Somatosensory Evoked Potentials in Assessment of Pathological Autorhythmicity in Patients with Chronic Myofascial Pain

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Abstract: *Objective:* Objectivation of functional shifts in the nervous system through somatosensory evoked potentials at chronic nociceptive afferentation induced by the pathological neuronal autorhythmicity.

Methods: Pre- and post-treatment assessment of evoked potential component parameters in patients with muscular pain and myofacsial trigger points with different pain severity. The active pre-treatment autorhythmicity was treated as the amplitude increase and any component latency reduction. The autorhythmicity regression was treated as the amplitude reduction and the latency increase.

Results: Pain relief correlated with myofacsial trigger point transformation into the latent ones. As compared with the milder pain, the pronounced pain syndrome revealed more significant functional shifts (generators of autorhythmicity), prevailing at higher levels of the nervous system. However, in the milder pain, a number of low activity generators were detected that indicates an antinociceptive system under activity. After the treatment, the number of pathologically enhanced excitation generators decreased significantly with migration to caudal parts of the nerve system.

Such shifts may serve as a neurophysiological basis for further myofascial pain exacerbations.

Keywords: Somatosensory evoked potentials, autorhythmicity, generators of pathologically enhanced excitability, myofascial pain.

1. INTRODUCTION

Chronization of pain syndromes is a topical problem of neurology. Despite numerous concepts in explanation of reasons and mechanisms of emergence of myofascial pain syndromes (MPS) of various location, neurophysiological aspects of either local or generalized muscle pain are still studied insufficiently [1, 2, 15].

This causes topicality of issues of instrumental objectification of functional shifts in the nervous system with the help of clinical electrophysiology methods in terms of chronic nociceptive afferentation from myofascial trigger points (MTP). Research of indicators of the evoked activity is an adequate test which is at the same time methodically accessible in clinical terms and characterizes an integrative response of the nervous system to nociceptive afferentation. Realization of the sensory function is known to be multi-level and includes segmental and supra segmental mechanisms [5, 22].

We have studied parameters of components of somatosensory evoked potentials (SSEP) of near and far field in patients with myofascial pain before and after treatment. We have paid special attention to changes of latencies of SSEP components, since reduction of latencies of the evoked potential (EP) is considered to be the sign of sensitization of neuronic chains with formation of positive responses which transform the physiological functional algic system into the pathological one. Pathophysiological basis of such transformation is pathological autorhythmicity or generators of pathologically enhanced excitability (GPEE) according to Kryzhanovsky G.N. [16, 17]. Also they are known as pacemarkers or the process of ”kindling” [6, 9, 19, 20].

The source of pathological autorhythmicity is the aggregate of hyperactive neurons producing excessive, uncontrolled flow of impulses due to insufficiency of locking mechanisms in the population of its neurons as a result of effect of pathogenic agents. Disinhibition of neurons and primary firing in the form of steady depolarization of neurons take place, causing secondary insufficiency of locking mechanisms [3]. GPEE is formed in the affected nervous system from the primary and secondary changed neurons and is of polietiologic nature. It is distinguished by the ability to develop self-sustained activity which promotes formation of the pathological system in the central nervous system (CNS), underlying the relevant neuropathological syndrome. GPEE hyperactivates the CNS division where it emerged and as a result this division becomes the pathological determinant [17]. In case of myofascial pain such generators are represented by neuromotor system of MTP on various levels of the nervous system.

GPEE is characterized by increase of capacity, reduction of latent period as evidence of synchronization

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of neuron firing and fast integration of the neuron pool into common reaction as indicators of electrogenesis.

Besides, under the influence of GPEE functional state of neurons of the upper levels change increasing their excitability and hyperactivity, including that with formation of secondary foci (generators) of autorhythmicity in them. As a result of this a new pathological integration - pathological algic system (PAS) is formed from the primary and secondary changed formations of the algesthesis system. Therefore PAS shows itself in extension of the area of maximum manifestation of SSEP components. Herewith various level of extension of latency of SSEP components can also take place depending on how fast GPEE develops its activity with the relevant activation of PAS [16].

In case of myofascial pain such generators are represented by neuromotor system of MTP on various levels of the nervous system. Increase of EP latencies and extension of response basis are considered to be extensive reduced activation of neuronic GPEE. Selective activation of GPEE due to initial weakness of the locking processes limiting extension of influence of generators, during electric stimulation of peripheral nerves is an adequate approach to studying peculiarities of perception of sensory flow from the focus of the chronic disease.

As it was mentioned, we paid special attention to dynamics of indicators of latency of SSEP components, since amplitude is more variable and ambivalent feature of SSEP components when compared to latency.

