after 2 trials or the SVIN disappearance at the second trial makes the test negative;
- an intensity or SVIN SPV < 2°/s makes the test result doubtful or negative.

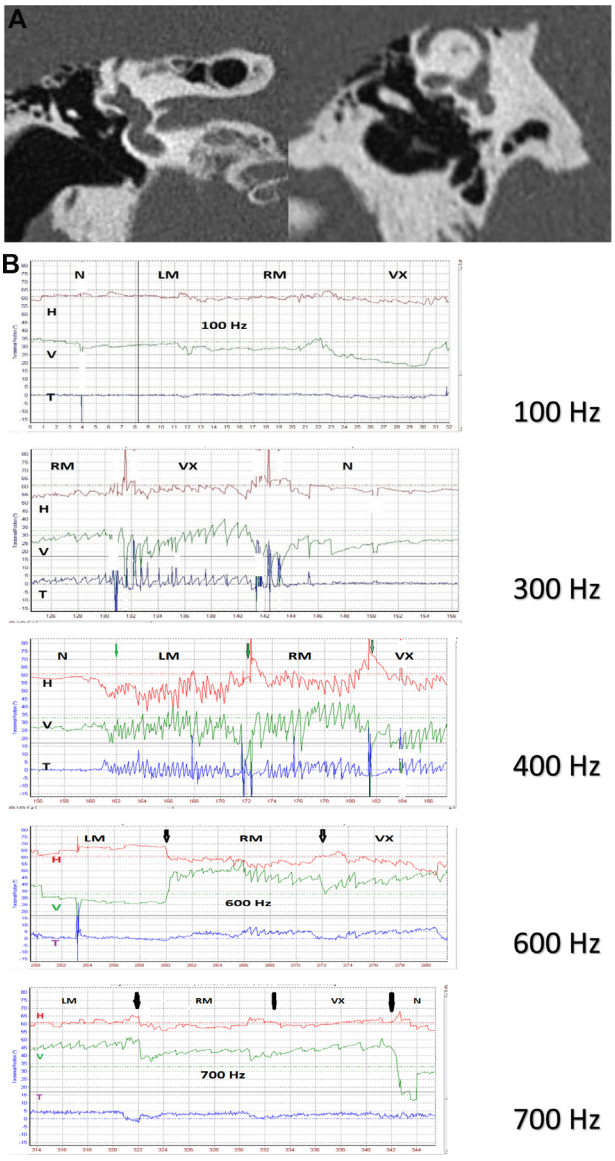

Fig. 2. SVIN in a right uSCD. A. CT scan reformatted in Pöschl plane. B. Stimulation ranging from 100 to 700 Hz. 3D recording; direct tract. Strong responses are observed at 300 and 400 Hz with an equivalent representation of horizontal, torsional and vertical components. The nystagmus axis can be horizontal, vertical or torsional and its direction can be right or left, upward or downward, rotatory clockwise or counter-clockwise. By definition, a torsional nystagmus is clockwise when it is beating toward the right of the patient [the upper part of the eye is beating toward the patient's right]. It is counter-clockwise when the upper part of the eye beats toward the patient's left. The device conventionally displays the clockwise fast phase of the eye movement as a down beating nystagmus. In this example SVIN is horizontal beating toward the right, torsional clockwise (beating toward the patient's right side) and vertical down beating.

3.2.1.4. Comparison of frequency sensitivity in uSCD and SUVL (Fig. 1). For the horizontal component, the SVIN SPV in uSCD was significantly higher than in SUVL at 400 Hz for RM stimulation ($P < 0.01$) and at 500 Hz for LM stimulations ($P < 0.05$). For vertex stimulations in uSCD, the SVIN horizontal SPV at 300 Hz was significantly higher than in SUVL ($P < 0.001$). Similar results showing a higher sensitivity to high frequencies in uSCD were observed for the torsional component for mastoids stimulations at 400 and 500 Hz ($P < 0.05$).

Similar results were observed for the vertical component and greater responses were obtained in uSCD than in SUVL at high frequencies between 400 to 700 Hz for mastoid and vertex stimulations ($P < 0.05$).

