Short Tone Burst–Evoked Myogenic Potentials on the Sternocleidomastoid Muscle

Are These Potentials Also of Vestibular Origin?

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Objectives: To show that short tone bursts (STBs) evoke myogenic potentials from the sternocleidomastoid muscle (SCM) that are of vestibular origin.

Design: Evoked potential activity was recorded from the SCMs of normal volunteers and from patients with vestibulocochlear disorders.

Setting: This outpatient study was conducted at the Department of Otolaryngology, University of Tokyo, Tokyo, Japan.

Subjects: Nine normal volunteers and 30 patients (34 affected ears) with vestibulocochlear disorders were examined.

Intervention: Diagnostic.

Outcome measures: Sound-evoked myogenic potentials in response to STBs were recorded with surface electrodes over each SCM. Responses evoked by STBs in patients were compared with responses evoked by clicks.

Results: In all normal subjects, STBs (0.5, 1, and 2 kHz) evoked biphasic responses on the SCM ipsilateral to the stimulated ear; the same was true for clicks. Short tone bursts of 0.5 kHz evoked the largest responses, while STBs of 2 kHz evoked the smallest. In patients with vestibulocochlear disorders, responses to STBs of 0.5 kHz were similar to responses evoked by clicks. Thirty (88%) of the 34 affected ears demonstrated the same results with 0.5-kHz STBs and with clicks. Responses were present in patients with total or near-total hearing loss and intact vestibular function. Conversely, patients with preserved hearing but with absent or severely decreased vestibular function had absent or significantly decreased myogenic potentials evoked by STBs.

Conclusions: Short tone bursts as well as clicks can evoke myogenic potentials from the SCM. Myogenic potentials evoked by STBs are also probably of vestibular origin.

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It has been reported that relatively loud clicks can cause responses on the sternocleidomastoid muscle (SCM).1-6 This response is unique because it is probably of vestibular origin. Colebatch and Halmagyi1 first reported that this response disappeared after vestibular nerve section despite preservation of hearing. It has been called the vestibular evoked myogenic potential (VEMP).3-5 Neurophysiological studies using guinea pigs and cats revealed that vestibular nucleus neurons and primary vestibular afferents, especially saccular afferents, can respond to relatively loud sound stimuli.7-11 These findings support the theory that VEMP is probably of saccular origin. Furthermore, McCue and Guinan11 report that sound-sensitive vestibular afferents show a broad, V-shaped tuning curve, with the best frequencies between 0.5 and 1 kHz. Their findings suggest that VEMP can be evoked by short tone bursts (STBs) of 0.5 to 1 kHz as well as by clicks. We report the findings of VEMP by STB in normal subjects and patients with vestibulocochlear disorders. This article demonstrates that STBs evoke myogenic potentials on the SCM and that STB-evoked myogenic potentials on the SCM are probably of vestibular origin.

NORMAL SUBJECTS

Myogenic potentials on the SCM were evoked by STBs of 0.5, 1, and 2 kHz in all subjects (Figure 2). Amplitudes of the first positive-negative response showed...
SUBJECTS AND METHODS

Nine normal volunteers (7 men, 2 women; age range [median], 28-39 [33] years) on the medical staffs of our departments were enrolled as control subjects. Six normal subjects (6 men; age range [median], 28-38 [32.5] years) underwent testing by 3 kinds of STBs. Six normal subjects (4 men, 2 women; age range [median], 31-39 [33] years) underwent testing for interaural difference analyses with STBs of 0.5 kHz.

Thirty patients with vestibulocochlear disorders (34 affected ears; 12 men and 18 women; age range [median], 25-75 [47.5] years) underwent testing of STB-and click-evoked myogenic potentials on the SCM. Diagnoses of these patients were Ménière disease (n = 11), acoustic neuroma (n = 8), benign paroxysmal positional vertigo (n = 3), sensorineural hearing loss (n = 3), delayed endolymphatic hydrops (ipsilateral type, n = 2), vestibular neuritis (n = 2), and Ramsay Hunt syndrome (n = 1).

Recording methods have been described in detail elsewhere. Subjects were placed in the supine position and surface electromyographic (EMG) activity was recorded from symmetrical sites over the upper half of each SCM with a reference electrode on the lateral end of the upper sternum. During recording, subjects were instructed to rotate their heads to the opposite side of the stimulated ear to activate the SCM. EMG activity was monitored during recording to maintain muscle activity at a constant level. The EMG signal from the stimulated side was amplified and the bandpass was filtered (20-2 kHz). The stimulation rate was 5 Hz; analysis time was 50 ms.

STBs of 0.5, 1, and 2 kHz (95-dB normal hearing level; rise/fall time, 1 ms; plateau time, 2 ms) were presented to the right ear through a headphone. To avoid any effect from the order of the 3 STBs presented, we assigned 6 different orders to subjects. Responses to 100 stimuli were averaged twice. Frequency spectra of presented STBs were analyzed using a RION SA-27 one-third octave band real-time analyzer (RION Co Ltd, Kokubunji, Japan) (Figure 1). STBs of 0.5 kHz (95-dB normal hearing level; rise/fall time, 1 ms; plateau time, 2 ms) were presented through headphones to examine interaural differences.

