Vibration-Induced Shift of the Subjective Visual Horizontal

A Sign of Unilateral Vestibular Deficit

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Background: Vibration to the head or neck excites vestibular and neck muscle spindle afferents. Can such vibrations improve the sensitivity of the subjective visual horizontal (SVH) test to chronic unilateral deficit of the vestibular system?

Design: Controlled experimental study.

Setting: Tertiary referral center.

Patients and Controls: Thirteen healthy subjects and 23 patients with chronic unilateral vestibular deficits after vestibular neurectomy or neurolabyrinthitis. Results of head-impulse test showed unilateral loss of function of all 3 semicircular canals in 14 patients and loss of anterior and lateral semicircular canals in 9 patients.

Intervention: Unilateral vibration (92 Hz; 0.6-mm amplitude) applied to sternocleidomastoid muscle (SCM) or mastoid bone.

Main Outcome Measure: Results of SVH test (in degrees).

Results: Without vibration, 13 of 23 patients and all healthy subjects had SVH of less than 3° (sensitivity, 43%; specificity, 100%). During vibration to the ipsilesional SCM, SVH increased to greater than 3° in 21 of 23 patients but in only 1 of 13 healthy subjects (sensitivity, 91%; specificity, 92%). The patient group had significantly greater SVH shifts to the ipsilesional side than did healthy subjects in response to SCM and mastoid bone vibration on either side. The SVH shift during vibration to the ipsilesional SCM was significantly greater than that during vibration to the contralesional muscle (P<.001) or to the mastoid bone on either side (P<.05). The vibration-induced SVH shift was significantly greater in those patients with loss of 3 semicircular canals than in those with loss of 2 (P<.01).

Conclusions: The sensitivity of the SVH test to chronic unilateral vestibular deficits can be improved by applying vibration to the SCM. The magnitude of vibratory SVH shift is related to the extent of unilateral deficit of the otolithic organs, vertical canals, or both.

SUBJECTS AND METHODS

PATIENTS AND CONTROL SUBJECTS

We studied 23 patients (12 men and 11 women; mean age, 53.6 years; range, 25-73 years) with well-defined unilateral vestibular deficits. They were recruited from the outpatient clinic at the Department of Neuro-otology, Royal Prince Alfred Hospital, Sydney, Australia, or from among patients in our clinical database. Head impulses in the planes of the 3 pairs of SCCs were studied in all patients to disclose the function of the 6 individual SCCs (Figure 1). Detailed descriptions of the equipment and the procedures of head-impulse testing have been presented elsewhere.11,13,16 Eleven patients, 8 with vestibular schwannoma and 3 with Meniere disease, had undergone unilateral vestibular neuromyotis. They had unilateral loss of function of all 3 SCCs. Twelve patients had permanent unilateral peripheral loss of vestibular function after vestibuloulabyrinthitis, and all had a unilateral canal paresis found on results of caloric testing. Of these 12 patients, 3 had lost function of all 3 SCCs, and 9 had lost function of the anterior and lateral SCCs (ie, “superior vestibular neuritis”).22 The average time since the vestibular lesion occurred was 35 months (range, 1-144 months). The clinical data of the patients are presented in Table 1.

We also studied 13 healthy subjects (7 men, 6 women; mean age, 32 years; range, 19-66 years) who were recruited from among the hospital and laboratory staff. None of the subjects had any history of cochlear, vestibular, central nervous system, or neck disorders. All subjects gave their written informed consent after being briefed about the examination. The local ethics committee approved the experimental procedures. All experiments were performed in accordance with the Helsinki II Declaration.

VIBRATORY STIMULUS

We used a battery-powered, handheld vibrator (Mini Vibrator NC70209; North Coast Medical, Inc, San Jose, Calif) with a frequency of 92 Hz and an amplitude of 0.6 mm. The frequency did not change with increased pressure to the neck or skull, as tested in a separate experiment on 3 healthy subjects. The vibrating silicon tip was semispherical, with a radius of 8 mm. For head vibrations, the tip of the mastoid bone was marked with a pen. The vibrator was positioned on the marked spot, perpendicular to the skin and held in position by hand (Figure 2A).