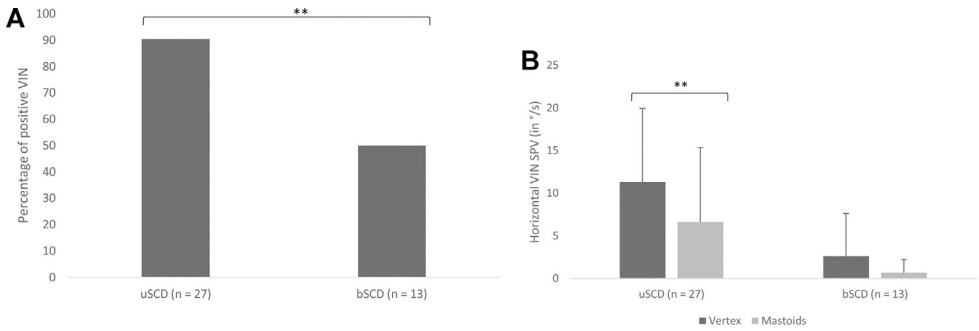

Fig. 3. Comparison of horizontal SVIN in uSCD and bSCD. A. Percentage of positive VIN in patients with uSCD and bSCD after vertex stimulation. B. Horizontal VIN SPV (in°/s) between uSCD and bSCD collected after vertex or mastoids stimulations. The value for mastoid stimulations is the mean value of right and left mastoid stimulation. uSCD, unilateral superior canal dehiscence ($n = 27$); bSCD, bilateral superior canal dehiscence ($n = 13$). ** $P < 0.01$. Results are expressed as mean value ± SD. A. Fisher test. B. Mann and Whitney test.

Conversely, the SVIN SPV at 30, 60 and 100 Hz was higher in SUVL than in uSCD for both mastoid stimulations for the horizontal component ($P < 0.001$).

3.2.2. Optimal location in uSCD vs. SUVL

In uSCD, vertex stimulation was significantly more efficient than mastoid stimulation for the horizontal, vertical and torsional components at 300 Hz ($P < 0.05$) but not for higher frequencies. In SUVL, mastoid stimulation at 30, 60 and 100 Hz was significantly more efficient for horizontal and torsional components than vertex stimulations ($P < 0.001$) (Fig. 1). The value of mastoids SVIN SPV were significantly higher in SUVL vs. uSCD at 30, 60 and 100 Hz ($P < 0.001$, Anova).

The values of vertex SVIN SPV were significantly higher in uSCD vs. SUVL for the horizontal component at 300 Hz ($P < 0.001$), for the vertical component at 60 Hz ($P < 0.01$), 300 Hz ($P < 0.001$) and 400 Hz ($P < 0.05$) and for the torsional component at 300 Hz ($P < 0.001$).

In controls, no optimal location could be identified since no clear acceptable responses could be identified. When a SVIN is identified the direction changes with the side of stimulation (inconsistency) and the Horizontal SVIN–SPV mean value is lower than 2°/s.

3.3. Experiment 3: Comparison of SVIN results in uSCD and bSCD

During vertex stimulation, SVIN was more commonly observed in uSCD (88%) than in bSCD (50%) ($P = 0.009$) (Fig. 3 & Table 2). In uSCD, SVIN beat mostly toward the lesion side (95%). The vertical component was mainly up beating (60%). During vertex and mastoid process stimulations in uSCD patients a coherent SVIN beating toward the same direction accordingly to previously enacted criteria (positive test) was observed in 70%.

The SVIN SPV was significantly higher in uSCD than in bSCD ($P = 0.0082$):

The vertex stimulation showed a main effect of the pathologies, with SPV being higher in uSCD [5.66 ± 8.40°/s] than bSCD [1.50 ± 3.95°/s] (F = 7.5; $P = 0.0082$, Anova).

The mastoid stimulation showed a main effect of the pathologies, with SPV being higher in uSCD [4.09 ± 6.93°/s] than bSCD [0.84 ± 2.03°/s] (F = 6.24; $P = 0.0147$, Anova).