As about the SSEP method applied in given research it has distinct basis.

At present the three traditional modalities of evoked potentials exist: somatosensory, acoustic and visual. However this conventional neurophysiological techniques do not assess the function of nociceptive pathways and are inadequate to detect abnormalities in patients with small-fiber damage, in particular. Now in this aspect such methods as laser evoked potentials (LEP), contact heat evoked potentials (CHEP), pinprick-evoked potentials (PEP) with different modifications of their application are available. All they aimed to evaluate the function of different types of fibres (Aß, Aδ- and C-fibers).

LEPs. The cerebral responses to LEPs are correlated with the activation of the nociceptors Aδ (myelinic fibres with rapid conduction) and C (non-myelinic fibres with slow conduction).

In view of the reduced activation time of the nociceptors using the laser (the pulse varies between 1ms and 20ms), it is possible to simultaneously activate the rapid conduction Aδ fibres (that constitute the primary pain pathway) and the slow conduction C fibres (the secondary pain pathway). In this way the two distinct LEPs are recorded and then assessed, since the temporal sum contribution is either absent or greatly reduced.

LEPs are characterised by a wide signal amplitude and low noise level (N-2P2), two-phase signal.

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As a rule, heat evoked potentials appeared for 100 ms later than corresponding potentials after electric stimulation of nerve. It is believed that similarity in intepeak latencies and scalp topography indicates similar cerebral processing of electric and heat signals (Treede R.D., Kief S., Holzer T. *et al*., 1988).

The study of LEPs is extremely useful in the analysis of painful syndromes of different origin, including those caused by peripheral neuropathies, myelopathies, cerebral infarction, post-herpetic neuralgia, trigeminal nerve neuralgia, temporo-mandibular dysfunction, fibromyalgia and headaches [13].

CHEPS. They appear to produce similar cerebral evoked nociceptor potentials to laser stimulation in patients (Aδ- and C-fibers). In comparison with lasers, the CHEPS system is easy to operate and calibrate, and it allows for repetitive stimulation or "wind-up", avoiding any risk of superficial burns. However, CHEPS does involve contact with the skin, which may be uncomfortable in some patients with allodynia, and, in principle, the skin contact may affect pathways not activated by heat or noxious stimuli. A discussion comparing thermal conduction (CHEPS) and thermal radiation (LEPS) in the study of the nociceptive system has been conducted. CHEPS may be used for the

assessment of small fibre neuropathy, correlations with skin flare responses [7].

Both CHEPs and pinprick-evoked potentials (PEP) may also be convenient tools to assess sensitization of the nociceptive system. In future studies, small-fiber evoked potentials may also be used in studies that aim to understand pain mechanisms including different neuropathic pain phenotypes, such as cold- or touch-evoked allodynia, and to identify predictors of response to pharmacological pain treatment [4].

Compared to other stimulation techniques of evoked potentials, such as CHEPs (Contact Heat Evoked Potentials), the advantage of laser is that it can be used to stimulate any given body area with variable spot sizes (CHEP probes require a flat surface to be correctly positioned). The main disadvantage of the CHEPs compared to the LEPs is that in many cases the former fail to induce signals relating to the activation of the C fibres. Consequently CHEPs may provide incorrect diagnoses, suggesting that the functioning of the C fibres is absent, whereas this can be detected when LEPs are used [13].

As is well-known, under pain phenomena, which are not accompanied with damage of afferent systems, SSEP are specifically characterized by increase in amplitudes and decrease in latencies. When somatosensory afferent transmitters are involved, on the contrary, there is increase in latencies and decrease in amplitudes (Holmgren H. *et al.,* 1990).

On another hand when painful LEPs applied the two-phase negative-positive potential in vertex is registered. It`s amplitude usually correlates positively with the intensity of pain sensations that conditioned by afferent A-delta and C-fibres associated with the basic afferent nociceptive afferents (Carmon *et al*., 1976).

The differences were shown between SSEP and LEPs. In particular the approximately similar conduction velocity (10 to 15 m/second) of peripheral nerves for both pain SSEP (Electrical) and pain SSEP (LEP) in a range of A-delta fibers. The generator sources are considered to be the secondary somatosensory cortex and insula, and the limbic system, including the cingulate cortex, amygdala, or hippocampus of the bilateral hemispheres. The latencies and amplitudes are clearly affected by various kinds of stimulation applied simultaneously with pain. Abnormalities of pain SSEP (L) reflect an impairment of pain-temperature sensation, probably relating to dysfunction of A-delta fibers of the peripheral nerve and spinothalamic tract. In contrast, conventional SSEP after nonpainful electrical stimulation reflects an impairment of tactile, vibratory, and deep sensation, probably relating to dysfunction of A-alpha or A-beta fibers of the peripheral nerve and dorsal column.