For study subjects, STBs of 0.5 kHz (95-dB normal hearing level; rise/fall time, 1 ms; plateau time, 2 ms) and clicks (95-dB normal hearing level; 0.1 ms) were presented. Methods of recording were the same as those for normal subjects.

We analyzed the amplitude of the first positive-negative responses (P13-N23) for click VEMP2,4,5 on the SCM ipsilateral to the stimulated ear (Figure 2). The average of 2 runs was regarded as the amplitude. The evoked potential (EP) ratio was evaluated as 100 \([\frac{(Au - Aa)}{(Au + Aa)}]\), where \(Au\) is the amplitude on the unaffected side and \(Aa\) is the amplitude on the affected side. In normal subjects, the EP ratio was calculated as 100[(Ar - Al)/(Ar + Al)], where \(Ar\) indicates the amplitude on the right and \(Al\) the amplitude on the left.3

Patients also underwent pure-tone audiometry and caloric tests. Caloric nystagmus was recorded using an electroneystagmograph. Canal paresis was calculated using the maximal slow-phase eye velocity of caloric nystagmus.

relatively large intersubject differences because tonic EMG activities were controlled so that they were at the same level only in individual ears. Therefore, instead of absolute amplitudes, the ranks of amplitudes of different STBs were used for analysis. While the 0.5-kHz STB evoked the greatest response in every subject, the 2-kHz STB evoked the smallest responses (Figure 3). Concerning latencies of the first positive and negative peaks, we could not detect any tendency (Figure 4).

In the study of interaural differences, the EP ratio ranged from 4.3 to 25.6 (n = 6) (mean ± SD, 13.6 ± 10.2). We defined 34 (mean + 2 SDs) as the upper limit of the normal range of the EP ratio for a 0.5-kHz STB. We used the same upper limit of the normal range of the EP ratio for clicks.5
Figure 2. Short tone burst–evoked myogenic potentials on the sternocleidomastoid muscle in a normal subject (32-year-old man). All short tone bursts evoked biphasic responses similar to click-evoked vestibular evoked myogenic potentials. The largest response was evoked by short tone bursts of 0.5 kHz, the smallest response by short tone bursts of 2 kHz. I and II represent the first positive and negative peaks, respectively.

Figure 3. Amplitudes of the first positive-negative peak in 6 normal subjects. In all subjects, 0.5-kHz short tone bursts evoked the largest response and 2-kHz short tone bursts evoked the smallest response.

Figure 4. Latencies of the first positive (A) and negative (B) peaks in 6 normal subjects. We could not determine any tendency. Latencies (mean ± SD) of the first positive and negative peaks were 14.9 ± 0.53 and 23.5 ± 1.21 milliseconds, respectively.
PATIENTS WITH VESTIBULOCOCHLEAR DISORDERS

We summarized the results of 30 patients in Table 1. Short tone burst–evoked responses in 24 (71%) of the 34 ears were the same as those for click-evoked responses. However, when differentiating normal responses from abnormal responses, STB-evoked responses in 30 ears (88%) were the same as those for click-evoked responses. Only 4 ears (12%) showed different results. Three ears (9%) showed normal STB-evoked responses and decreased click-evoked

Table 1. Short Tone Burst (STB)–Evoked Myogenic Potentials (0.5 kHz) and Click-Evoked Myogenic Potentials

<table>
<thead>
<tr>
<th>Click-Evoked Myogenic Potentials</th>
<th>STB-Evoked Myogenic Potentials, No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Increased</td>
<td>1</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
</tr>
<tr>
<td>Decreased</td>
<td>0</td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 2. Results of Neuro-otological Testing in 8 Selected Patients

<table>
<thead>
<tr>
<th>Patient No./Age, y/Sex</th>
<th>Diagnosis</th>
<th>Click-Evoked Myogenic Potentials</th>
<th>STB-Evoked Myogenic Potentials</th>
<th>Pure-Tone Audiometry</th>
<th>Caloric Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/57/F</td>
<td>L sudden deafness</td>
<td>Normal</td>
<td>Normal</td>
<td>THL</td>
<td>Normal</td>
</tr>
<tr>
<td>2/25/M</td>
<td>R delayed endolymphatic hydrops</td>
<td>Increased</td>
<td>Increased</td>
<td>THL</td>
<td>Right CP, 21%</td>
</tr>
<tr>
<td>3/31/M</td>
<td>B sensorineural hearing loss</td>
<td>Normal</td>
<td>Normal</td>
<td>Nearly THL</td>
<td>Normal</td>
</tr>
<tr>
<td>4/33/M</td>
<td>L delayed endolymphatic hydrops</td>
<td>Decreased</td>
<td>Normal</td>
<td>THL</td>
<td>Normal</td>
</tr>
<tr>
<td>5/32/M</td>
<td>L acoustic tumor</td>
<td>Absent</td>
<td>Absent</td>
<td>34-dB HL</td>
<td>No response</td>
</tr>
<tr>
<td>6/32/F</td>
<td>L acoustic tumor</td>
<td>Decreased</td>
<td>Decreased</td>
<td>30-dB HL</td>
<td>Left CP, 33%</td>
</tr>
<tr>
<td>7/40/M</td>
<td>R vestibular neuritis</td>
<td>Absent</td>
<td>Decreased</td>
<td>8-dB HL</td>
<td>No response</td>
</tr>
<tr>
<td>8/58/M</td>
<td>R Ramsay Hunt syndrome</td>
<td>Absent</td>
<td>Absent</td>
<td>26-dB HL</td>
<td>No response</td>
</tr>
</tbody>
</table>