Unilateral vibration to the sternocleidomastoid muscle (SCM) has no effect on the SVH in healthy subjects. However, in a small series of patients with uVD, vibration applied to neck muscles or to the mastoid bone induces nystagmus.17,19 Vibratation has been shown to excite semicircular canal (SCC) and otolith afferents in different animal species.20-22 Consequently, the perceptual and ocular motor effects have been attributed to a direct vibratory stimulation of intact vestibular receptors.14.18,19 However, vibration also increases the firing in muscle spindle afferents,22 and others have attributed the vibratory effects to an interaction between neck proprioceptors and the vestibular system.11,13,15,16

MEASUREMENT OF THE SVH

The subject sat upright in a dark room with the head immobilized using a head holder. This consisted of a molded neck rest that covered the back of the head and neck and kept the head horizontal. The neck rest could be adjusted in the vertical and anterior-posterior directions to fit every subject. The subject’s head was firmly held in the neck rest by a forehead holder with 3 padded clamps that could be individually adjusted. In front of the subject at a distance of 1.3 m was a dim light bar, 2 mm wide and 120 mm long. It could be rotated about its midpoint by means of an electric motor and a remote-control device. The task for the subject was to adjust the bar to parallel alignment with the perceived gravitational horizon. Owing to ocular torsion toward the side of vestibular loss, a patient with a unilateral vestibular lesion will, in the absence of other visual cues, perceive a truly horizontal line as being tilted to the intact side. The same subject will set the light bar tilted to the side of the vestibular lesion when asked to set it to the horizon (Figure 2B). During each test, subjects performed 10 settings of the light bar with both eyes open. The average of the 10 settings was used as the measure of SVH. There was no time limit for performing the test. The time to complete 1 set of 10 settings ranged from 60 to 120 seconds across the subjects. Each subject first performed the SVH test without vibration (baseline), then while vibration was applied to the right- and left-sided SCMs and the right- and left-sided mastoid bones. The same test sequence was used for all subjects. Between each test, the subjects rested for at least 1 minute.

STATISTICAL ANALYSIS

To enable the recordings from all patients to be used for statistical analysis, individual data of SVH were pooled as if all patients had right-sided vestibular lesions. A 2-tailed t test for paired or unpaired observations was used to evaluate differences within the patient group and between patients and healthy subjects. A difference of P<.05 was considered statistically significant.
RESULTS

All healthy subjects and 13 of the 23 patients had SVH within ±3° without vibration (Table 1). This yields a sensitivity of the SVH test to chronic unilateral vestibular deficits of 43% and a specificity of 100%. During vibration to the ipsilesional SCM, SVH increased to greater than 3° in 21 of the 23 patients (Table 1 and Figure 3). Vibration applied to the mastoid bone or to the SCM of the healthy subjects had small and inconsistent effects (Figure 3, Figure 4, and Figure 5 and Table 2). Only 1 of these subjects increased his SVH to more than 3° during SCM vibration. Thus, although the sensitivity of the SVH test increased from 43% to 91%, the specificity de-

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Table 1. Summary of the Patients’ Clinical Data and SVH Results

