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Clinical interest of postural and vestibulo-ocular reflex changes induced by cervical muscles and skull vibration in compensated unilateral vestibular lesion patients

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Abstract. Skull vibration induces nystagmus in unilateral vestibular lesion (UVL) patients. Vibration of skull, posterior cervical muscles or inferior limb muscles alters posture in recent UVL patients. This study aimed to investigate the postural effect of vibration in chronic compensated UVL patients.

Vibration was applied successively to vertex, each mastoid, each side of posterior cervical muscles and of triceps surae in 12 UVL patients and 9 healthy subjects. Eye movements were recorded with videonystagmography. Postural control was evaluated in eyes open (EO) and eyes closed (EC) conditions. Sway area, sway path, anteroposterior and medio-lateral sways were recorded. A vibration induced nystagmus (VIN) beating toward the healthy side was obtained for each UVL patient during mastoid vibration. In EO, only sway path was higher in UVL group during vibration of mastoids and posterior cervical muscles.

The EO postural impairments of UVL patients could be related to the eye movements or VIN, leading to visual perturbations, or to a proprioceptive error signal, providing an erroneous representation of head position. The vibration-induced sway was too small to be clinically useful. Vestibulo-ocular reflex observed with videonystagmography during mastoid vibration seems more relevant to reveal chronic UVL than vestibulo-spinal reflex observed with posturography.

Keywords: Inner ear, compensation, nystagmus, posturography, videonystagmography

1. Introduction

Immediately after sudden bilateral or unilateral loss of vestibular function, patients are ataxic with severe postural instability, which eases over weeks and

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months through the process of vestibular compensation [14]. Vestibular compensation occurs in patients with vestibular neuritis, vestibular schwannoma or surgery in Menière's disease [5,7,17,22]. For example, in patients with vestibular schwannoma, vestibular compensation accompanies the gradual vestibular dysfunction caused by the tumor growth [7]. The surgical removal of the tumor, which corresponds to an acute unilateral vestibular deafferentation, leads to a decompensation of this previously compensated situation, and mainly explains the severe vertigo and balance impairments reported by patients after surgery. Therefore, balance is impaired after surgical removal and recovers progressively after surgery [19,22,29]. Thus, compensation plays a major role in reorganizing and normalizing the vestibulo-ocular reflex (VOR) and the vestibulospinal reflex (VSR) in patients with unilateral vestibular lesion (UVL) [20].

Vestibular lesions are often so effectively compensated that their diagnosis and the determination of the compensation level may be difficult to realize. Norré et al. [20] reported that the compensatory and adaptive processes for the VOR and the VSR, evaluated respectively by pendular rotary vestibular testing and posturography, evolve independently. In this respect, some tests (e.g. pendular rotary vestibular testing) are inefficient to reveal a latent vestibular lesion. The vibration-induced nystagmus (VIN) test has been proposed, developed and used as a complementary tool to reveal vestibular asymmetry under direct observation and can always be detected in even longstanding or chronic well compensated UVL [9,10,13,15]. Indeed, vibration of the skull induces reproducible nystagmus in darkness in UVL patients, which begins with the stimulation (vibration), beats toward the healthy side whatever the stimulation location on the skull, and disappears with the end of the stimulation [9,10,13,15] . In addition, nystagmus has also been observed during posterior cervical muscle vibration, but its slow phase velocity is lower than during skull stimulation [10,13,16].

Vibration also has consequences on postural control with eyes closed in UVL patients and in healthy subjects. Magnusson et al. [18] observed that bilateral vibration applied on posterior cervical muscles induces mainly forward postural displacement in healthy subjects, which is in accordance with the previous observations of Popov et al. [24] who applied unilateral vibration. Moreover, the ipsilesional vibration of posterior cervical muscles induces postural deviations toward the lesion side [24,32] or backward [24] in patients with acute or sub-acute UVL. In addition, Lacour et al. [17] described in UVL patients (neurotomy in Menière's disease) a faster and more efficient compensation of the static vestibular deficits (e.g. subjective visual vertical (SVV) or static posturography) than for the dynamic vestibular deficits (e.g. gain of the vestibulo-ocular reflex (VOR)).