The authors confirm that combining the study of pain SSEP (L) and conventional SSEP is useful to detect physiologic abnormalities, and some times subclinical abnormalities, of patients with peripheral and central nervous system lesions [14].

In this aspect some types of fibre can be activated by electric stimulation, and contribution of psycho-emotional component can be evaluated implicitly as well.

There were investigated effects of sleep on pain-related somatosensory evoked potentials (SEP) following painful electrical stimulation of the left index finger. The biggest advantage of this method is that signals ascending through both A-beta fibers relating to touch and A-delta fibers relating to pain can be recorded simultaneously. While the subject was awake, non-painful stimulation evoked early- and middle latency components, N20, P30 and N60, at the C4 electrode, and painful stimulation evoked not only early- and middle latency components at the C4 but also later pain-specific components, N130 and P240, at the Cz electrode. During sleep, N20 and P30 did not show a significant change in amplitude, N60 showed a slight but significant amplitude reduction, and N130 and P240 significantly decreased in amplitude or disappeared, as compared with those while awake. So, there are speculations on the mechanisms generating each component as follows; (1) N20 and P30 are the primary components generated in SI ascending through A-beta fibers. (2) N60 is the secondary component generated in SI involving cognitive function to some degree. (3) N130-P240 are the pain-specific components ascending through A-delta fibers, and closely related to cognitive function, because they were much affected by consciousness, different from the components ascending through A-beta fibers [23].

Also it was shown that cortical evoked potentials (CEP) to mechanical and electrical stimulation of the esophagus are mediated by similar afferent pathways, most likely a combination of both vagal and spinal afferents, and that the early components of the CEP to both stimulation modalities are mediated *via* Aδ-fibers. The combined use of both may have an important role in the assessment of esophageal sensory processing in disease states [12].

Besides it was revealed that SEP amplitude and subjective pain intensity estimated by visual-analog scale (VAS) following electrical tooth stimulation can be decreased by noxious stimuli to hand. This finding show that heterotopic painful stimulation attenuates experimentally-induced tooth pain suggests a triggering of diffuse noxious inhibitory control (DNIC) with after-effect in trigeminal region [18]. Specifically, predominant damage of dense myelinated fibres eliminates blocking of nociceptive impulsation. Cognitive and emotional and affective components of central mechanisms of pain perception are included (Melzack R., 1981).

As about location of the human nociceptive area(s) there are multiple lines of evidence from electrophysiological and imaging studies in humans indicate that parasylvian cortex is prominently involved in the processing of nociceptive stimuli. Our data suggest that the inner vertical surface of the operculum is activated most rapidly, but evidence from other studies indicates that the nociceptive area in this region extends into the adjacent dorsal insula. The functions of this operculoinsular region are still a matter of debate and may involve sensory integration of tactile, nociceptive, and visual input, as well as spatially directed attention (Treede *et al*. 2000). The insula is also considered a visceral sensory and visceral motor area (Augustine 1996) and may thus serve a sensory integrative function for pain, taste, and other visceral sensations as well as tactile and vestibular input. Because of its projection to the amygdala, the insula may also be involved in affective and emotional processes and in sensory memory-particularly memory of previously experienced pain (Lenz *et al*. 1995). Detailed analysis of the interconnections of this nociceptive cortical network and particularly of its functional significance for human pain sensation remains a challenging task for the future [10].

Taking into account that many levels and systems of nervous system participate in pain formation, largely the method of somatosensory EP is employed to study the state and role of different brain mechanisms in organization of somatosensory, as well as painful, afferentation (Bromm B. *et al.,* 1998).

The advantage is multicomponent approach which allows to document SSEP components at different levels of nervous system.

Taking into account that components of painful and non-painful SEP better describe topographically neuroanatomical and neurofunctional structure of CNS, as well as wide range of sensitivity types, there was chosen the electric stimulation slightly exceeding pain threshold according to VAS results.

Despite the specificity of LEP to noxious pathways the pain-related SSEP, as was mentioned above, are involving both A-beta fibers relating to touch and A-delta fibers relating to pain.

Also the advantage of SSEP is a presence of several numbers of components which have consecutive time origin at different levels of nerve system. Therefore it is possible to estimate from caudal to oral level the autorhythmical, generator-like changes of SSEP parameters and to have imagination about relationship between regions of enhanced excitability and their migration during the process of treatment.