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Lesion</th>
<th>Duration, mo</th>
<th>Baseline</th>
<th>Mastoid Bone</th>
<th>Sternocecidomastoid Muscle</th>
<th>Mastoid Bone</th>
<th>Sternocecidomastoid Muscle</th>
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<tbody>
<tr>
<td>1/F/45 VNx</td>
<td>30</td>
<td>4.9</td>
<td>ND</td>
<td>11.5</td>
<td>ND</td>
<td>16.4</td>
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</tr>
<tr>
<td>2/M/51 VNx</td>
<td>18</td>
<td>5.9</td>
<td>ND</td>
<td>10.0</td>
<td>ND</td>
<td>15.7</td>
<td></td>
</tr>
<tr>
<td>3/F/61 VNx</td>
<td>84</td>
<td>1.9</td>
<td>7.4</td>
<td>3.6</td>
<td>5.7</td>
<td>7.9</td>
<td></td>
</tr>
<tr>
<td>4/M/54 VNx</td>
<td>30</td>
<td>8.5</td>
<td>22.1</td>
<td>15.7</td>
<td>21.9</td>
<td>17.8</td>
<td></td>
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<tr>
<td>5/M/48 VN2</td>
<td>24</td>
<td>2.4</td>
<td>7.6</td>
<td>6.4</td>
<td>7.9</td>
<td>10.5</td>
<td></td>
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<tr>
<td>6/F/64 VN3</td>
<td>12</td>
<td>1.4</td>
<td>0.6</td>
<td>7.7</td>
<td>2.1</td>
<td>9.1</td>
<td></td>
</tr>
<tr>
<td>7/M/34 VN3</td>
<td>4</td>
<td>0.2</td>
<td>5.3</td>
<td>2.4</td>
<td>5.1</td>
<td>4.9</td>
<td></td>
</tr>
<tr>
<td>8/M/40 VNx</td>
<td>60</td>
<td>3.3</td>
<td>11.6</td>
<td>6.6</td>
<td>9.9</td>
<td>8.2</td>
<td></td>
</tr>
<tr>
<td>9/M/25 VN3</td>
<td>2</td>
<td>4.5</td>
<td>4.8</td>
<td>6.5</td>
<td>6.8</td>
<td>9.2</td>
<td></td>
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<tr>
<td>10/F/68 VNx</td>
<td>96</td>
<td>0.2</td>
<td>5.3</td>
<td>2.4</td>
<td>5.1</td>
<td>4.9</td>
<td></td>
</tr>
<tr>
<td>11/M/67 VN2</td>
<td>24</td>
<td>4.0</td>
<td>6.4</td>
<td>4.0</td>
<td>5.8</td>
<td>8.0</td>
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<tr>
<td>12/M/73 VNx</td>
<td>36</td>
<td>3.4</td>
<td>4.0</td>
<td>5.5</td>
<td>4.7</td>
<td>7.0</td>
<td></td>
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<tr>
<td>13/F/52 VNx</td>
<td>42</td>
<td>5.3</td>
<td>3.7</td>
<td>3.7</td>
<td>11.0</td>
<td>8.8</td>
<td></td>
</tr>
<tr>
<td>14/F/57 VN2</td>
<td>3</td>
<td>1.1</td>
<td>2.2</td>
<td>3.5</td>
<td>3.9</td>
<td>4.6</td>
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<tr>
<td>15/F/69 VNx</td>
<td>144</td>
<td>6.1</td>
<td>6.1</td>
<td>8.0</td>
<td>10.0</td>
<td>9.4</td>
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<tr>
<td>16/M/55 VN2</td>
<td>11</td>
<td>2.8</td>
<td>0.9</td>
<td>3.7</td>
<td>2.8</td>
<td>6.1</td>
<td></td>
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<tr>
<td>17/F/65 VNx</td>
<td>144</td>
<td>0.6</td>
<td>0.1</td>
<td>2.5</td>
<td>3.0</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>18/M/53 VN2</td>
<td>3</td>
<td>2.2</td>
<td>2.2</td>
<td>3.1</td>
<td>3.9</td>
<td>5.3</td>
<td></td>
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<tr>
<td>19/M/43 VN2</td>
<td>12</td>
<td>0.4</td>
<td>0.1</td>
<td>0.5</td>
<td>1.8</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>20/M/65 VN2</td>
<td>1</td>
<td>0.3</td>
<td>2.3</td>
<td>0.3</td>
<td>0.8</td>
<td>2.8</td>
<td></td>
</tr>
<tr>
<td>21/F/43 VN2</td>
<td>10</td>
<td>3.6</td>
<td>6.3</td>
<td>5.8</td>
<td>6.0</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>22/F/68 VN2</td>
<td>5</td>
<td>2.7</td>
<td>3.6</td>
<td>3.2</td>
<td>3.6</td>
<td>3.9</td>
<td></td>
</tr>
<tr>
<td>23/F/32 VNx</td>
<td>60</td>
<td>2.4</td>
<td>1.9</td>
<td>0.3</td>
<td>1.0</td>
<td>2.6</td>
<td></td>
</tr>
</tbody>
</table>

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increased slightly from 100% to 92% during SCM vibration (Figure 3).