To our knowledge, no study has investigated the effect of vibration on postural control in chronic, severe or profound, UVL patients. With eyes closed, vibration is expected to induce a medio-lateral deviation toward the lesion side. This study thus aimed to investigate, in eyes open and closed conditions, the effects of vibration on postural control in compensated chronic UVL patients and to evaluate its clinical relevance.

2. Methods

2.1. Participants

Twelve patients with compensated left UVL participated in the study (median age $= 54 \pm$ interquartile range (IQR) $= 8$ years; median weight $= 64 \pm 26$ kg). The vestibular unilateral weakness ranging from 88% to 100% at the caloric test indicated a severe hypofunction or areflexia (Table 1). The patients thus presented total or profound UVL. The mean time since the onset of UVL was 65 months (median: 32 ± 25 months) (Table 1). Symptoms and signs of acute UVL were absent in each patient: absence of vertiginous symptoms, dizziness or clinical balance disorders (absence of definite deviation at the Fukuda-Unterberger test, absence of oscillopsia during rapid head or body movements); absence of significant directional preponderance to the pendular rotary vestibular testing ($< 20\%$ asymmetry on the standard directional preponderance formula and a DC Bias < 2); and SVV normalization (deviation $< 2.8°$). Nine healthy participants, without any vestibular disease, were included in the control group (median age $= 43 \pm 15$ years; median weight $= 71 \pm 12$ kg). They had no hearing problem and a normal caloric test. Each participant had no history of psychiatric or neurological disorders, or showed any orthopaedic disorders either of the trunk or the lower limbs that could affect postural performances. Moreover, neither the patients nor the control subjects had taken any medication. Tests were carried out in the Department of ENT, Briançon Hospital. All the participants gave written informed consent before data collection, in accordance with the current ethical laws in France.

Table 1
Characteristic of the population. Population of left unilateral vestibular lesion (UVL) group with pathology details, vestibular unilateral weakness (determined by caloric test), subjective visual vertical (SVV) and time to onset of the vestibular lesion. Concerning the caloric test, a vestibular unilateral weakness higher than 20% indicated a hypofunction while a vestibular unilateral weakness higher than 80% indicated a profound hypofunction. The SVV was impaired when the deviation was higher than 2.8°. Positive values indicated that SVV is deflected toward the right. Negative values indicated that SVV is deflected toward the left. (—): missing data

UVL group ($n = 12$)	Pathology	Vestibular unilateral weakness (in %)	SVV (in °)	Time to onset (in months)
1	Translabyrinthine ablation of vestibular schwannoma	100	2.1	118
2	Translabyrinthine ablation of vestibular schwannoma	100	−2.4	52
3	Translabyrinthine ablation of vestibular schwannoma	100	−1.1	25
4	Vestibular neurectomy	100	1.0	35
5	Vestibular neurectomy	98	—	25
6	Intratympanic Gentamicin labyrinthectomy	97	0.5	25
7	Intratympanic Gentamicin labyrinthectomy	100	−2.0	49
8	Intratympanic Gentamicin labyrinthectomy	90	—	49
9	Intratympanic Gentamicin labyrinthectomy	100	−0.5	29
10	Vestibular Neuritis	99	−0.6	6
11	Vestibular Neuritis	88	1.0	10
12	Major cochleo-vestibular hypoplasia	99	—	360

2.2. Vibratory stimulation

Vibration was induced during 10 s by a portable 85 Hz-vibratory stimulator fixed by rubber straps (Fig. 1) [18]. Vibratory stimulation was applied on vertex, right and left mastoid processes (i.e. level with the external hearing duct), right and left posterior cervical muscles (i.e. on the inferior 1/3 part of the ascending trapezius muscle), and right and left triceps surae (i.e. on junction of Achilles tendon and muscle), successively. Videonystagmography (i.e. eyes open behind a videoscopic helmet in a dark room) was used to record the possible VIN occurrence. The test was considered positive when the VIN was reproducible, began with the stimulation, beat toward healthy side and disappeared with the stimulation withdrawal [10]. The slow phase eye velocity (in °/s) of the VIN was recorded.

