

EMG Potentials Elicited by Forehead Taps in the Sternocleidomastoid Muscles and Lower Leg Muscles

— A study on patients with vestibular lesions —

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The aim of the present study was to determine whether a tap on the forehead evokes a vestibulospinal reflex in the sternocleidomastoid muscles and the lower leg muscles in patients with vestibular lesions. Although first positive-negative short-latency EMG potentials with a mean positive potential peak of about 12.6 ms and a mean negative potential peak of about 17.3 ms were found in the bilateral sternocleidomastoid muscles in normal subjects, they were lacking in patients with vestibular lesions. The first negative short-latency EMG potentials had a mean latency of 48.3 ± 3.1 ms (onset) to 98.3 ± 6.3 ms (end) in both gastrocnemius muscles in normal subjects, but they were delayed in patients with vestibular lesions.

Key Words : Forehead tap, Vestibulospinal reflex, Vestibular lesion, EMG potentials, Sternocleidomastoid muscle, Lower leg muscle

INTRODUCTION

Vestibulospinal reflexes from otoliths have been studied in unexpected falls in humans and cats. In humans a fall evoked an initial electromyography (EMG) potential with a latency of 60 to 80 ms in the lower limb muscles [6, 9]. Brandt and coworkers [1, 5] studied click-evoked myogenic reflexes (otolithic vestibulospinal reflex) of the lower leg in a patient with the otolithic Tullio phenomenon. They detected short-latency activation with latencies ranging from 47 to 80 ms for the ipsilateral tibialis anterior muscle. Halmagyi et al. [7] reported that a gentle head tap evoked short-latency EMG potentials of a mean latency of 9.8 ms in sternocleidomastoid muscles. We observed forehead tap-evoked myogenic reflexes with a mean positive potential peak of 12.6 ms and a mean negative potential peak of 17.3 ms in the bilateral sternocleidomastoid muscles as well as forehead tap-evoked myogenic reflexes with a mean latency of 48.3 ms to 98.3 ms in the bilateral gastrocnemius muscles in normal human subjects. The aim of the present study was to evaluate how

vestibulospinal reflexes in the sternocleidomastoid muscles and lower leg muscles elicited by a forehead tap in normal subjects differ from those elicited in patients with vestibular lesions.

SUBJECTS AND METHODS

The EMG activity elicited by head taps in the sternocleidomastoid muscles and the lower leg muscles (gastrocnemius muscle and tibialis anterior muscle) was measured according to the method of Halmagyi et al. [7]. For measurement of EMG activity in the sternocleidomastoid muscles, the subject lay in a supine position with the head elevated and eyes closed. For measurement of EMG activity in the lower leg muscles, the subject stood on a slanted platform with the eyes closed and toes tilted upward by 5° . Twenty normal human volunteers (22 to 42 years of age, mean age 29.6) participated in the study of EMG activity in the sternocleidomastoid muscles, and ten normal human subjects in the study on the lower leg muscles. Three patients with vestibular lesions in whom caloric responses were abolished (two with idiopathic bilateral vestibulopathy [2], and

one with von Recklinghausen's disease who underwent bilateral vestibular neurectomy) participated in both studies of EMG activity in the sternocleidomastoid muscles and lower leg muscles. Forehead taps were given at random manually with a reflex hammer. Rectified EMG responses of the sternocleidomastoid muscles and the lower leg muscles were recorded and averaged ($n = 100-200$). The active surface electrode was placed over the middle of each muscle and the reference electrode over each tendon. To trigger the averager, we used either an accelerometer mounted on the forehead or an electric switch affixed to the hammer. All subjects gave their informed consent to participate in the study after the experimental procedure had been explained in accordance with the Helsinki Declaration.

