Heterotopic Nociceptive EMG-Reactions in *M. masseter*

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Peculiarities of EMG-reactions of *m. masseter* to noxious homo- and heterotopic stimulation were studied on healthy volunteers. Homotopic noxious electrical stimulation of *n. mentalis* elicited several successive responses in *m. masseter* consisting of R-component, early exteroceptive suppression period, early excitation period, late exteroceptive suppression period, and late excitation period. Heterotopic noxious stimulation (forehead, ear lobe, index finger) induced only the late exteroceptive suppression period and late excitation period. It is concluded that the excitatory components have a reflex nature, and that the late exteroceptive suppression period is formed under the effect of central cerebral structures, which mediate their influence via the spinal-corticospinal return loop.

Key Words: *pain; exteroceptive suppression*

Perioral electrical stimulation induces two successive periods of suppression of tonic EMG activity in masticator muscles, denoted as ES1 and ES2 [7]. The latencies of the early (ES1) and late (ES2) periods of suppression are 10-15 and 22-55 msec, respectively [4,9]. The degree of exteroceptive suppression in masticator muscles increases during homotopic nociceptive activity in trigeminal afferents, which is used in clinical practices to quantitative assess pain in patients with headache and facial pain [1,5]. The detailed mechanisms of ES1 and ES2 genesis are unknown. It is considered that ES1 results from oligosynaptic activation of trigeminal nuclei interneurons by trigeminal afferents and subsequent suppression of masticator muscle motoneurons by these interneurons [4], while ES2 is mediated by a polysynaptic reflex arc comprising the neurons from the medullar part of the spinal trigeminal nucleus [12]. At the same time, there are data that electrical stimulation of fingers reduces ES2 in masticator muscles [8]. This suggests that the mechanisms of ES2 development are more complex and involve the supraspinal centers. To test this hypothesis, we studied exteroceptive suppression of masticator muscles during heterotopic nociceptive electrical stimulation.

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MATERIALS AND METHODS

Eleven healthy male volunteers (age 35-55 years) were studied in compliance to International Association of Study of Pain (IASP). All volunteers had no chronic facial pain, head- or toothache, or other diseases related to temporomandibular pathology. They were adapted to comfortable posture in a chair with a head- and elbow-rests and were asked to clench the teeth with force, at which the level of EMG oscillations varied in a range of 100-200 μV equal to 30% EMG potential developed during maximum clench of the teeth. This level of arbitrary tonic activity was visually self-controlled during entire stimulation period (5-15 min) without fatigue.

EMG was recorded via bipolar surface electrodes (6 mm diameter) set along the muscle filaments over the belly of *m. masseter* with interelectrode distance of 2 cm. EMG was amplified in an Tiesy-8 electrophysiological system within a frequency band of 2-10,000 Hz. The amplified signal was digitized and

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fed to a computer, which averaged it directly or by modulus (averaging of integrated EMG). The original software recorded evoked EMG with a repetition rate of 5000 Hz and averaged 40-100 records.

The reflex EMG responses of *m. masseter* were evoked by electrical stimulation of the skin surface in *n. mentalis* area, ipsilateral upper part of the forehead, index finger, and the ear lobe. The latency of suppression and enhancement of EMG tonic activity in *m. masseter* were determined according to the cross of the corresponding curve with the baseline indicating the mean level of tonic EMG.

Electrical stimulation was performed with single rectangular pulses (0.2 msec duration, 2-3 sec between pulses) applied via round surface electrodes with interelectrode distance of 1 cm. Stimulation strength was controlled according to the amplitude of applied current and the degree of pain sensation assessed with a 100 mm visual-analog scale (VAS).

The results were analyzed statistically using Student's *t* test at p=0.05.

RESULTS

When the strength of electrical stimulus applied to *n. mentalis* was threshold for tactile sensation, and it produced no effect on the current voluntary EMG activity of the examined muscles (Fig. 1, *1*). The increase in stimulating current to 1.5 tactile thresholds (6.2±1.4 mA) elicited clear ES1 and subsequent B1 periods both in ipsi- and contralateral *m. masseter* (Fig. 1, *2*). In addition to this well-known exteroceptive EMG responses, a short-latency (R) potential appeared on the ipsilateral side, which preceded ES1. In various subjects, the latency of this response varied between 6 and 8 msec (6.9±0.5 msec). Further increase in the strength of stimulating current to the pain threshold provoked a distinct pain sensation (50-60% VAS) and induced clear-cut ES2 and B2 periods in EMG (Fig. 1, *3*). The amplitude-temporal parameters of exteroceptive suppressive and excitatory EMG responses of *m. masseter* to noxious stimulation are summarized in Table 1.