2. MATERIAL AND METHODS

There were 130 patients with myofascial pain (50 men and 63 women) examined in the age from 20 to 55 years (42.8±0.8 years in the average) with duration of the disease up to 20 years (4.6±2.7 years in the average). The control group was comprised of 100 healthy test persons in the age from 25 to 50 years. The Double blind rating was used with the following classification of patients in groups in accordance with clinical investigation, complaints and history of cases, SSEP data.

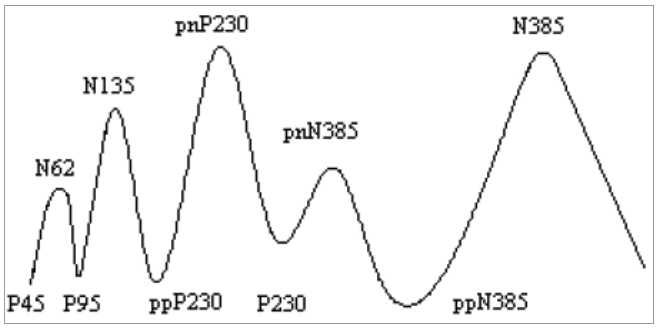
Patients were selected on the grounds of data of complex examination. MTP located in the area of scapular waist were studied. The three groups of patients were singled out according to the degree of pain syndrome intensity — with light (45 people), medium (40) and intensive (28) degree of painful symptoms based on the data of kinesthetic muscle palpation [21]. The three degrees of painfulness were defined: extensive — 3 points, moderate — 2 points, light — 1 point. Total painfulness of the studied MTP before and after treatment is presented in the schedule. MTP was considered active: 1) in case of referred spontaneous pain or evoked pain, 2) in case of local spastic response to cross palpation of MTP, 3) in case of disappearance or reduction of referred pain after postisometric or postreciprocal relaxation. In case of absence of referred pain and local spastic response MTP was considered latent though there retained induration and local tenderness when the muscle was stretched.

Brain Surveyor brain mapper of Italian-Japan company "Saico" was used for registration of SSEP. SSEP was recorded in the points of somatosensory

projections of electric stimulation of contralateral median nerve in the area of wrist on the side of MTP localization. Rectangular current impulses with duration of 50 ms were applied in a casual manner with intervals between stimuli from 2 to 2.5 s. More frequent stimuli were applied for registration of EP of the far field. Intensity of stimulation was standardized according to slight movement of III finger of a hand (5—10 V beyond motional limit). The median nerve was selected in view of the fact that it`s scalp projection points in electrical stimulation are standardized and well known. Also the stimulation electrode fixation on wrist in this case is most easy-to-use.

The active pickup electrode was located 1cm behind the auricular vertical and in 6—7cm aside from the median sagittal line, reference electrodes (2 in total) were located on the lobes of ears. Monopolar leads were registered. The lower band pass was 5.3Hz, the upper one was 15 kHz. Epoch of analysis was 50 ms for the early SSEP components, 200 ms for the intermediate ones and 500 ms for late ones in accordance with division of SSEP into the groups of early (20—80 ms), intermediate (80—200 ms) and late (200—400 ms) components [24]. Every 130 responses were averaged.

Positive-negative and negative-positive diphase fluctuations preceding the main late component accepted in nomenclature—*P*230 and *N*385, were marked as *pp*P230 and *pn*N385. In these abbreviations the first letter *p* is on the analogy of the English word preperceptual, used by L. Garcia Larrea and co-authors. [8] for designation of vertex-potentials in registration of EP, connected by stimulus; the second letter *p* or *n* reflects respective positive or negative nature of fluctuations in relation to isoelectric line. L.R. Zenkov and co-authors point to presence of additional poly-phase fluctuations preceding the late components of SSEP, noting their appearance in case of extensive frequency band of the brain mapper (5 Hz—5 kHz) and their multi-functional nature (Picture **1**).



Picture 1: The example of somatosensory evoked potentials configuration after contralateral median nerve stimulation in healthy persons.