The same body locations were stimulated by vibration during the 25.6 s of each situation of the balance control evaluation. Vibration was induced by the same vibratory stimulators (i.e. portable 85 Hz-vibratory stimulators) fixed by rubber straps (Fig. 1) [18] to avoid participant's displacements in the direction of the push that could be induced by hand-held vibratory stimulations.

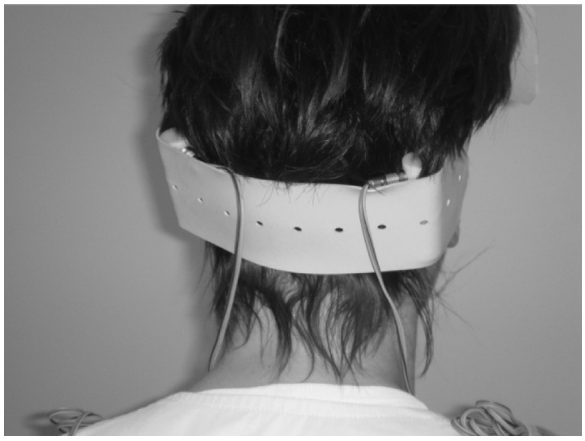

Fig. 1. Vibration was induced by a portable vibratory stimulator fixed by rubber straps. Vibratory stimulators were fixed on both sides for mastoids (here), posterior cervical muscles and triceps surae, but were activated only unilaterally. For mastoid vibration, stimulators were applied over the projection of the level of external meatus and lateral canal, avoiding the tip of the mastoid bone.

2.3. Balance control evaluation

All participants were tested on a vertical force platform (Medicapteurs, Balma, France) mounted on three strain-gauge force transducers, providing a measurement of the body sway in terms of displacement of the centre of foot pressure (CoP) in a two-dimensional horizontal plane (recording time: 25.6 s, acquisition frequency: 40 Hz). Participants were instructed to maintain a barefoot upright stance, feet 30° apart and arms along the sides, with as little postural sway as possible in different situations, with (see "*vibratory stimulation*" section for details) or without (standard situation) vibration, performed in eyes open (EO) (looking straight forward at a dot at eye level on a wall) and eyes closed (EC) conditions. Between each posturographic evaluation, the participants were allowed to rest and sit down for one min. Anteroposterior sway (in mm) was recorded; negative values and positive values represent backward and forward CoP mean positions, respectively. Medio-lateral sway (in mm) was recorded; negative values and positive values represent CoP mean po-

Table 2
Vibration-induced nystagmus test. Slow phase velocity (in °/s) of the vibration-induced nystagmus (VIN) observed during vibration of vertex, right and left mastoids, right and left posterior cervical muscles, and right and left triceps surae in unilateral vestibular lesion (UVL) group

Patient ($n = 12$)	1	2	3	4	5	6	7	8	9	10	11	12	median (IQR)
Vertex (°/s)	4.3	0	2.2	0	5.0	0	0	6.1	3.2	2.0	3.4	0	2.10 (3.62)
Right mastoid (°/s)	7.0	10.0	5.0	5.0	9.0	6.0	7.0	15.0	30.0	17.0	25.0	7.0	8.50 (8.75)
Left mastoid (°/s)	2.0	11.0	2.0	10.0	1.0	5.0	10.0	15.0	30.0	7.0	24.0	4.0	8.50 (8.50)
Right posterior cervical muscles (°/s)	6.4	8.2	9.5	4.0	0	4.0	8.0	11.0	8.8	2.5	9.1	4.0	7.20 (4.87)
Left posterior cervical muscles (°/s)	4.9	9.0	0	6.0	5.0	3.0	9.0	12.0	10.6	8.7	17.0	3.0	7.30 (4.97)
Right triceps surae (°/s)	0	0	0	0	0	0	0	0	0	0	0	0	0 (0)
Left triceps surae (°/s)	0	0	0	0	0	0	0	0	0	0	0	0	0 (0)