RESULTS

NORMAL HUMAN SUBJECTS

Tapping of the forehead evoked reflexes, namely the first positive-negative short-latency

EMG potentials and the second additional EMG potentials in both sternocleidomastoid muscles. The mean latency peak of the first positive EMG potential peak was about 12.6 ± 1.9 ms and that of the negative potential peak about 17.3 ± 1.7 ms bilaterally in a supine position (Figure 1). Forehead taps elicited short-latency inhibitory EMG responses of a mean latency of 48.3 ± 3.1 ms (onset) to 98.3 ± 6.3 ms (end) in both gastrocnemius muscles in an upright posture (Figure 2), but no EMG responses were elicited in the bilateral tibialis anterior muscles.

PATIENTS WITH VESTIBULAR LESIONS

The EMG in the sternocleidomastoid muscles showed the first short-latency EMG potential changes on the affected sides, but the second additional EMG potentials were found on both sides. The reflexes disappeared on the affected side muscles in two patients with idiopathic vestibulopathy (Figure 3) and one with von Recklinghausen's disease who underwent

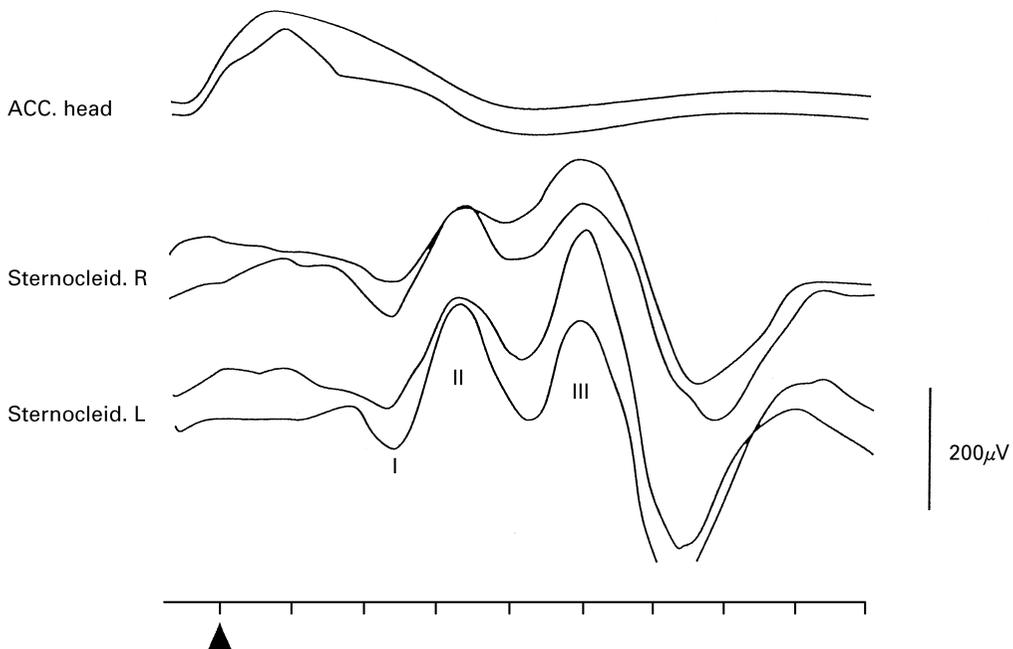


Fig. 1 EMG responses in the bilateral sternocleidomastoid muscles elicited by head taps on the forehead in a normal human subject. Top trace shows triggering by head taps, and the middle and bottom traces show the EMG potentials in the right and left sternocleidomastoid muscle respectively. Traces of two recordings are shown. Arrow shows the time of the trigger. Each time scale division equals 5 ms. Short-latency EMG potentials I; positive wave with a peak latency of 12.3 ms on the right and about 12.4 ms on the left. Negative EMG potentials II follow the first positive wave with a peak latency of 17.2 ms on the right and 17.4 ms on the left sternocleidomastoid muscle. An additional wave III was found on both sides.