3.4. Experiment 4: Comparison of SVIN results in uSCD with other vestibular tests (Table 2)

In uSCD ($n = 27$) the SVIN sensitivity was higher than the Caloric, head shaking test, and VHIT sensitivity ($P < 0.001$, Cochrane test), SVIN was positive in 24/27 cases, CaT in 3/23, HST in 5/25 and VHIT in 6/21 but was not different from cVEMP (19/26) ($P > 0.5$) (Table 2). In bSCD, no difference of sensitivity between the tests SVIN (7 positive cases among 13), CaT (3/12), HST(5/11), VHIT (3/11, cVEMP (9/12)) was observed ($P = 0.88$).

4. Discussion

The skull vibration induced nystagmus test can predict the vestibular asymmetry in unilaterally vestibular lesioned patients [5,9,30] and is used as a vestibular Weber Test [10,21]. Neural evidence shows that the bone conducted vibration stimulates both canal and otolith afferents [13,14]. This test has a potential utility to orientate further examination, vestibular exploration or targeted imagery in order to enhance diagnosis accuracy and surgical decision.

In conductive hearing loss with CT-verified SCD, we previously reported that SVIN horizontal and torsional components beat toward the lesion side (in relation to the bone conducted facilitation of the inner ear favored by the existence of a third window) [21] and is associated to a “bony hyperacousis” [16,17]. Unlike severe unilateral vestibular lesions where the SVIN is of the lesional type beating toward the intact side [5,10], a SVIN beating toward the lesion side is strongly suggestive of SCD [21]. However, it can also be observed in other labyrinthine malformations associated to a third window (e.g. enlarged vestibular aqueduct) [31]. In SUVL (normally encased labyrinth), greater responses are observed at 100 Hz for the horizontal component (there were poor responses for the vertical and torsional component) and no responses at 500 Hz. In contrast, uSCD patients show greater sensitivity to high frequencies; higher responses were observed at approximately 500 Hz without clear-cut specific frequency and the SPV values of the vertical and torsional components were higher than in SUVL. The utility of very high frequency stimulations in SCD has already been signaled for oVEMP [32]. Our results suggest that different anatomical structures may be stimulated by SVIN in these 2 populations, mainly horizontal canal and at a less degree utricle at 100 Hz in SUVL, while both canal (mainly superior canal) and utricular neurons are activated in uSCD at 500 Hz. In uSCD, the most often observed component is torsional and horizontal and the vertical component is most often up beating suggesting that the superior SCC is probably not the only stimulated structure. However, we have observed in 23% of our patients a down beating SVIN when the gaze was directed toward the lesion side at vertex stimulation and the eye axis placed in the same plane of the dehiscent canal; this sign was more often observed in our series than the Hennebert sign which we seldom noted. The usual torsional ipsilateral and down beating nystagmus induced by sound (Tullio phenomenon) or pressure (Hennebert sign) attributed to the dehiscent superior canal activation [16,17] does not explain the totality of the SVIN components here observed. The horizontal component may be explained by the concomitant utricle stimulation as suggested by Halmagyi [26] and

Table 2
SVIN in uSCD and bSCD at 100 Hz (Experiment 3 & 4).