Statistical comparison of values of latencies and amplitudes of SSEP components was conducted among the groups of patients before and after treatment, and with the control group. Student's criterion for dependent and independent pairs of values, sign test, Wilcoxon criterion for related and unrelated totals were applied. Processing was made with the use of Statistica 6.0 software. When values were compared according to Student's criterion there were degrees of differences of latencies and amplitudes detected: $p<0.001$ — highly significant, $p<0.05$ — significant, $p<0.09$ — slightly significant differences. It was reasonable to estimate their dynamics with presentation of the relevant graphical curves not according to numerical values of the said characteristics but based on the values of statistical differences. It allowed to detect tendencies of EP change within one histogram (see picture **2**). Four degrees of differences were selected for this, where the first indicator corresponds to latency and the second one - to the amplitude:

0 — no differences, correspond to $p>0.09$;

–1 or +1 — slightly significant differences, correspond to $p<0.09$;

–2 or +2 — significant differences, correspond to $p<0.05$;

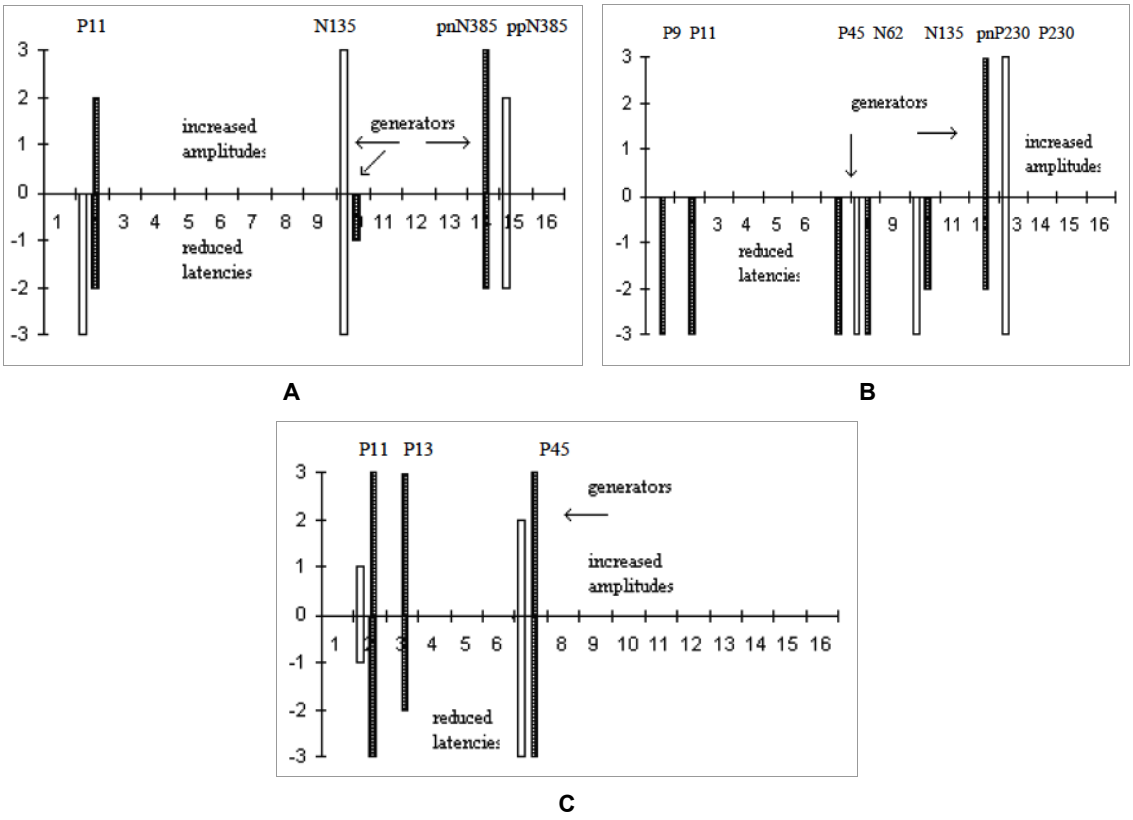
–3 or +3 — highly significant differences, correspond to $p<0.001$.

Positive values point to the tendency of increase of amplitude, negative values point to the tendency of reduction of latency before or after treatment.

3. RESULTS

The patients complained about tender, dull continuous pains, feeling of woodiness, contracture in the depth of various muscles of scapular waist. Most frequently trapezius muscles, inferior oblique muscle of the head, greater pectoral muscle, deltoid muscle and splenius muscle were affected.

Aggravation of myofascial pain corresponded to increase of MTP activity, in this case local induration, referred pain and local spasm of the muscle during its cross palpation were always registered. Management of the pain syndrome was correlated with transformation of the said MTP into latent ones. The side of pain corresponded to localization of active and latent MTP. The schedule **1** shows that the number of MTP and the relevant degree of painfulness was more



Picture 2: Dynamics of modification of the evoked brain activity in the process of pain syndrome management. **A:** intensive, **B:** moderate, **C:** slight pain syndrome. EP components of the near and far fields are presented along the X-line in the ascending order (time); Student's criterion is presented along the Y-line. Positive values in the columns correspond to the amplitude, negative ones — to the latency of EP components (in comparison with the norm). Light columns - before treatment, dark columns — after treatment.