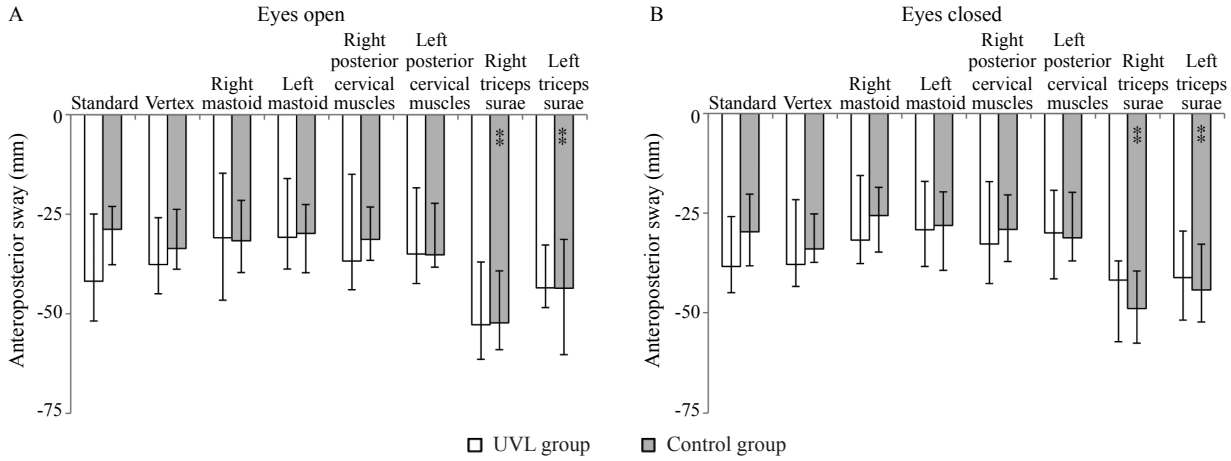

Fig. 2. Posturographic evaluation: anteroposterior sway – Median values, associated with the interquartile range, of anteroposterior sway (in mm) of the center of foot pressure in standard situation and during vibration of vertex, right and left mastoids, right and left posterior cervical muscles, right and left triceps surae in eyes open (panel A) or eyes closed (panel B) conditions in unilateral vestibular lesion (UVL) group and control group. Intragroup comparisons (compared to standard situation): $^{**}p \leqslant 0.01$.

sition to the left and the right, respectively. Sway area (in mm^2) and sway path (in mm) displacements, which characterized CoP movements, were recorded [23].

2.4. Statistical analysis

Quantitative data were expressed as medians associated with an interquartile range (IQR). On account of the relatively small sample size, intergroup comparisons were performed using the non-parametric Mann-Whitney Test. The intragroup comparisons were performed using the non-parametric Wilcoxon Test where postural parameters recorded during vibration were compared to those recorded in standard situation in both visual conditions. For controlling the False-Discovery Rate (FDR), the explorative Simes procedure (a stepwise multiple testing procedure) was used [2,26]. $H_1 \ldots H_m$ were the null hypotheses and $p_1 \ldots p_m$ were their corresponding p-values, where m was the number of tests (i.e. eight in our study). These p-values were ranked in an increasing order. For α (5%), the largest k were searched such that $p_{(k)} \leqslant \alpha(k/m)$ and all H_i for $i = 1, \ldots, k$ were rejected.

3. Results

No significant difference was observed for age and weight between the two groups.

3.1. Vibration-induced nystagmus

In the UVL group, a nystagmus beating toward the intact side was found during vibration of vertex, right and left mastoids, and right and left posterior cervical muscles. The slow phase velocity of the VIN is reported for each patient in Table 2. During the vibration of the triceps surae, no VIN was observed. In the control group, no VIN was observed whatever the location of the vibration.

