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THE ROLE OF MYOFASCIAL SYNDROME IN THE GENESIS OF NOCTURNAL PAINFUL PARESTHESIAS

Abstract

The **objective** of our study was to investigate clinical and neurophysiological features of nocturnal painful paresthesias in the upper limbs.

Material and methods. The article presents the results of the study of 107 patients with pain and nocturnal paresthesias in their hands. It was revealed that the syndrome of nocturnal painful paresthesias is mixed in etiology and has myofascial pain syndrome as an initial part. The clinical symptoms of nocturnal pains and paresthesias in the hands of patients with myofascial pain syndrome of the shoulder girdle and upper limbs were described.

Results. It is shown that active myofascial trigger points are the key link in the clinical pattern formation of the syndrome of nocturnal painful paresthesias in patients with myofascial pain syndrome. In the study of short-latency somatosensory evoked potentials from the upper extremities, the pathological peak Px in the CVII-Fpz lead is described, which is the marker for the presence of a pathologically enhanced excitation generator in the suprasegmental structures.

Conclusions. The syndrome of nocturnal painful paresthesias is mixed in etiology and has, as an initial link, myofascial pain syndrome. Detection of a pathologically enhanced excitation generator in the suprasegmental sections of the sensitive pathway in the registration of short-latency somatosensory evoked potentials is an adequate method for diagnosing painful paresthesias.

Key words: *nocturnal painful paresthesias, myofascial pain, somatosensory evoked potentials*

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MPS — myofascial pain syndrome, VAS — visual analogue scale, MTP — myofascial trigger point, NCV — nerve conduction velocity, AP — action potential, SSEP — somatosensory evoked potentials

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Introduction

Hand paresthesia is a common symptom of somatic, neurological, and mental disorders. To manage this symptom, patients seek help from various healthcare specialists [1]. According to S. Marshall, C. Murray, 7.4 to 45% of adult population report pain and nocturnal paresthesias in their hands [2]. Medical literature assigns a major role in the development of nocturnal paresthesias to myofascial pain phenomena [3, 4, 5]. This is due to the fact that the most important signs of myofascial pain syndrome (MPS) are: pain, and psychovegetative, dissomnic and motor disorders [6, 7].

Dysfunction of afferent systems of the brain and spinal cord plays an important role in the pathogenesis of MPS. At the same time, an analysis of somatosensory evoked potentials (SSEP) allows us to clarify the proportions of central and peripheral mechanisms in the genesis of MPS. Numerous publications describe nocturnal painful paresthesias; however, there has yet to be a classification of their clinical and physiological mechanisms. The role of MPS in the pathogenesis of this disease has not been studied. The state of afferent systems of the brain and spinal cord in nocturnal painful paresthesia syndrome has not been evaluated.

The **goal** of our study was to investigate clinical and neurophysiological features of nocturnal painful paresthesias in the upper limbs.

The **objectives** of the study were:

1) To investigate clinical symptoms of nocturnal pains and paresthesias in the hands.
2) To investigate the role of myofascial syndrome in the development of nocturnal painful paresthesias in distal and proximal parts of the upper limbs.
3) To provide neurophysiological assessment of the functional status of peripheral and central regions of the somatosensory analyzer in patients with nocturnal painful paresthesias.

Materials and Methods

The study was based on the clinical and neurophysiological examinations of 107 patients reporting nocturnal painful paresthesias in their hands, aged 17 to 63. Duration of paresthesias (the reported time since the appearance of first clinical signs) varied from between several days to 12 years. Patients were selected based on a comprehensive examination of subjects seeking the assistance of a neurologist for nocturnal paresthesias in their hands.

The study had the following inclusion criteria:

- Crawling feelings in the upper limbs, mainly occurring at night.
- Active myofascial trigger points with marked tenderness of the shoulder girdle and upper limb muscles.

The study had the following exclusion criteria:

- Severe comorbidity (diabetes, alcohol abuse, kidney failure, endocrine, systemic and blood diseases).
- Organic lesions of central nervous system.
- Inflammation in the upper limb joints.
- Mental disorders and mental retardation.

The patients underwent clinical neurological examination of the spine and manual diagnosis [7, 8, 9, 10]. A visual analogue scale (VAS) was used to assess the severity of paresthesias and pains. Somatosensory evoked potentials were studied to assess the functional state of peripheral and central segments of the somatosensory analyzer. A multifunctional Neuro-MVP computer complex (NeuroSoft, Russia) was used in the study. To evaluate the integrity of peripheral nerves and to confirm neuropathy, we studied the action potential of a sensory nerve in response to electric stimulation.

Based on the results of clinical and neurophysiological examinations, all patients were divided into three groups: Group 1 (n = 40) — patients with impairment of the peripheral nervous system (tunnel mononeuropathies of the upper limbs); Group 2 (n = 37) — patients with impairment of an intervertebral disc accompanied by radiculopathy, cervicalgia, cervicobrachialgia, and clinical symptoms of fibromyalgia, MPS, active myofascial trigger points in the shoulder girdle and upper limb muscles; Group 3 (n = 20) — patients with pronounced clinical symptoms of fibromyalgia and MPS of the shoulder girdle and upper limb muscles.