Schedule 1: Average Quantity of MTP of Cervix Muscle, Scapular Waist and Relevant Total Painfulness before and after Treatment

Patients	Average Quantity of MTP		Total Painfulness of MTP, Points	
	Before Treatment	After Treatment	Before Treatment	After Treatment
With intensive pain	5	3	3	2
With moderate pain	6	4	3	1
With slight pain	4	3	2	1

before treatment than after it in case of intensive and moderate degree of clinical signs. In case of slight degree of clinical signs there were few differences in the degree of painfulness before and after treatment.

Dynamics of changes of latencies of EP component before and after treatment is represented in the Picture **2**. It was supposed that reduction of latency and increase of amplitude of the certain EP components, when compared to the norm corresponding to statistically highly significant differences (–3 for latency, +3 for amplitude), make it possible to characterize neuromotor system of the intensive MTP as an active GPEE in the stage of chronization of the pathological process. Statistical differences –2, –1 for latency and +2, +1 for amplitude of EP corresponded to the latent and secondary MTP. Combinations of these parameters allow to characterize MTP as a part of pathological algic system determining its dynamics and prognosis of the treatment effect.

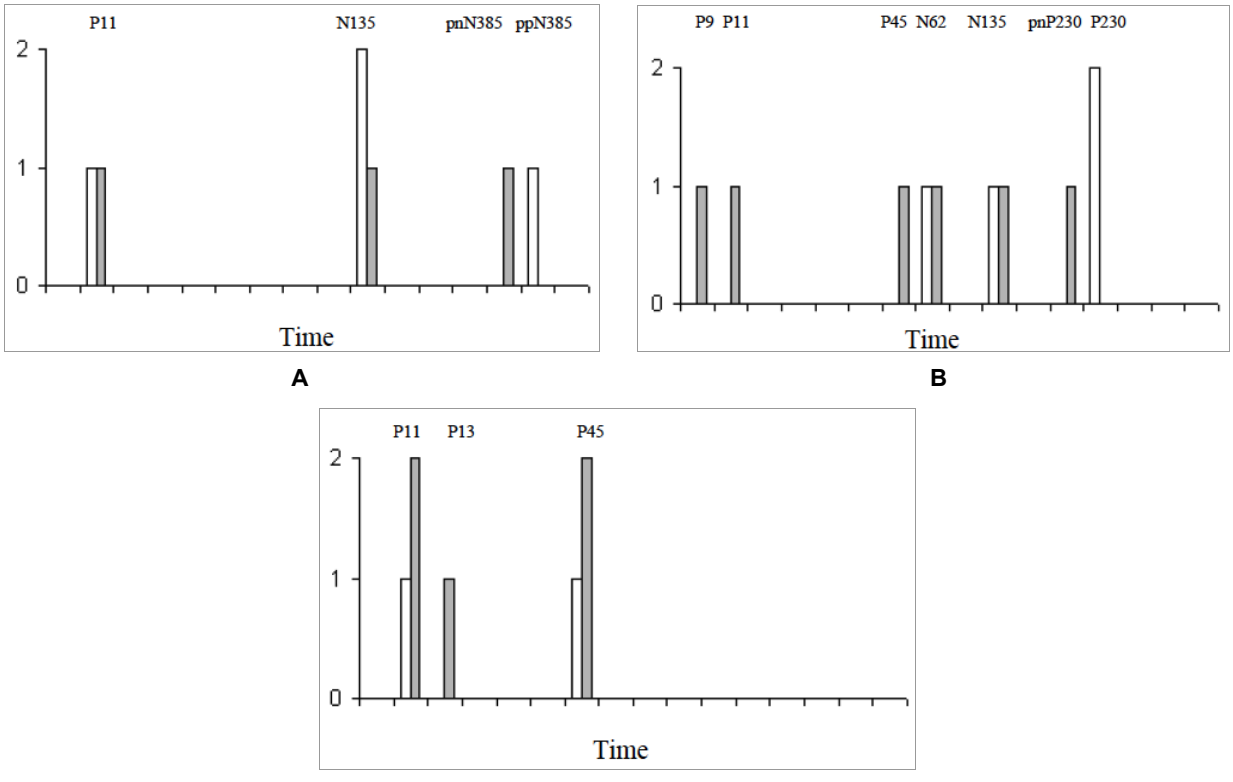
In general, each column shows GPEE of different degree of activity. The positive values point to increased amplitudes in comparison with healthy persons, whereas the negative values of the same column show decrease of latency in comparison with the same healthy volunteers (according to Student's criterion).