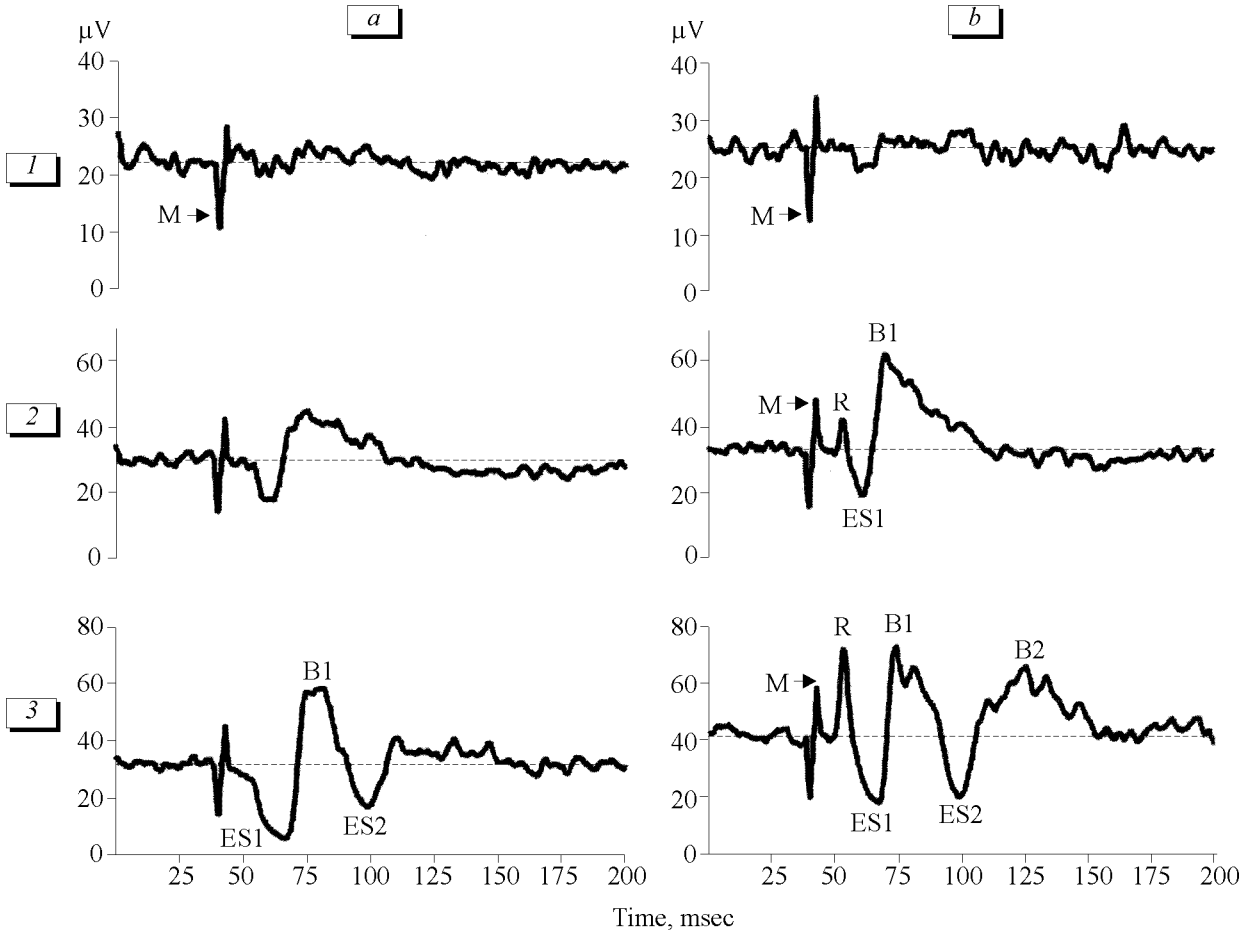

Fig. 1. EMG responses in contra- (*a*) and ipsilateral (*b*) *m. masseter* during stimulation of *n. mentalis* with electrical current corresponding to tactile threshold sensation (*1*), 1.5 tactile threshold (*2*) and 30% noxious level (visual analog scale, *3*). Dashed line is the baseline of averaged integrated tonic EMG activity, R is short-latency potential, and M is stimulation artifact.

