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.

Eleven patients, 8 with vestibular schwannoma and 3 with Meniere disease, had undergone unilateral vestibular neurectomy. They had unilateral loss of function of all 3 SCCs. Twelve patients had permanent unilateral peripheral loss of vestibular function after vestibular neuritis, 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"). 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 vibrator was positioned perpendicular to the skin overlying the mastoid bone behind the external ear canal and held in position by hand. For vibrations applied to the neck muscles, we chose to vibrate the SCM, as it is more superficial and easier to locate than the posterior neck muscles. To standardize the site of vibration, the belly of the SCM and the mastoid bone were palpated during active muscle contraction, and a point on the muscle belly 20 mm below 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). We did not change the position of the vibrator until an illusion of visual target movement was evoked as in previous studies. The same examiner (M.K.) delivered the vibrations to all subjects to reduce the variability of the stimulus.

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-

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>Mastoid Bone</th>
<th>Sternocleidomastoid Muscle</th>
<th>Sternocleidomastoid Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/45</td>
<td>VNx</td>
<td>30</td>
<td>4.9</td>
<td>ND</td>
<td>11.5</td>
<td>ND</td>
<td>18.4</td>
</tr>
<tr>
<td>2/M/51</td>
<td>VNx</td>
<td>18</td>
<td>5.9</td>
<td>ND</td>
<td>10.0</td>
<td>ND</td>
<td>15.7</td>
</tr>
<tr>
<td>3/F/61</td>
<td>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>
</tr>
<tr>
<td>4/M/54</td>
<td>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>
</tr>
<tr>
<td>5/M/48</td>
<td>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>
</tr>
<tr>
<td>6/F/64</td>
<td>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>
</tr>
<tr>
<td>7/M/34</td>
<td>VN3</td>
<td>4</td>
<td>0.5</td>
<td>4.7</td>
<td>4.1</td>
<td>3.4</td>
<td>7.1</td>
</tr>
<tr>
<td>8/M/40</td>
<td>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>
</tr>
<tr>
<td>9/M/25</td>
<td>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>
</tr>
<tr>
<td>10/F/68</td>
<td>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>
</tr>
<tr>
<td>11/M/67</td>
<td>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>
</tr>
<tr>
<td>12/M/73</td>
<td>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>
</tr>
<tr>
<td>13/F/52</td>
<td>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>
</tr>
<tr>
<td>14/F/57</td>
<td>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>
</tr>
<tr>
<td>15/F/69</td>
<td>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>
</tr>
<tr>
<td>16/M/55</td>
<td>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>
</tr>
<tr>
<td>17/F/65</td>
<td>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>
</tr>
<tr>
<td>18/M/53</td>
<td>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>
</tr>
<tr>
<td>19/M/43</td>
<td>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>
</tr>
<tr>
<td>20/M/65</td>
<td>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>
</tr>
<tr>
<td>21/F/43</td>
<td>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>
</tr>
<tr>
<td>22/F/68</td>
<td>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>
</tr>
<tr>
<td>23/F/32</td>
<td>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>
</tr>
</tbody>
</table>

*Statistics in bold indicate subjective visual horizontal (SVH) >3°; in italics, SVH tilted to the contralesional side. VNx indicates vestibular neurectomy; ND, no data; VN2, vestibular neurolabyrinthitis with deficits of anterior and lateral semicircular canals (SCCs); and VN3, vestibular neurolabyrinthitis with deficits of anterior, lateral, and posterior SCCs.
creased 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 $13.5^\circ$ during mastoid bone vibration and $11.5^\circ$ 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 contralesional 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 contralesional 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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**Figure 5.** Subjective visual horizontal (SVH) shift from baseline during vibration to the sternocleidomastoid muscles (SCMs) (A) and to the mastoid bones (B) in healthy subjects ($n = 13$) and patients with unilateral vestibular deafferentation ($n = 23$; mastoid bone vibration, $n = 21$). In all patients except patient 23, the largest SVH shift was toward the ipsilesional side, irrespective of the side of vibration. Other abbreviations are defined in the legend to Figure 4.
Vibration on Ispilesional Side
Sternocleidomastoid Muscle
Mastoid Bone

Roll24 induced by neck muscle vibration and might rep-
vibration; Shift, SVH during vibration minus SVH during baseline; ellipses, not applicable; and SCC, semicircular canal.

A high correlation between SVH and static tor-
sional eye position has been reported after uVD.26 In pa-
tients with vestibular neuritis, a high correlation was found
between the shifts in horizontal eye position and subject-
ive straight-ahead position induced by neck vibra-
tion.3 A possible way to differentiate the effects of vestibular
vibration might thus be presumed to be in the roll plane.

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 Ispilesional Side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absolute</td>
<td>Shift</td>
<td>Absolute</td>
</tr>
<tr>
<td>Healthy subjects</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>(n = 13)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients with uVD</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>(n = 23)</td>
<td></td>
<td></td>
<td>2.2 (1.2 to 3.2)</td>
</tr>
<tr>
<td>Patients with loss of 2 SCCs</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>(n = 9)</td>
<td></td>
<td></td>
<td>1.2 (0.3 to 2.1)</td>
</tr>
<tr>
<td>Patients with loss of 3 SCCs</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>
<tr>
<td>(n = 14)</td>
<td></td>
<td></td>
<td>2.8 (1.3 to 4.4)</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.

ment of the extent (total or partial loss) of the vestibular
lesion.

Vibration to the mastoid bone or SCMs on either side
induced a shift of the SVH toward the ipsilesional side
in our patients. To our knowledge, this is the first time
that vibration to the mastoid bone or contralesional SCM
has been shown to shift the SVH in patients with uVD. The
net effect of an oscillating mechanical stimulus deliv-
ered to the hair bundle of a vestibular receptor cell is
excitatory.29 Vibrations with frequencies above 80 Hz deliv-
ered to the heads of squirrel monkeys have been shown
to excite SCC and otolith afferents.30 Thus, a possible ex-
planation of our results is that vibration to the mastoid
bone or to the SCMs results in a direct vibratory stimu-
lization of the intact vestibular receptors. However, we
found that the shift of the SVH induced by ipsilesional
SCM vibration was significantly larger than that indi-
cated by contralesional vibration or by vibration to the
mastoid bone on either side. This is in accord with pre-
vious reports of changes in visual perception in yaw13 and
roll24 induced by neck muscle vibration and might rep-
resent an increased central weighting of somatosensory
note 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 un-
til the subject perceived an illusion of movement of a sta-
nary 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 mas-
toid bone might also propagate to neck muscles and thus
stimulate the neck proprioceptors. However, propaga-
tion of vibration from the skull is probably not confined
to those neck muscles that induce movements in a cer-
tain plane. The direction of illusions of movement
during vibration to a muscle depends on the natural ac-
tion of the vibrated muscle.30 The SCMs are contracted
or stretched during head rotations about the naso-
ocipital 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 stimu-
late both SCMs simultaneously. As the afferent informa-
tion from the muscle spindles then would signal neck ex-
tension (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 re-
quired for the spindles of lower leg muscles in humans
to return to normal resting activity and stretch sensitivi-
ty.32 In our study, the subjects rested at least 1 minute
during the different vibrations. However, it is not known
whether neck muscle spindles manifest the same adap-
tive behavior or whether there is central adaptation. Al-
though there was no obvious order effect, a larger vari-
bility of the SVH shifts was found in response to mastoid
bone vibration, which was always performed last in our
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.24 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, but 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