3.2. Vibration-induced sway

No difference of anteroposterior sway was found be-

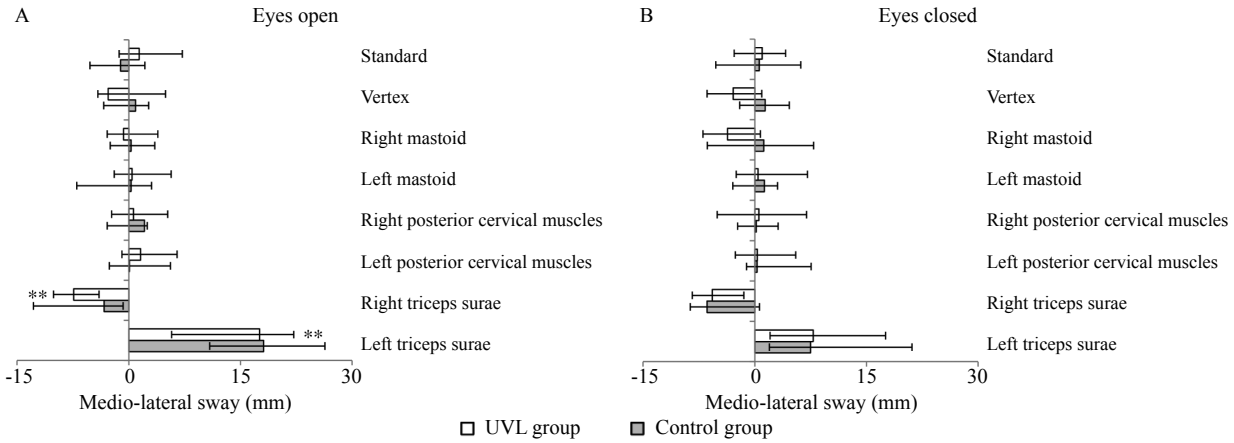

Fig. 3. Posturographic evaluation: medio-lateral sway – Median values, associated with the interquartile range, of medio-lateral sway (in mm) of the center of foot pressure in standard situation and during vibration of vertex, right and left mastoids, right and left posterior cervical muscles, right and left triceps surae in eyes open (panel A) or eyes closed (panel B) conditions in unilateral vestibular lesion (UVL) group and control group. Intragroup comparisons (compared to standard situation): $^{**}p \leqslant 0.01$.

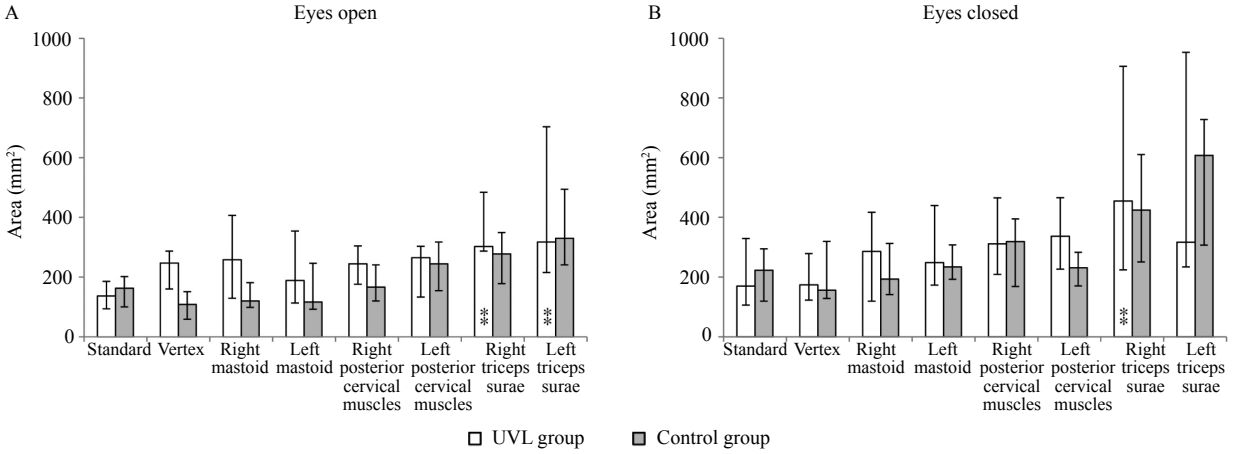

Fig. 4. Posturographic evaluation: area – Median values, associated with the interquartile range, of area (in mm^2) of the center of foot pressure in standard situation and during vibration of vertex, right and left mastoids, right and left posterior cervical muscles, right and left triceps surae in eyes open (panel A) or eyes closed (panel B) conditions in unilateral vestibular lesion (UVL) group and control group. Intragroup comparisons (compared to standard situation): $^{**}p \leqslant 0.01$.