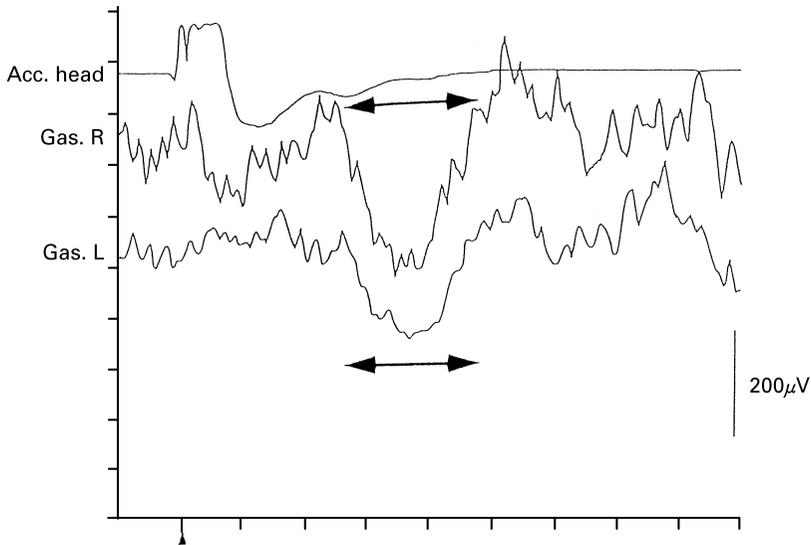


Fig. 2 EMG responses of the right and the left gastrocnemius (Gas. R, Gas. L) muscles elicited by forehead taps in a normal subject. Trigger: an electric switch affixed to the hammer. Acceleration of head (Acc. head) was measured by accelerometer mounted on the forehead. Top trace shows the triggering by forehead taps, and the middle and bottom show the EMG potentials in the right and left gastrocnemius muscles respectively. Arrow shows the time of trigger. Each time scale division equals 20 ms. Note that activity of the right gastrocnemius muscle is inhibited with a latency of 51.5 (onset) ms to 92.3 ms (end), and activity of the left gastrocnemius muscle is inhibited with a latency of 52.4 (onset) ms to 89.6 ms (end).

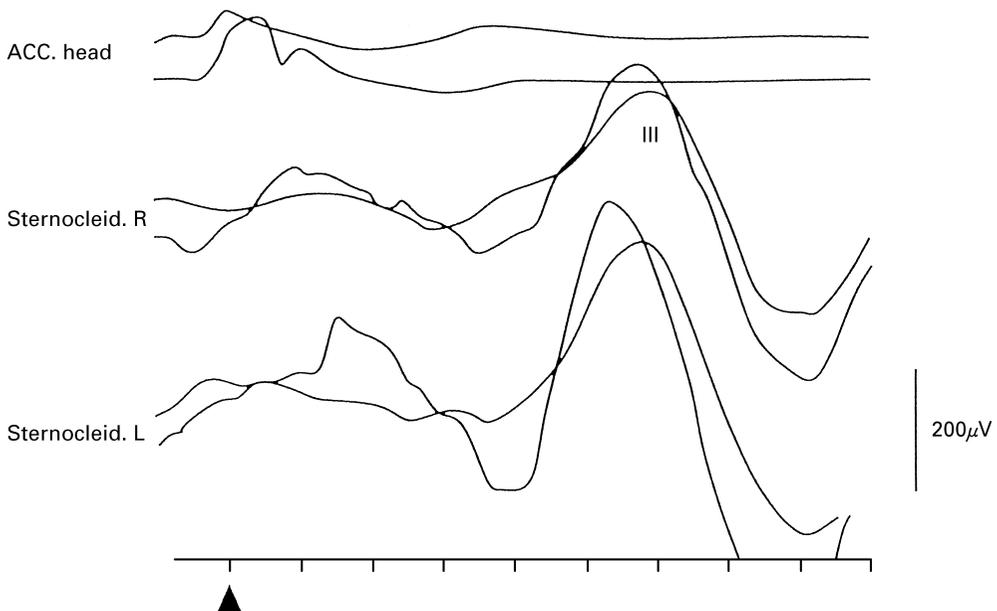


Fig. 3 EMG responses in the bilateral sternocleidomastoid muscles elicited by head taps on the forehead in a patient with bilateral vestibulopathy (idiopathic). Traces of two recordings are shown. Arrow shows the time of the trigger. Time scale division equals 5 ms. Short-latency EMG potentials I & II in the sternocleidomastoid muscles on both affected sides were abolished. An additional wave III was found on both sides.