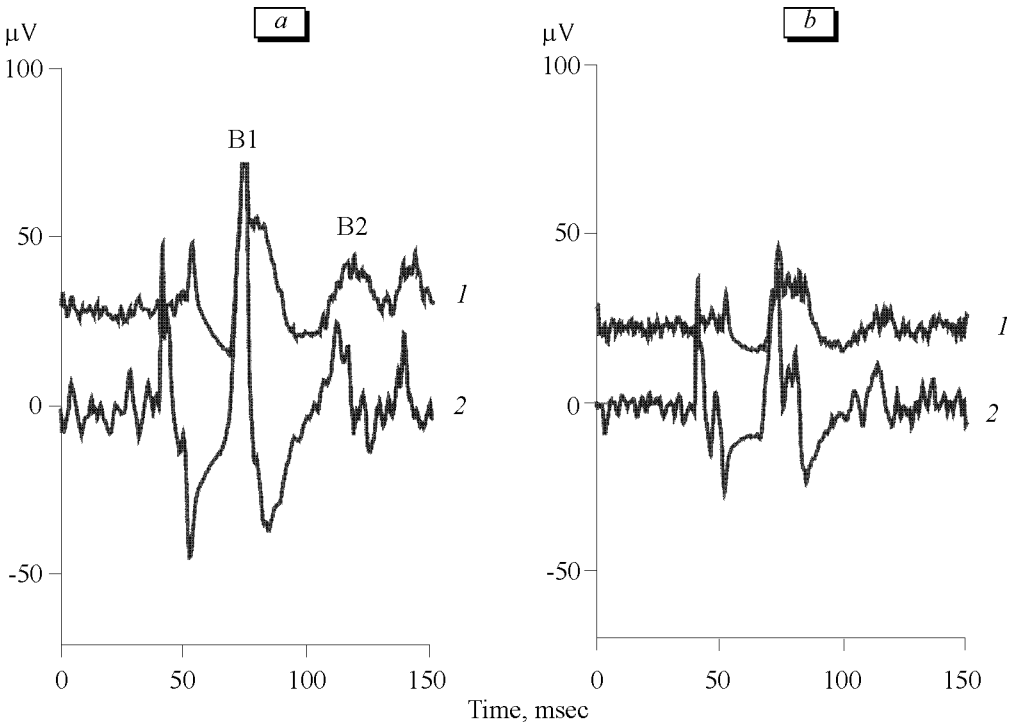

Fig. 2. EMG responses averaged by modulus (*1*) and direct (*2*) methods during painful electrical stimulation of right (*a*) and left (*b*) *n. mentalis*.

Direct averaging of EMG elicited in *m. masseter* in response to noxious stimulation of *n. mentalis* revealed periods, which were synchronized with B1 and B2 components of EMG revealed by modulus averaging (Fig. 2).

In contrast to homotopic stimulation, the heterotopic subnoxious stimuli produced no changes in EMG activity. Increase of stimulation to painful level elicited the inhibitory and excitatory EMG responses in the tonic EMG activity of *m. masseter* characteristic of ES2 and subsequent B2 periods (Fig. 3). The amplitudes of ES2 and B2 periods increased in parallel with the rise of stimulus intensity from the pain threshold to severe pain (70-80% VAS). The latency of ES2 period elicited by stimulation of forehead and ear did not significantly differ from ES2 latency in EMG elicited by stimulation of *n. mentalis*, while this parameter significantly surpassed the above values, when ES2 was evoked by index finger stimulation (62.4±4.6 msec, $p<0.01$). In addition, even strong heterotopic stimulation (70-80% VAS) elicited no R-, ES1, or B1 responses observed during perioral stimulation (Fig. 3).

The present data corroborate the current view that ES1 is caused by oligosynaptic activation of interneurons in trigeminal nuclei by low-threshold myelinated trigeminal afferents producing an inhibitory effect on motoneurons of masticator muscles [4]. It is attested by short latency, low threshold of ES1 elicited in response to stimulation of *n. mentalis*, and the absence of this component during heterotopic stimulation. The short-latency ipsilateral R-response of *m. masseter* to stimulation of *n. mentalis* is probably analogous to R1-component of the opticofacial reflex of eye orbicular muscle elicited by stimulation of supraorbital nerve [10] or to so-called short-latency nonmonosynaptic responses in the limb muscles [6].