tween the two groups for all stimulations (Fig. 2). In the UVL group, no significant anteroposterior deviation was observed. In the control group, CoP moved backward with vibration of right (eyes open: $z = -2.65$, $p = 0.008$; eyes closed: $z = -2.65$, $p = 0.008$) and left (eyes open: $z = -2.58$, $p = 0.010$; eyes closed: $z = -2.65$, $p = 0.008$) triceps surae.

No difference of medio-lateral sway was found between the two groups (Fig. 3). In the UVL group, CoP deviated to the left with vibration of right triceps surae (eyes open: $z = -3.09$, $p = 0.002$) and to the right with vibration of left triceps surae (eyes open: $z = -2.75$, $p = 0.006$). No significant deviation was observed in the control group.

No difference of sway area was found between the two groups (Fig. 4). In the UVL group, the sway area increased with vibration of the right (eyes open: $z = -3.09$, $p = 0.002$; eyes closed: $z = -2.88$, $p = 0.004$) and left (eyes open: $z = -2.62$, $p = 0.009$) triceps surae. No significant modification of sway area was observed in the control group.

Higher sway path values were observed in the UVL group than in control group during the vibration of the right (eyes open: $z = -2.49$, $p = 0.013$) and left (eyes open: $z = -2.42$, $p = 0.016$) mastoids, and the right (eyes open: $z = -2.84$, $p = 0.004$) and left

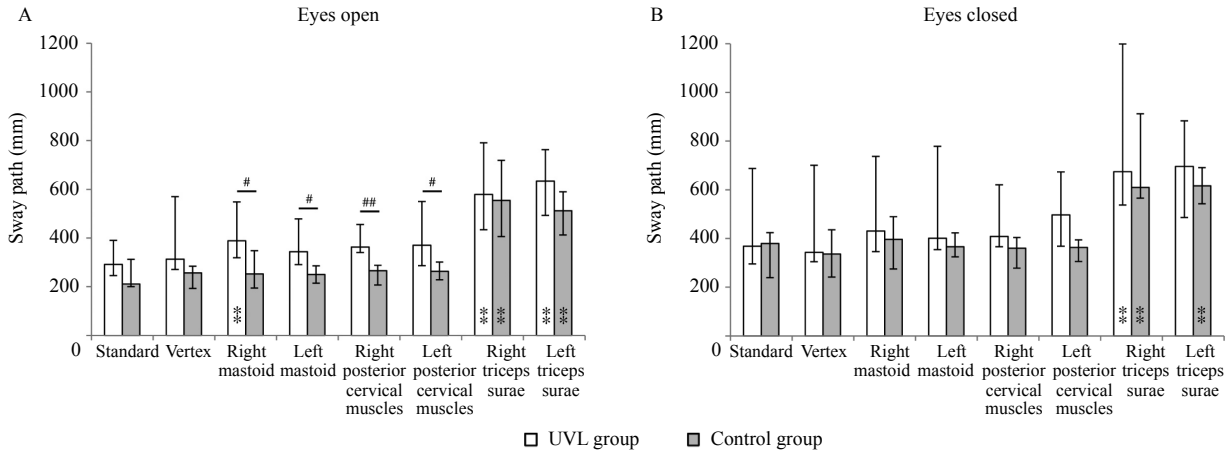

Fig. 5. Posturographic evaluation: sway path – Median values, associated with the interquartile range, of sway path (in mm) of the center of foot pressure in standard situation and during vibration of vertex, right and left mastoids, right and left posterior cervical muscles, right and left triceps surae in eyes open (panel A) or eyes closed (panel B) conditions in unilateral vestibular lesions (UVL) group and control group. Intragroup comparisons (compared to standard situation): $^{**}p \leqslant 0.01$. Intergroup comparisons (UVL group vs. control group): $^{\#}p \leqslant 0.05$, $^{\#\#}p \leqslant 0.01$.