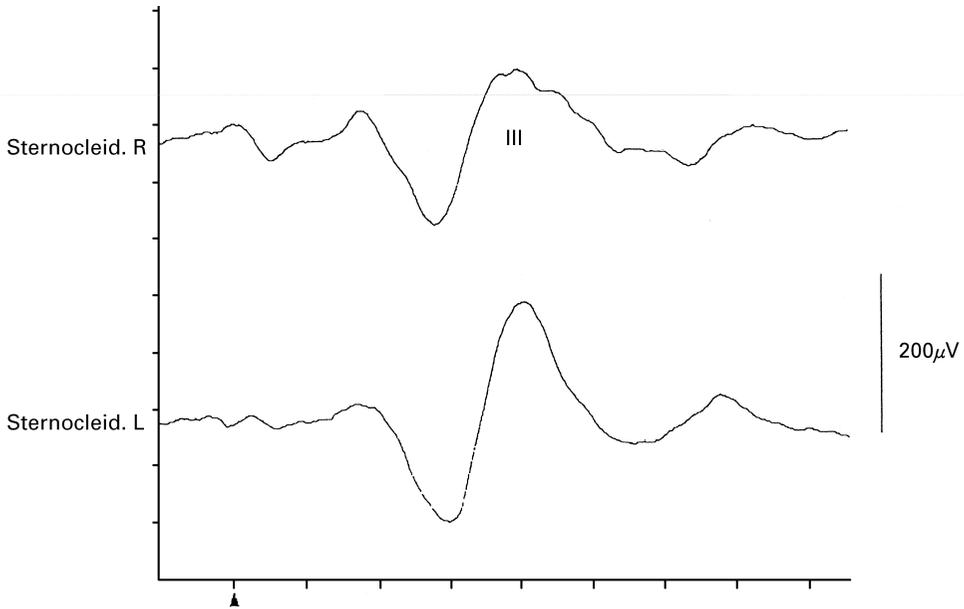


Fig. 4 EMG responses in the bilateral sternocleidomastoid muscles elicited by head taps on the forehead in a patient with bilateral vestibular neurectomy due to von Recklinghausen's disease. Arrow shows the time of the trigger. Time scale division equals 5 ms. Short-latency EMG potentials I & II in the sternocleidomastoid muscles were abolished bilaterally, but an additional wave III was found on both sides.