Appearance of ES2 during noxious heterotopic stimulation indicates participation of the central cerebral structures in the formation of this response, which

TABLE 1. Amplitude-Temporal Parameters of the Periods of Exteroceptive Suppression and Enhanced Excitation in Tonic EMG Activity of *m. masseter* during Painful (50% VAS) Electrical Stimulation of *n. mentalis* in Healthy Volunteers (*M*±*m*)

Component of EMG response	Latency, msec	Position of EMG maximum, msec	Maximum amplitude, μV	Duration, msec	Area, μV²
ES1	13.5±0.5	21.0±0.8	-31.1±7.8	23.8±1.7	468.5±119.2
B1	30.3±1.3	36.0±1.2	22.7±10.1	12.3±0.5	155.4±67.4
ES2	40.4±1.8	52.4±3.9	-39.1±4.7	30.6±4.9	704.4±197.1
B2	72.2±4.4	91.6±6.2	35.8±7.3	79.4±15.6	1242±238

contradicts the view on the brain stem polysynaptic nature of ES2 [11]. The detailed analysis of genesis of the excitatory periods is necessary for better understanding of the nature of exteroceptive suppression of *m. masseter*. According to published data [9,12], enhancement of EMG activity in *m. masseter* between two inhibitory periods is not a reflex response, but a continuous recurrent "rebounding" activity determined by the inhibitory periods. Our data suggest that B1 and B2 periods appearing in voluntary tonic activity of *m. masseter* after ES1 and ES2 periods result from reflex excitatory influences on the motoneurons innervating masticator muscles. This conclusion is based upon possibility to reveal the early and late responses in the tonic EMG activity by direct averaging. These responses reflect contractile activity of *m. masseter* with the latency corresponding to the latency of B1 and B2 responses revealed by modulus averaging technique. In addition, peculiarities of appearance of B1 and B2 components in EMG of *m. masseter* are comparable to defensive motor reflexes characteristic of leg flexor [13] and arm [2] muscles denoted as RII- and RIII-reflexes. Similar to B1 component, the RII-response is elicited by homotopic stimulation, while RIII-response and B2 component are also evoked by heterotopic stimulation [3]. These date show that the late ES2 and B2 periods of *m. masseter* EMG have a suprasegmentary level of closing common to all nociceptive motor reactions, which is probably mediated via the spinal-corticospinal return loop.

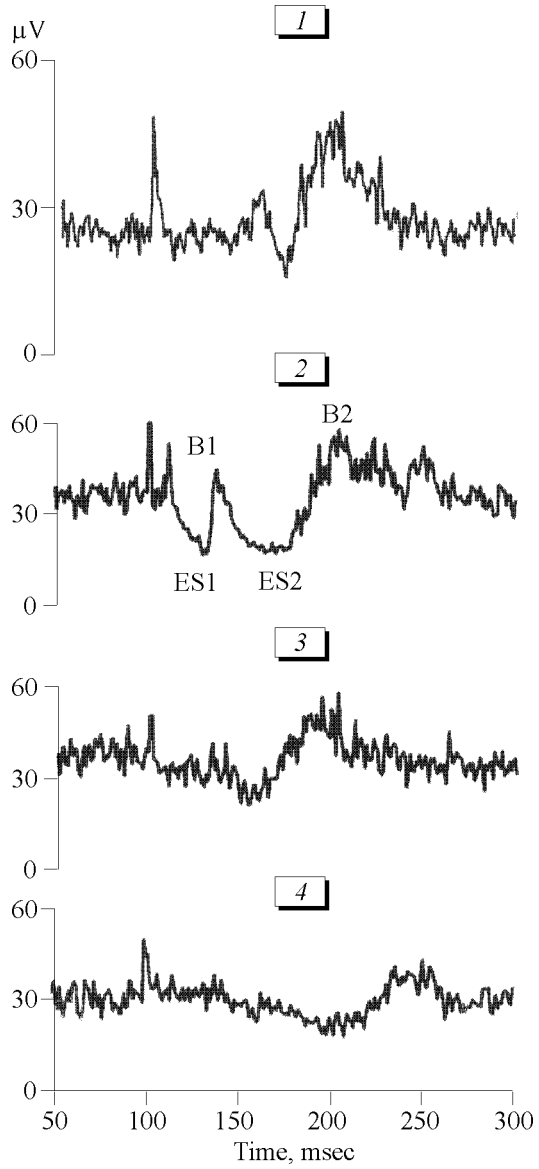

Fig. 3. Modulus-averaged EMG responses of ipsilateral *m. masseter* evoked by electrical stimulation of the ear lobe (*1*), *n. mentalis* (*2*), forehead (*3*), and the index finger (*4*).

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