(eyes open: $z = -2.27$, $p = 0.023$) posterior cervical muscles (Fig. 5). In the UVL group, the sway path increased with vibration of right mastoid (eyes open: $z = -2.62$, $p = 0.009$), and right (eyes open: $z = -3.09$, $p = 0.002$; eyes closed: $z = -3.09$, $p = 0.002$) and left (eyes open: $z = -3.09$, $p = 0.002$) triceps surae. In the control group, the sway path increased with vibration of right (eyes open: $z = -2.65$, $p = 0.008$; eyes closed: $z = -2.65$, $p = 0.008$) and left (eyes open: $z = -2.65$, $p = 0.008$, eyes closed: $z = -2.65$, $p = 0.008$) triceps surae.

4. Discussion

This study investigated the effects of body vibration, especially skull vibration, on postural control in chronic compensated unilateral vestibular lesion (UVL) patients. In the UVL group, postural control impairments were observed in the eyes open condition during vibration of the mastoids or posterior cervical muscles. Conversely, no significant difference in sway was observed between UVL patients and controls in the eyes closed condition. Moreover, triceps surae vibration induced nearly similar postural modifications in both groups. The vibration-induced sway (e.g. during mastoids or posterior cervical muscles) was quite small (even though it was statistically significant in the eyes open condition) and thus unlikely to be useful clinically.

During skull (i.e. ipsi- and contralesional mastoids) vibration, postural performances were more impaired in the UVL group than in control group in the eyes open condition. Vibration to the mastoid bone (or to the posterior cervical muscles) may result in a direct stimulation of the intact vestibular receptors [15], especially the utricle [8,28], which in turn activate the VOR [31]. This vibration induces a nystagmus leading to blur. Visual input is therefore inefficient because of the loss of visual landmarks. Moreover, vibration of the mastoids has been shown to induce tonic ocular torsion leading to modification of subjective visual horizontal [15]. In this respect, visual input in UVL patients is therefore unreliable during skull vibration. Moreover, Cornell et al. [6] observed in control subjects, during skull vibration, very small eye movements minimally affected by gaze position. However, these changes are probably less important than in UVL patients who have asymmetric vestibular function. Control subjects may therefore use visual cues more efficiently than UVL patients. However, no relation between postural sway and subjective visual horizontal and vertical was observed in UVL patients [12]. The postural impairments during skull vibration in the eyes open condition may also indicate that UVL patients have difficulties shifting toward other reliable sensory inputs (i.e. proprioception). Postural stability decreased more in the UVL group than in the control group in the eyes open condition during vibration of ipsilesional or contralesional posterior cervical muscles. In normal subjects bilateral skull vibration gives little or no postural reactions while bilateral vibration of posterior cervical muscles induces postural reactions [1] within 70–90 ms and the cervical in-

put seems therefore to dominate over the vestibular one [18]. However the situation can be assumed to be different in patients with vestibular asymmetries. First, skull vibration will cause an asymmetric vestibular input in the case of a unilateral vestibular loss as there is an asymmetric disposition of the otolithic organs. Vibration to posterior cervical muscles might thus to some extent propagate to the healthy vestibular organ and induce tonic ocular torsion limiting therefore the efficient use of visual input [15]. Secondly, a vestibular asymmetry may cause an asymmetric vestibulo-spinal activation as seen in the Fukuda/Unterberger test, thus causing a cervical stimulation to return an asymmetric response in its own right. Indeed, the changes in visual perception induced by neck muscle vibration might represent an increased central weighting of somatosensory neck information from the side with the lesion, which substitutes for a missing vestibular input [27]. Moreover, cervical muscular afferents seem to play a dominant role over vestibular afferents [18] and the postural impairments could also be explained by proprioceptive error signals, induced by the neck vibration, combined with an asymmetrical vestibular input, due to a UVL, which provide an erroneous representation of head position in patients resulting in a redirection of their body sway [24].