The average baseline SVH was significantly larger in patients than in healthy subjects (P<.001) (Tables 1 and 2). Mastoid bone and SCM vibration shifted the SVH to the ipsilesional side, irrespective of the side vibrated, ie, clockwise (to the right side) in patients with right-sided vestibular lesions and counterclockwise (to the left side) in patients with left-sided lesions, except for patients 13 and 23 (Figures 4 and 5). The maximal SVH shift was 13.5° during mastoid bone vibration and 11.5° during SCM vibration (Figure 5). The vibration-induced shifts in SVH were significantly larger in patients than in healthy subjects (P<.001) (Table 2).

Vibration to the ipsilesional SCM shifted the SVH significantly more than did vibration to the contralateral side (P<.001) or vibration to the mastoid bones on either side (P<.05). No significant differences between the shifts of the SVH were found during vibration to the contralateral SCM and vibration to the mastoid bone on either side (P=.34) or between vibration to the mastoid bone on either side (P=.20) (Table 2).
There was no difference in the baseline SVH between patients with unilateral loss of 3 SCCs and those with loss of 2 SCCs (P = .25). The patients with loss of 3 SCCs showed significantly larger SVH shifts than patients with loss of 2 SCCs did in response to SCM vibration (P < .05) and a tendency to larger shifts during mastoid bone vibration (P = .10) (Table 2). If SVH results from mastoid bone vibration to the ipsilesional and contralesional sides were pooled together, patients with loss of 3 SCCs had significantly larger SVH shifts (mean, 3.8°; 95% confidence interval [CI], 1.4°-6.2°) than patients with loss of 2 SCCs (mean, 1.9°; 95% CI, 1.1°-2.6°; P < .05). Pooled data from SCM vibration to both sides showed that the patients with loss of 3 SCCs had significantly larger SVH shifts (mean, 4.4°; 95% CI, 2.6°-6.2°) than patients with loss of 2 SCCs (mean, 2.3°; 95% CI, 1.4°-3.2°; P < .01).

Comparison of the vibration-induced effects on the SVH with an independent test of otolith function would be ideal. Unfortunately, we have no direct test of utricular function. Ipsilateral myogenic potentials can be recorded from tonically activated SCMs during repeated monaural auditory stimulation (vestibular evoked myogenic potentials [VEMPs]) and probably reflect saccule function. In 6 of the 9 patients with loss of 2 SCCs, we had recordings of VEMPs. No differences were found in baseline SVH or in vibration-induced shift of the SVH between the 3 patients with loss of VEMPs on the ipsilesional side and the 3 patients with preserved VEMPs. Thus, we had to rely on results of head-impulse testing of the SCCs to get a reliable measure-
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Table 2. Baseline and Vibration-Induced Shifts in SVH in Healthy Subjects and Patients With uVD *

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Baseline SVH, Degrees</th>
<th>Vibration on Contralesional Side</th>
<th>Vibration on Ipsilesional Side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absolute</td>
<td>Shift</td>
<td>Absolute</td>
</tr>
<tr>
<td>Healthy subjects (n = 13)</td>
<td>0.4 (-0.1 to 0.9)</td>
<td>0.1 (-0.5 to 0.6)</td>
<td>0.9 (0.6 to 1.1)</td>
</tr>
<tr>
<td>Patients with uVD (n = 23)</td>
<td>2.8 (1.8 to 3.8)</td>
<td>5.1 (3.0 to 7.1)</td>
<td>2.7 (1.1 to 4.3)</td>
</tr>
<tr>
<td>Patients with loss of 2 SCCs (n = 9)</td>
<td>2.2 (1.3 to 3.0)</td>
<td>3.5 (1.8 to 5.2)</td>
<td>1.6 (0.6 to 2.7)</td>
</tr>
<tr>
<td>Patients with loss of 3 SCCs (n = 14)</td>
<td>3.2 (1.7 to 4.7)</td>
<td>6.2 (3.0 to 9.4)</td>
<td>3.5 (0.8 to 6.1)</td>
</tr>
</tbody>
</table>

*Data are given as mean (95% confidence interval). SVH indicates subjective visual horizontal; uVD, unilateral vestibular deafferentation; Absolute, SVH during vibration; Shift, SVH during vibration minus SVH during baseline; ellipses, not applicable; and SCC, semicircular canal.