No.	Side	Size (mm) L	Size (mm) R	CaT (Hypofunction %)	HST	VHIT	cVEMP	SVIN									
								Mastoid				Vertex				Vertical	
								Ipsilateral to the lesion		Contralateral to the lesion		Ipsilateral to the lesion		Contralateral to the lesion			
								H	T	H	T	H	T	H	T	Mastoid	Vertex
1	L	5		. . ..	0	N	-	+	+	-	-	++	++	-	-	Up	Up
2	L	3		67 %	R	LH	+	-	-	+	+	-	-	+	-	Dw	Dw
3	L	7		. . ..	....	....	+	-	-	-	-	+	+	-	-	0	Up
4	R		4	N	0	RA	—	+	-	-	-	++	+	-	-	Up	Dw
5	L	3		. . ..	0	N	+	-	-	-	-	+	+	-	-	Up	Up
6	L	5		N	0	N	-	+	+	-	-	++	++	-	-	Dw	Up
7	L	5		N	0	N	+	+	+	-	-	+	+	-	-	0	Up
8	L	8		N	L	N	++	+	+	-	-	+	++	-	-	0	0
9	L	4		81 %	0	N	-	+	-	-	-	+	-	-	-	Up	Up
10	R		4	N	0	N	+	-	+	-	-	+	+	-	-	Dw	Dw
11	L	4		30 %	0	N	....	-	-	+	+	+	+	-	-	Dw	0
12	L	4		N	0	N	+	-	+	+	-	++	+			Up	Up
13	R		3	N	0	....	++	-	-	+	-	++	++	-	-	Up	Up
14	R		6	N	0	N	+	-	-	-	-	+	-	-	-	Dw	Dw
15	L	3		N	0	....	-	+	-	-	-	+	+	-	-	Up	0
16	R		4	N	0	N	+	-	-	+	-	+	+	-	-	Up	Up
17	R		8	N	0	RA	+	+	+	-	-	+	-	-	-	Up	Up
18	L	4		N	0	LA LP	-	-	....	-	-	-	—	-	-	0	0
19	L	5		N	0	N	+	-	....	+	....	+	—	-	—	Up	Up
20	R		4	N	0	....	-	+	-	-	-	+	+	-	-	Up	Dw
21	L	4		N	L	N	+	-	+	-	-	+	+	-	-	Up	Up
22	L	3		N	0	....	+	-	....	-	-	-	—	-	-	0	0
23	R		4	N	0	RA	-	-	....	-	-	+	—	-	-	Up	0
24	L	7		N	0	RA	+	+	....	-	-	+	—	-	-	Up	Up
25	L	5		....	....	N	-	-	+	-	-	+	+	-	-	Dw	0
26	L	5		N	L	N	-	-	+	-	-	+	+	-	-	Dw	Dw
27	L	3		N	R	....	+	-	....	-	....	....	—	----	----	0	—

No.		L	R	CaT (Hypo %)	HST	VHIT	cVEMP	H Left T		H Right T		H Left T		H Right T		Vertical Mastoid	Vertex
28	Bilat	7	3	N	. . ..	LA	L+	-	-	-	-	-	-	-	-	0	0
29	Bilat	2	3	N	0	N	R+	-	-	-	-	-	-	-	-	0	0
30	Bilat	3	5	R 42 %	R	N	Large bil	-	....	+	....	-	....	++	....	Up	Up
31	Bilat	5	4	N	0	N	-	+	....	-	-	....	....	-	-	Up	....
32	Bilat	6	6	N	0	N	Large bil	-	....	-	....	-	....	-	....	0	0
33	Bilat	5	5	N	0	N	-	-	....	-	....	-	....	-	....	0	0
34	Bilat	5	4	L 79 %	R	....	-	-	....	+	....	....	....	....	....	Up	....
35	Bilat	8	8	N	....	RL	Large bil	-	....	-	....	-	....	+		-	Up
36	Bilat	7	5	....	L	....	....	-	-	-	-	+	+	-	-	Up	Up
37	Bilat	4	4	N	0	N	R+	-	-	-	-	-	-	-	-	0	0
38	Bilat	4	6	N	R	N	Large bil	-	....	+	....	-	....	++	....	Up	0
39	Bilat	6	5	R 39 %	0	LA	L	-	-	-	-	+	+	-	-	0	Dw
40	Bilat	5	6	N	L	N	Large bil	-	-	-	-	-	....	-	....	0	0