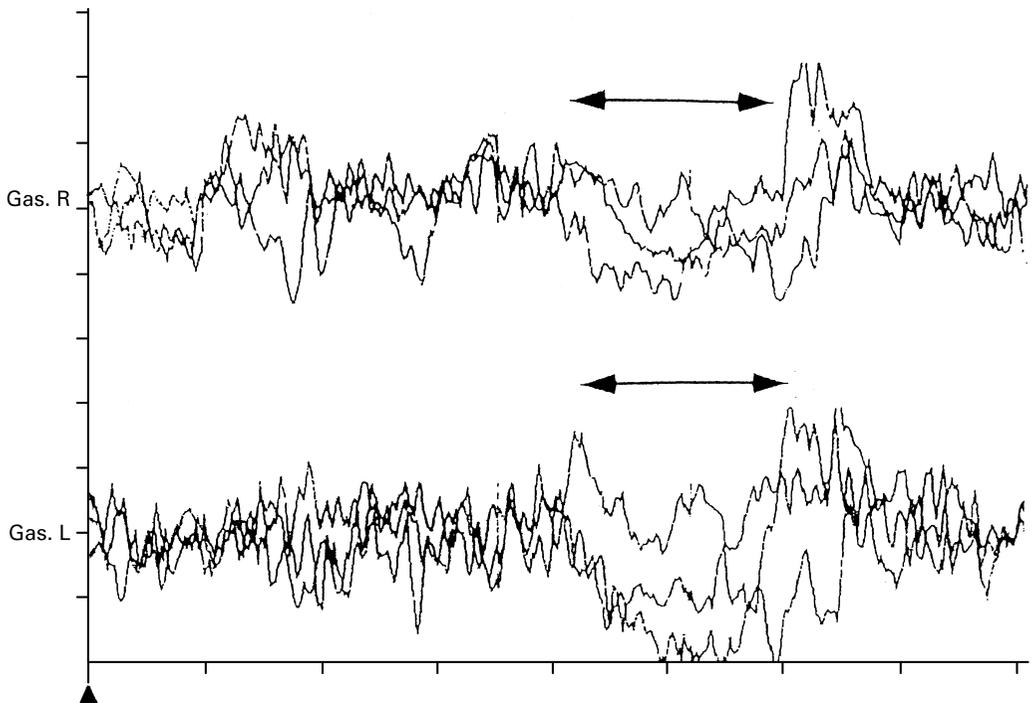


Fig. 5 EMG responses of the right and the left gastrocnemius (Gas. R, Gas. L) muscles elicited by forehead taps in a patient with bilateral vestibulopathy (idiopathic). Top and bottom traces show EMG potentials in the right gastrocnemius and left muscle respectively. Arrow shows the time of the trigger. Each time scale division equals 20 ms. Note that activity of the right gastrocnemius muscle is inhibited with a latency of 84.8 (onset) ms to 118.6 ms (end), and activity of the left gastrocnemius muscle is inhibited with a latency of 83.2 (onset) ms to 120.5 ms (end).