In other words, degree of hyperexcitability may be described as follows: the higher negative value, the more intensive hyperexcitability and the higher positive value of the same column, the more intensive hyperexcitability as well.

Picture **2** can be simplified by summarizing the negative values (decreased latency) and positive values (increased amplitudes) on Y-axis corresponding to one generator (component) and positive values 1 and 2 in one column. Value one corresponds to latent generator, whereas value two shows availability of active generator. Herewith, light columns - before treatment, dark columns – after treatment (see Picture **3**).

In case of intensive pain syndrome *N*35 component is mostly detected with highly significant reduction of latency and increase of amplitude within the limits of multi-synaptic heterospecific brain divisions, which can correspond to the active GPEE (–3; +3). Appearance of *P*11 component shows highly significant decrease of latency and *ppN*385 — significant decrease of latency, which can be the sign of the less active GPEE: *P*11 (–3; 0) and *ppN*385 (–2; +2) relatively in comparison with *N*35 (–3; +3) (see picture **2A,** Table **2A**). After treatment there is intensive increase of latency of *N*135 component observed up to the degree of slightly significant differences with normalization of amplitude, i.e. GPEE activity is decreased. Parameters of *ppN*385 component are normalized, however there are slight decrease of latency and highly significant increase of amplitude of the earlier *pnN*385 component detected, which corresponds to the latent generator *pnN*385 (–2; +3). Increase of the amplitude can be caused by inductive effect of the restored afferent sensory flow from the muscles. On the cervical level of the spinal cord *P*11 component has significantly increased latency (restoration of negative feedback on the level of *P*11) and amplitude, possibly due to the restored afferent impulsation from the muscles.

In case of moderate degree of clinical signs before treatment highly significant reduction of latency and increase of amplitude of *P*230 component are detected which can prove the presence of the active GPEE on the level of non-specific thalamic nuclei of *P*230 (–3; +3), and highly significant reduction of latency of *N*62, *N*135 components is detected due to incomplete realization of pathological shifts on the level of fast



Picture 3: Simplified and Summarizing Version of Picture **2**. On X-axis – sequence of SEP components. On Y-axis 1 corresponds to latent or subactive generator, and 2 – active generator, where latency and amplitude have highly significant differences in comparison with the norm (P<0.001). Light columns – before treatment, dark columns – after treatment. **A:** intensive, **B:** moderate, **C:** slight pain syndrome.

passing fibers of lemniscal system of *N*62 (–3; 0) and multi-synaptic heterospecific brain divisions of *N*135 (–3; 0). After treatment latency in the area corresponding to *N*62 component did not change (the same generator remained), on the level of *N*135 there was increase of latency up to the degree of significant differences, i.e. a generator with less intensive synchronization was detected — *N*135 (–2; 0) instead of *N*135 (–3; 0). Parameters of *P*230 were normalized and slightly and highly significant reductions of latency and increase of amplitude of the previous *pnP*230 component were observed. In other words, GPEE with decreased activity, but more caudal (see Picture **2B,** Table **2B**) appeared instead of active cortical and rostral one. Besides, highly significant reduction of latencies of *P*9, *P*11, *P*45 components was detected, i.e. there was a series of generators with incomplete realization revealed, which were obviously suppressed by the dominating focus of activity, — *P*9 (–3; 0), *P*11 (–3; 0), *P*45 (–3; 0).

In case of slight degree of clinical signs before treatment there was one component with highly significant decrease of latency and significant increase of amplitude detected during stimulation of the median nerve, which apparently corresponded to the more active generator *P*45 (–3; +2). *P*11 component had slightly significant reduction of latency and increase of amplitude which can correspond to the latent generator *P*11 (–1; +1). The generator *P*45 (–3; +2) was obviously secondary; the former primary *P*11 was reduced, possibly under the influence of locking mechanisms. This can evidence the tendency of movement of generator in rostral direction (see

Schedule 2: The relevant degrees of statistical differences of parameters of SSEP components corresponding to active and latent generators of autorhythmicity in comparison with the norm before and after treatment according to Student's criterion for independent pairs. A: intensive pain, B: moderate pain, C: slight pain syndrome (see Picture 2)

Table 2A: Intensive Pain

Before Treatment			After Treatment		
	Lat	ampl		Lat	ampl
P11	P<0.001	P>0.09	P11	P<0.05	P<0.05
N135	P<0.001	P<0.001	N135	P>0.09	P>0.09
ppN385	P<0.09	P>0.09	ppN385	P<0.05	P<0.001
ppN385	P<0.05	P<0.05	ppN385	P>0.09	P>0.09

