

Changes in Parameters of Laser-Induced Potentials After Transcutaneous Electroneurostimulation

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 150, No. 10, pp. 454-456, October, 2010
Original article submitted July 21, 2009

Changes in nerve conduction after 10-min electroneurostimulation of the posterior surface of the neck were studied. Changes in the parameters of laser-induced potentials obtained during stimulation of C7 dermatome on hands and posterior surface of the neck were found. Decrease in the amplitude and shortening of the component latency were shown. Method of laser-induced potentials was concluded to provide unbiased estimation of the level and peculiarities of analgesic effects of physical factors.

Key Words: *transcutaneous electroneurostimulation; laser-induced potentials*

Transcutaneous electroneurostimulation (TENS) is a noninvasive method for symptomatic reduction of acute and chronic pain [2]. Effects of TENS at segmental level are mediated by selective activation of A δ -afferent nerve fibers and inhibition of pain sensory input from A δ - and C-fibers through spinal cord interneurons [5]. TENS is used at submaximal level (maximal level before pain) with a frequency from 1 to 200 Hz and the duration of biphasic rectangular pulse from 50 to 500 μ sec [3].

Despite numerous investigations of TENS physiology and wide use in clinical practice, extrasegmental mechanisms of its action are insufficiently studied. In order to objectify the analgesic effects, we used modern method of examination of pain-conducting pathways, laser-induced potentials (LIP). LIP method lies in stimulation of evoked brain potentials by infrared laser light, which stimulates thermo- and nociceptive nerve endings. Bioelectrical brain potentials were recorded using EEG. In addition, EEG-activity reflected changes in functional activity of the cortex areas responsible for reception and processing of input nociceptive information transmitted by A δ - and C-fibers.

MATERIALS AND METHODS

Experiment involved 10 males (age 26.14 ± 0.54 years, body weight 62.33 ± 4.90 kg, height 172.16 ± 2.35 cm). Subjects were mentally sane and had no dysesthesia or neurological diseases. Experimental protocol was approved by Ethic Committee of Institute of Physical Culture.

Nociceptive stimulation was performed using CO₂-laser pulses (device for thermo-optical diagnostics and correction, Institute of Physical Culture and Sport). The chosen laser pulse intensity 1.2-1.5-fold exceeded subject's pain threshold (intensity 250-350 mJ/mm², pulse duration 20 msec, diameter 5 mm). Stimulation was performed at the C7 dermatome area on hands and posterior surface of the neck.

To perform LIP, 20 laser pulses were applied to each area. In order to avoid changes in LIP characteristics due to nociceptor adaptation, 10-sec pause was made after each stimulation and the location of stimulation was slightly changed (within 2 cm from the initial stimulation site). Evoked potentials were simultaneously recorded using EEG. Investigator and subject used protective glasses to protect eyes from the laser.

EEG was recorded using Neocortex EEG recording device (Neurobotics) via 5 electrodes (Fz, Cz, Pz,

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TABLE 1. Mean Values of N2 and P2 Components before and after TENS ($M \pm m$)

Experimental conditions	N	Stimulation area	N2 \pm SD (latency, msec)	P2 (latency, msec)	Amplitude, μ V
Before TENS	9	Hand	188 \pm 10	288 \pm 18	12 \pm 5
	9	Posterior surface of the neck	133 \pm 19	206 \pm 10	14 \pm 7
After TENS	9	Hand	176 \pm 10	266 \pm 16	10 \pm 5
	9	Posterior surface of the neck	119 \pm 8	179 \pm 19	8 \pm 4*

Note. N2: late negative component of evoked potential, P2: late positive component of evoked potential. * $p < 0.05$ compared to value obtained before TENS.

T3, T4) placed in accordance to international electrode placement system 10/20. Averaging of two ear electrodes (A1+A2)/2 served as the reference electrode. EEG registration complied the following requirements: impedance lower 10 k Ω , band pass 0.2-200 Hz, sample rate 1000 Hz. Registration was performed in a room isolated from noise and bright light in the sitting position with open eyes. EEG analysis was performed within 1000 msec epochs, with 100 msec preceding the stimulus. Oculomotor and electromyographic artifacts were excluded before epoch averaging. LIP amplitude and latency were measured using data obtained from Cz-electrode. Amplitude of the potentials was measured from N2 to P2.

After LIP registration, each subject was examined for submaximal current on non-affected skin area using device TENS-01-Skenar (Ritm). The subject was asked to sit and his cervical spine was paravertebrally exposed to the influence at the area of VI-VII cervical vertebra. Subjectively-dosed regimen was employed during the procedure; we used labile method of selected area treatment, when after electrode application to the skin it was repeatedly moved downwards within the treated area (frequency 90 Hz, Fm mode), procedure duration 10 min).

After TENS, LIP stimulation and registration procedure was repeated. The data was analyzed using sign test. Differences were considered to be significant when $p < 0.05$.

RESULTS

LIPs were registered from 9 of 10 subjects. In one subject, LIPs were not expressed, and he was excluded from the analysis. Mean intergroup N2 and P2 latencies are represented (Table 1).

Registered LIP latencies and amplitudes corresponded to the LIP parameters previously obtained in the group of healthy subjects [1].

We observed shortening of the latencies of LIP components (insignificant differences: hand $z = 0.89$, $p = 0.37$, posterior surface of the neck $z = 0.89$, $p = 0.37$) and a decrease in LIP amplitude (insignificant differences: hand $z = 1.5$, $p = 0.13$, posterior surface of the neck $z = 1.8$, $p = 0.04$). The decrease in LIP amplitude (~20%) immediately after TENS procedure demonstrated the effects of this manipulation at the extrasegmental level. Despite the fact that A β -afferent fibers serve as a substrate during exposure to TENS, the decrease in LIP amplitude is most likely determined by gate control of pain impulsion from A δ - and C-fibers at the level of associative interneurons of spinal cord substantia gelatinosa. The mechanism for LIP latency shortening is unclear. This phenomenon is probably associated with electrophysiological properties of nerve conductors. Electrostimulation is known to increase conduction velocity and decrease electroexcitability threshold [3]. LIP approach provides the possibility of unbiased assessment of the level and peculiarities of the effects of various factors, both pharmacological [4] and physical, which was demonstrated in our study.

The study was supported by Ministry of Education and Science of Russian Federation (government contract 02.522.11.2015) within the framework of State goal-directed program "Investigation and Development on Priority Orientations of Scientific-Technological Complex in Russia 2007-2012 years".

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