SVIN results at 100 Hz stimulation for different SVIN components recorderd either in 2D or 3D [H: horizontal component; V: vertical; T: torsional] and directions (Up: up beating; Dw: down beating). uSCD: Unilateral Superior canal dehiscences; bSCD: bilateral canal dehiscences. The estimate width of dehiscence is given in mm; Side of lesion (R: right; L: left). Results of different vestibular tests compared to SVIN are given as follows: CaT caloric test: N: no hypofunction; RH: right hypofunction; LH: left hypofunction. HST: 0: no HSN; L: left HSN; R: Right HSN. VHIT: N: normal; LA: hypofunction on the left anterior canal; LP: hypofunction on the left posterior canal; LLat: hypofunction on the left lateral canal; RL Lat: Dysfunction of both lateral canals on the right and left side. cVEMP:+ result positive ipsilaterally to the lesion; -: normal result (no asymmetry); . . ..: no data. SVIN H horizontal component: R beating toward the right; L beating toward the left. V vertical component: up: up beating; dw: down beating. T torsional component: R: the upper part of the eye is beating toward the patient's right; L: the upper part of the eye is beating toward the patient's left.

by physiology [24]. In addition, Tullio in alert animal's studies with artificially created SCD described head movements or muscles contractions. In pigeons ear he observed the membranous wall of the utricle oscillate and in a few animals (rabbits) eye modifications equivalent to an ocular tilt reaction response.

These results are consistent with the response of primary semicircular canal afferent neurons in guinea pigs in response to BCV stimulation: in animals with normally encased bony labyrinths, canal neurons can be activated by BCV only at low frequencies (100–200 Hz) but not at frequencies of 500 Hz [13]. This low frequency tuning of canal neurons is consistent with the low frequency tuning of SVIN in SUVL patients found here. In SUVL patients, the direction of the quick phases of nystagmus is away from the affected side since only neurons on the remaining healthy side are activated. After an uSCD, canal neurons can be activated by BCV at low frequencies but now also up to high frequencies, even above 745 Hz [1,13]. This finding is consistent with the upward frequency shift of SVIN reported here in uSCD patients. In uSCD patients, the direction of the nystagmus is "toward" the affected side since canal neurons on the affected side will be activated, resulting in nystagmus with slow phases away from the affected side. In this way, we conclude that SVIN acts as a vestibular Weber test [10,21].

The observation that SVIN sensitivity is lower in bSCD than in uSCD may be related to the fact that SVIN acts as an indicator of vestibular asymmetry (vestibular Weber test). In case of partial bilateral symmetrical or slightly asymmetrical lesion, the test remains negative or shows a very low SVIN SPV. Therefore, in bSCD with both sides affected, the asymmetry may be small and so cVEMP seem to be more relevant since it analyzes separately each side.

In clinical practice SVIN, a first-line examination test for VOR exploration, may help the clinician to discriminate and make a decision in ambiguous cases (true dehiscence or thin bone over the SCC) with disabling vertigo. Radiological results have shown possible discrepancies in SCD between CT scanner results and real anatomically verified lesions [27,33,34], as well as similar symptoms in near dehiscent superior canals [27]. It has been suggested that cVEMP is the best clinical indicator in SCD [35]. Here we show that SVIN in uSCD yields responses as efficient as cVEMP, but more efficient than other common vestibular tests. Govender et al., 2016 [35] showed that cVEMP were modified significantly in only 82% of SCD. Zuniga et al. [36] showed in 29 surgically verified SCD that cVEMP threshold had a sensitivity of 80% (specificity of 100%) and the study of oVEMP amplitude showed a sensitivity of 90%. In our study, we observed a SVIN sensitivity of 88% (vertex stimulation) and specificity of 62% (total absence of nystagmus).

5. Conclusion

In patient with unilateral conductive hearing loss and normal ear drum, a Skull Vibration Induced Nystagmus observed preferentially on Vertex location and with a torsional and horizontal component beating toward the lesion side at 100 Hz and higher frequencies up to 700 Hz is strongly suggestive of a Superior canal dehiscence. Skull Vibration Induced Nystagmus reveals instantaneously a characteristic nystagmus and acts as a vestibular Weber test. It is more sensitive to reveal unilateral than bilateral superior canal dehiscence. A primarily down beating nystagmus is preferentially observed in patients when the gaze is directed toward the plane of the superior dehiscent canal. We propose that Skull Vibration Induced Nystagmus test should be considered as a useful indicator for unilateral superior canal dehiscence diagnosis in clinical practice.

Disclosure of interest

The authors declare that they have no competing interest.

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