The net effect of an oscillating mechanical stimulus delivered to the hair bundle of a vestibular receptor cell is excitatory.29 Vibrations with frequencies above 80 Hz delivered to the heads of squirrel monkeys have been shown to excite SCC and otolith afferents.30 Thus, a possible explanation of our results is that vibration to the mastoid bone or to the SCMs results in a direct vibratory stimulation of the intact vestibular receptors. However, we found that the shift of the SVH induced by ipsilesional SCM vibration was significantly larger than that induced by contralesional vibration or by vibration to the mastoid bone on either side. This is in accord with previous reports of changes in visual perception in yaw13 and roll34 induced by neck muscle vibration and might represent an increased central weighting of somatosensory neck information from the side with the lesion, which substitutes for missing vestibular input.13

The neck muscle vibrations of previous studies were standardized by adjusting the position of the vibrator until the subject perceived an illusion of movement of a stationary visual target.11-13,16 This position dependency has been used as an argument against vibratory stimulation of vestibular receptors.11 However, the positioning of the vibrator when it is applied to the head also affects the direction of perceptual illusions. Vibration to the top of the head induces illusions of vertical target movement, and vibration to the mastoid bone induces illusions of horizontal movement.31 Vibration applied to the mastoid bone might also propagate to neck muscles and thus stimulate the neck proprioceptors. However, propagation of vibration from the skull is probably not confined to those neck muscles that induce movements in a certain plane. The direction of illusions of movement during vibration to a muscle depends on the natural action of the vibrated muscle.30 The SCMs are contracted or stretched during head rotations about the naso-occipital axis,31 and perceptual effects induced by SCM vibration might thus be presumed to be in the roll plane. A possible way to differentiate the effects of vestibular stimulation vs neck muscle afference might be to stimulate both SCMs simultaneously. As the afferent information from the muscle spindles then would signal neck extension (bilateral lengthening) instead of roll tilt (unilateral lengthening), any effects on the SVH would probably be due to stimulation of intact vestibular receptors.

Muscle spindle primary endings (type Ia) increase their firing harmonically in response to vibrations up to about 80 Hz, but at higher frequencies they start to fire in subharmonic patterns.25 Thus, 92 Hz, as used in our study, is an adequate frequency for stimulating muscle afferents. After a 30-second vibration, 40 seconds are required for the spindles of lower leg muscles in humans to return to normal resting activity and stretch sensitivity.32 In our study, the subjects rested at least 1 minute between the different vibrations. However, it is not known whether neck muscle spindles manifest the same adaptive behavior or whether there is central adaptation. Although there was no obvious order effect, a larger variability of the SVH shifts was found in response to mastoid bone vibration, which was always performed last in our

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test sequence. As the same stimulus sequence was used for all tested subjects, adaptation or fatigue might have accounted for this result.

During head tilt to one side, the SVH shifts to the opposite side, which is the so-called E-effect. If the pressure of the vibrator induced head tilts, we would expect an effect dependent on which side was vibrated. We did not find this. The effect of vibration was instead related to the side on which the vestibular lesion was located. Tactile information regarding earth horizontal might be conveyed to the subject undergoing testing by pressure from the chair and the head holder. This information remains unchanged during the test, and we believe that it did not influence the results.

To sum up, the results show that vibration applied to the head or neck is a simple way to increase the sensitivity of the SVH test to chronic unilateral vestibular deficits. During vibration to the SCM, the sensitivity of the test increased from 43% to 91%, whereas the specificity only decreased marginally from 100% to 92%. Patients with unilateral loss of all 3 SCCs showed larger vibration-induced shifts of SVH than did patients with loss of only the anterior and lateral SCCs. This indicates that the magnitude of the vibration-induced shifts in SVH reflects the extent of unilateral vertical SCC deficit or otolithic deficit or both, not the extent of the lateral canal deficit. The test results thus give information that cannot be gained from the caloric test results.

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REFERENCES


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