Table 2B: Moderate Pain

Before Treatment			After Treatment		
	Lat	ampl		Lat	ampl
P9	P>0.09	P>0.09	P9	P<0.001	P>0.09
P11	P>0.09	P>0.09	P11	P<0.001	P>0.09
P45	P>0.09	P>0.09	P45	P<0.001	P>0.09
N62	P<0.001	P>0.09	N62	P<0.001	P>0.09
N135	P<0.001	P>0.09	N135	P<0.05	P>0.09
pnP230	P>0.09	P>0.09	pnP230	P<0.05	P<0.001
P230	P<0.001	P<0.001	P230	P>0.09	P>0.09

Table 2C: Slight Pain Syndrome

Before Treatment			After Treatment		
	Lat	ampl		Lat	ampl
P11	P<0.09	P<0.09	P11	P<0.001	P<0.001
P13	P<0.09	P<0.09	P13	P<0.05	P<0.001
P45	P<0.001	P<0.05	P45	P<0.001	P<0.001

Picture, **C**). After treatment there were highly significant reduction of latency and increase of amplitude noted, i.e. there was active generator *P*11 (–3; +3) observed instead of the latent generator in the area of *P*11 (–1; +1). Retention of the degree of reduction of latency of *P*45 component and highly significant increase of its amplitude were detected, which correspond to the active GPEE — *P*45 (–3; +3). Besides, there was significant reduction of latency and highly significant increase of amplitude of *P*13 component. This peculiarity can be associated with less intensive activity of antinociceptive system in case of slight degree of myofascial pain or restoration of the managed generator activity. In addition limitation of impact of MTP on changes of parameters of the later EP components was observed in comparison to the first and second groups of patients.

In case of intensive and moderate clinical signs the active generator moves in caudal direction after treatment, i.e. there is something like "shift back" of the core pathological algic system — to the area of the earlier primary GPEE.

4. DISCUSSION

Comparative prevalence of the quantity of subactive generators in the group of patient with moderate painfulness after treatment can be explained by the fact that in case of intensive painfulness the most sensitive generators - active GPEE are activated under the influence of electric impulse (see Picture **2A, 2B**) in contrast with moderate pain. That is why in case of severe pain small foci of GPEE are suppressed by activity of the main generator of the pathological algic system and are not detected. In case of moderate painfulness such GPEE, on the contrary, are reflected after treatment in disinhibition of activity (reduction of latency of the more number of EP component) of various relay structures, without any influence of the leading generator. This can be probably possible since GPEE, having own frequency and periodicity of discharges, are not subject to synchronization on the side of the active generator due to the limiting function of antinociceptive system. In the group of patients with slight degree of clinical signs, on the contrary, there were more active generator structures detected after treatment than those before treatment (decrease of latencies of the early and intermediate EP components - see Picture **2C**, Table **2C**). As it was mentioned this fact can be related to the less intensive activity of antinociceptive system in case of slight degree of myofascial pain or restoration of the managed generator activity. Therefore it can cause insufficient dominant (anta-gonistic) relations between the forming pathological algic system with incomplete realization and the antinociceptive system.

Considering unsteadiness of topology of GPEE and their modifications, dynamics of clinical signs of MTP as a result of treatment (transition of the active MTP into the latent ones) it is reasonable to suppose that there is a relation of the active GPEE with the total number of the active trigger points. Accordingly the latent GPEE reflect activity of the latent MTP. This can mean that after pain syndrome management and elimination of the active MTP there are latent generators reserved in the CNS which shift in caudal direction. Their activity does not disappear even in case of complete subjective recovery and elimination of the latent MTP. It has principal importance for understanding of mechanisms of myofascial pain backset under various initiating agents — in stress situations, in case of overcooling, intoxication etc. In other words, restoration of the pathological determinant system — in our case it is pathological algic system - occurs as per the mechanism of "the second strike" due to renewal of activity of its latent (including cortical) generators. According to G. N. Kryzhanovsky, "pathological determinant dies the last and revives the first", which is reflected in clinical signs of myofascial pain. On the other side, complex treatment of this syndrome, including not only local effects, is the basis of pathogenetically substantiated therapy practice, aimed at liquidation of the pathological algic system, formed under the influence of peripheral "harmless" local muscle induration.

CONCLUSIONS

There is correlation between the number of the active myofascial trigger points and the presence of the active generators of the pathologically enhanced excitement on various levels of CNS.

After treatment the generator of the pathologically enhanced excitement, being already latent, as a rule, moves in caudal direction — to the area of the former primary generator, which realized the developed myofascial pain.

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