Vestibular compensation, occurring over weeks and months after UVL, leads to the improvement of postural stability and the normalization of the SVV which occurs in most cases in 12 months [17,30]. Panosian and Paige [21] have shown that head-shaking test in the horizontal plane, which generates nystagmus in UVL patients, induces transient postural impairments recorded by dynamic posturography. As their posturographic results were not congruent with caloric asymmetry, they concluded that the association of the head-shaking test with dynamic posturography depicted more accurately on uncompensated vestibulopathy. In our study, vibration of the skull or posterior cervical muscles did not lead to postural decompensation in the eyes closed condition. Moreover, the lack of medio-lateral sway during vibration indicates also that the postural decompensation is probably not involved. In the eyes closed condition, Yagi et al. [32] observed an ipsilateral sway toward the lesion side in UVL patients (intragroup comparison) when vibration was applied on the dorsal neck muscles. Moreover, Popov et al. [24] observed significant postural modifications (deviation toward the lesion side and backward) after the ipsilesional vibration of cervical posterior muscles. In our study, no difference was observed between UVL patients and controls in the eyes closed condition. The differences of results could at least partly be explained by the duration of the lesions (i.e. 4 months in the study of Popov et al. [24] *vs.* 65 months in our study).

Vibration of the triceps surae induced mainly backward and controlateral displacements, and increased postural instability in both groups. These displacements, which do not differ from those observed in previous studies in healthy subjects [3,4], are explained by the vibration-induced illusion of muscle elongation compensated by muscle contraction [11]. Actually, such stimulations do not concern directly the vestibule and the main treatment of the response is regulated at a lower level as a primarily spinal reflex. However, previous studies reported significant differences between UVL patients and healthy subjects during calf vibration in the eyes closed condition [25,27]. The difference with our results might reside in the patients' characteristics, like the degree of vestibular dysfunction and the duration of compensation. The vibratory stimulation of the triceps surae, which triggered a tonic segmental reflex reinforced by proprioceptive misinformation, cannot discriminate between healthy controls and chronic compensated UVL patients, who may have previously equalized basic vestibular tonus on both vestibular nuclei and the ability to switch to other receptors involved in postural control in order to maintain stance. Vibration does not reveal a latent vestibular deficit and does not induce *de novo* medio-lateral postural deviations in compensated UVL patients in the eye closed condition. Medio-lateral sway was expected to be a relevant parameter to assess postural decompensation, but this was not confirmed. The evidence for posturographic changes after vibration in the eyes closed condition is limited and the effect of vibration on posture in clinical practice seems marginal. Moreover, triceps surae vibration, which alters postural control, does not discriminate patients from controls, and does not concern primarily the vestibular pathways but mainly a spinal reflex. This vibration could be useful in rehabilitation but cannot be used as a tool to reveal a chronic and compensated UVL. Postural abnormalities in chronic UVL patients have only been observed by posturographic parameter analysis in the eyes open condition and seem therefore difficult to evaluate in the clinical setting. The clinical interest of the association of the vibration-induced nystagmus test with static posturography is thus limited, especially in chronic and well compensated UVL patients.

In conclusion, vibration applied to the mastoids or posterior cervical muscles, in compensated chronic

UVL patients, results in postural impairments only in the eyes open condition, probably through modifications of the proprioceptive inputs of the neck muscles and/or from stimulation of vestibular receptors on the intact side leading to visual perturbations generated by ocular movements. The postural response is therefore the result of several interacting mechanisms. However, the association of skull vibration and static posturography does not provide enough information (even though it was statistically significant in the eyes open condition) to motivate its introduction into the clinic. It does not uncover a chronic unilateral vestibular compensated asymmetry. Conversely, videonystagmography is sufficient to assess the effects of skull vibration and should be used as an adjunct to the caloric test.

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