*STB indicates short tone burst; L, left; THL, total hearing loss; R, right; CP, canal paresis; B, bilateral; HL, and hearing level.

Figure 5. Short tone burst (STB)–evoked myogenic potentials on the sternocleidomastoid muscle in patients with vestibulocochlear disorders. A, A 57-year-old woman with sudden deafness showed total hearing loss but normal caloric responses in the left ear. Myogenic potentials evoked by 0.5-kHz STBs as well as clicks were normal. B, A 58-year-old man with right Ramsay Hunt syndrome showed absent caloric response but preserved hearing. Auditory brainstem responses were normal on both sides. Myogenic potentials evoked by 0.5-kHz STBs as well as clicks were absent on the right. I and II represent the first positive and negative peaks, respectively.
responses, while 1 ear (3%) showed normal click-evoked responses and decreased STB-evoked responses.

From these patients, we selected 8 (6 men and 2 women; age range [median], 25-58 [36.5] years) and divided them into 2 groups of 4 patients each. The first group included 3 patients with unilateral total hearing loss and 1 patient with bilateral nearly total hearing loss. These 4 patients were selected because they seemed to have residual vestibular function despite severe cochlear and/or cochlear nerve damage. The second group included patients who seemed to have significantly decreased or absent vestibular function unilaterally despite preserved cochlear function. While 1 of 2 patients in the second group with acoustic neuroma showed prolonged interpeak latencies, the other 3 patients showed normal auditory brainstem responses.

We summarized the results of these 2 groups in Table 2. All patients with unilateral total hearing loss or bilateral nearly total hearing loss (n = 4) showed myogenic responses to 0.5-kHz STBs on the affected side as well as the unaffected side (Figure 5, A). Evoked potential ratios were normal in 3 patients, while the amplitude in 1 patient with delayed endolymphatic hydrops was significantly increased on the affected side.

However, all patients who showed significantly decreased or absent caloric responses despite preserved hearing (n = 4) showed significantly decreased or absent responses to 0.5-kHz STBs on the affected side (Figure 5, B).

Our results clearly show that STBs as well as clicks can evoke myogenic potentials on the SCM ipsilateral to the stimulated ear. In all normal subjects, the greatest responses were to 0.5-kHz STBs. These results are consistent with neurophysiological findings reported by McCue and Guinan. They reported that sound-sensitive vestibular afferents showed a broad, V-shaped tuning curve with the best frequencies between 0.5 and 1 kHz.

The fact that STBs can evoke myogenic responses does not necessarily mean that STB-evoked myogenic potentials are of vestibular origin. However, when differentiating normal responses from abnormal responses, the results of STB-evoked responses in 30 ears (88%) were the same as those for click-evoked responses. Only 4 ears (12%) showed different results. Short tone bursts of 0.5 kHz can evoke myogenic potentials in patients with a totally or nearly totally deaf ear, while myogenic potentials were absent or significantly decreased in patients with vestibular disorders and preserved hearing. These results support the finding that STB-evoked myogenic potentials are probably of vestibular origin, similar to click-evoked myogenic potentials.

In this study, 4 ears (12%) showed different results. Three ears had normal STB-evoked responses and decreased click-evoked responses, while 1 ear had normal click-evoked responses and decreased STB-evoked responses. We cannot determine whether the results in these patients were false-negative or false-positive. Click-sensitive vestibular hair cells might differ from STB-sensitive vestibular hair cells. We assumed that it would be better to use 2 kinds of sound to confirm the results of sound-evoked potentials on the SCM.

In conclusion, we found that STBs can evoke myogenic potentials on the SCM. The amplitudes evoked by 0.5-kHz STBs were the largest among the 3 STBs used in this study (0.5, 1, and 2 kHz). Myogenic potentials evoked by STBs were also probably of vestibular origin. We can call STB-evoked myogenic responses STB VEMPs. These results are consistent with the neurophysiological findings of McCue and Guinan. To confirm the results of sound-evoked potentials on the SCM, we recommend using 2 kinds of sound (ie, click and 0.5-kHz STB).

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COMMENT

REFERENCES