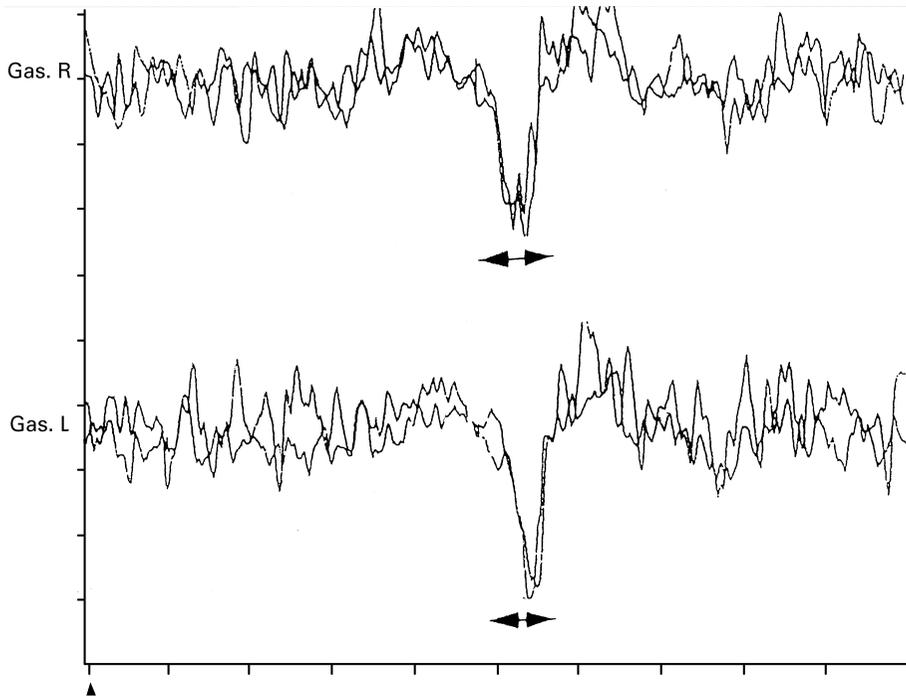


Fig. 6 EMG responses of the right and the left gastrocnemius (Gas. R, Gas. L) muscles elicited by forehead taps in a patient with von Recklinghausen's disease who underwent bilateral vestibular neurectomy. Top and bottom traces show EMG potentials in the right and left gastrocnemius muscles respectively. Arrow shows the time of trigger. Each time scale division equals 20 ms. Note that activity of the right gastrocnemius muscle is inhibited with a latency of 96.5 (onset) ms to 110.3 ms (end), and activity of the left gastrocnemius muscle is inhibited with a latency of 98.6 (onset) ms to 114.4 ms (end).

bilateral vestibular neurectomy (Figure 4). In the lower leg muscles, short-latency EMG potentials were delayed on the affected sides in the two patients with idiopathic vestibulopathy (Figure 5) and one patient with von Recklinghausen's disease who underwent bilateral vestibular neurectomy (Figure 6).

DISCUSSION

Myogenic potentials can be generated by click-evoked vestibulocollic reflexes [3, 4]. Actually, the first short-latency EMG potentials have disappeared or are attenuated in subjects with conductive hearing loss although they are present in subjects with profound sensorineural deafness [7]. The first short-latency EMG potentials are thought to be generated by vestibular afferents, which can be activated by vibration as well as by sound [11]. Halmagyi et al. [7] reported that head taps also evoked EMG potentials since the vestibulocollic reflexes in the sternocleidomastoid muscles respond to them. In normal subjects, click- or tap-

evoked myogenic reflexes from the sternocleidomastoid muscles comprise two distinct components. The first short-latency EMG potentials of a biphasic positive-negative wave with the mean latency of the initial positivity amounting to 9.8 ms (the mean latency of the initial negativity was 16.5 ms) are predominantly generated by afferents originating ipsilaterally at the vestibular division of the eighth nerve. The second and later component is not always present in normal subjects, but usually begins with a negativity that is generated by afferents originating from both ears. It probably arises from cochlear afferents and is thought to be related to the startle reflex. According to Halmagyi and coworkers [7], this first short-latency EMG potential evoked by tapping the head resembles the potentials generated by the click-evoked vestibulocollic reflex of the sternocleidomastoid muscles and probably proceeds along the same pathway from the vestibular apparatus to the muscles of the click-evoked myogenic reflex-

es. However, in contrast, they were not abolished or attenuated in ears with conductive hearing loss and were present in profound sensorineural deafness [7]. The tap-evoked myogenic reflexes, unlike the click-evoked myogenic reflexes, cannot activate the vestibular apparatus selectively. In our study, the short-latency EMG potentials of the bilateral sternocleidomastoid muscles elicited by forehead taps had a mean positive potential peak of about 12.6 ms and a mean negative potential peak of 17.3 ms, which were similar to those in Halmagyi's study [7]. Furthermore, the positive-negative potentials of the sternocleidomastoid muscles elicited by forehead taps in the patients were clearly affected; they were not present on the affected sides in two patients with idiopathic vestibulopathy and one patient with von Recklinghausen's disease who underwent bilateral vestibular neurectomy. It has not yet been clarified if the head-tap elicits reflexes from the otoliths to the lower leg muscles. Some studies have reported an initial muscle excitation of 60 to 80 ms in lower leg muscles after an unexpected fall or head displacements, and attributed it to otolith activation, possibly via connections to the reticular formation and then to the spinal cord [1, 6, 8, 10]. In our study, the conduction delay of the short-latency EMG potentials to the gastrocnemius muscles due to the head movement elicited by the tapping of the head amounted to about 50 ms. This matches the latency of vestibulospinal reflexes in patients with the Tullio phenomenon [1, 5]. In the present study, the short-latency EMG potentials in the lower leg muscles were delayed in all patients with vestibular lesions on the affected sides. This was thought to be generated by vestibular afferents, and converge with vestibular input for multisensory

control of posture.

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