Table 1. *Distribution of patients by gender*

Groups	Women		Men	
	Abs.	%	Abs.	%
1	32	80.0	8	20.0
2	32	86.5	5	13.5
3	20	100.0	0	0.0
4	5	50.0	5	50.0
Generally	89	83.3	18	16.8

Group 4 was a control group (n = 10) that included patients with peripheral nerve injuries who had permanent paresthesias in their hands. Patient distribution by gender is presented in Table 1.

The study group consisted mainly of women: 83.2% (men comprised 16.8%).

Twelve patients without upper limb disorders (healthy volunteers who did not report any crawling feelings [comparison group]) were examined in addition to the study patients (with disorders) to analyze and compare SSEP.

Student's t-test (for the comparison of quantitative variables between two groups), Newman–Keuls test (for the comparison of quantitative variables between three and more groups), and Student's t-test for proportions (for qualitative variables) were used to perform a statistical analysis of the clinical and neurophysiological findings. All calculations were made in the Biostatistica software.

Results

The analysis revealed that 99.0% of patients had reported nocturnal paresthesias in the upper limbs. All patients in Groups 1, 2, and the control group (100%) complained about nocturnal paresthesias. These patients comprised 97.3% of those in Group 3. Steady symptoms were more commonly reported (57.8%) than symptoms that increased (33.3%) or decreased (8.8%) in severity.

The localization of the paresthesia was an important factor. A total of 69.6% of patients reported hand paresthesias: Group 1 accounted for 62.5% of cases ($P_{I-III} < 0.05$), whereas Group 2 accounted for 64.9% ($P_{II-III} < 0.05$) and Group 3 accounted for 90.0%. Fingers (one or several) were affected in 51.0% of cases: Group 1 accounted for 52.5% of cases, whereas Group 2 accounted for 62.2% of cases and Group 3 accounted for 25.0% of cases. Therefore, when a peripheral nerve was damaged, the distribution of paresthesias was more distal, while the presence of myofascial trigger points (MTPs) led to paresthesias in hands and to a lesser degree in fingers. Active MTPs predominantly resulted in proximal paresthesias and distal pains. The distribution of paresthesias and pains was almost identical in Groups 1 and 2. This may indicate a key role of nerve tissue damage rather than MTPs in the development of pain and paresthesias.

The duration of the disease in Group 1 was 2.6 ± 0.3 years ($P_{I-III} < 0.05$), in Group 2 — 4.0 ± 0.3 years ($P_{I-II} < 0.05$, $P_{II-III} < 0.001$, $P_{II-IV} < 0.01$), and in Group 3 — 1.3 ± 0.2 years. The longest duration was reported in Group 2. This was due to a combination of peripheral nerve damage and MTPs. The shortest duration was reported in Group 1 (without active MTPs), which allowed estimating time required for MTP development after peripheral nerve damage.

83.8% of patients in Group 2 reported sleep disorders. Severity of nocturnal paresthesias: patients from Group 2 had severe paresthesias in 56.8% of cases; no patients from Group 3 had severe paresthesias. Severity of nocturnal paresthesias according to the VAS scale: Group 1 — 7.7 ± 0.3 ($P < 0.05$), Group 2 — 9.2 ± 0.4 ($P < 0.05$), and Group 3 — 4.2 ± 0.1 ($P < 0.05$).

According to our results, MTPs in Groups 2 and 3 were most common in the trapezius muscle as well as in the greater and smaller pectoral muscles. Group 3 included more patients with MTPs in the

Table 2. *Results of NCV study in patients with nocturnal painful paresthesia m/s (M ± SD)*

Groups	Side	
	Affected	Healthy
First (n = 12)	33.93 ± 3.14	52.06 ± 6.54
Second (n = 14)	40.02 ± 3.84	55.81 ± 5.66
Third (n = 10)	49.33 ± 4.18	53.12 ± 6.21

brachioradial and pronator teres muscles. MTPs in the brachioradial muscle induced pains and paresthesias in the wrist and the first web space. MTPs in the pronator teres led to the development of reflected pains in the forearm and deep in the palm of hand. The incidence of MTPs in Groups 2 and 3 was almost equal, which put into question the role of peripheral nerve damage in their development.

A study of sensory fibers conductivity in the upper limbs by electric stimulation allowed us to confirm nerve damage in patients from Groups 1 and 2. Nerve conduction velocity (NCV) in these fibers slowed down in distal regions, and the amplitude of the action potential decreased. NCV delay in sensory fibers dominated over amplitude decrease, which indicated predominant nerve demyelination typical for tunnel syndromes. In Group 3, no sensory fiber damage was found, which allowed us to classify deficiency symptoms as signs of myofascial pain syndrome.

Table 2 presents the results of NCV measurement in sensory fibers for patients from the study groups.

The analysis of the results indicated a relative decrease in the response amplitude in the affected side of patients from Groups 1 and 2. The amplitude of the sensory potential in the affected side of patients from Group 3 exceeded values obtained for Groups 1 and 2 ($P_{I-III} < 0.05$, $P_{II-III} < 0.05$). Therefore, the study of sensory fibers conductivity in the upper limbs provided a neurophysiological proof of nerve damage in patients from Groups 1 and 2. Patients from Group 3 had no neurophysiological signs of sensory fibers disorder in the upper limbs, both in the affected and healthy sides.

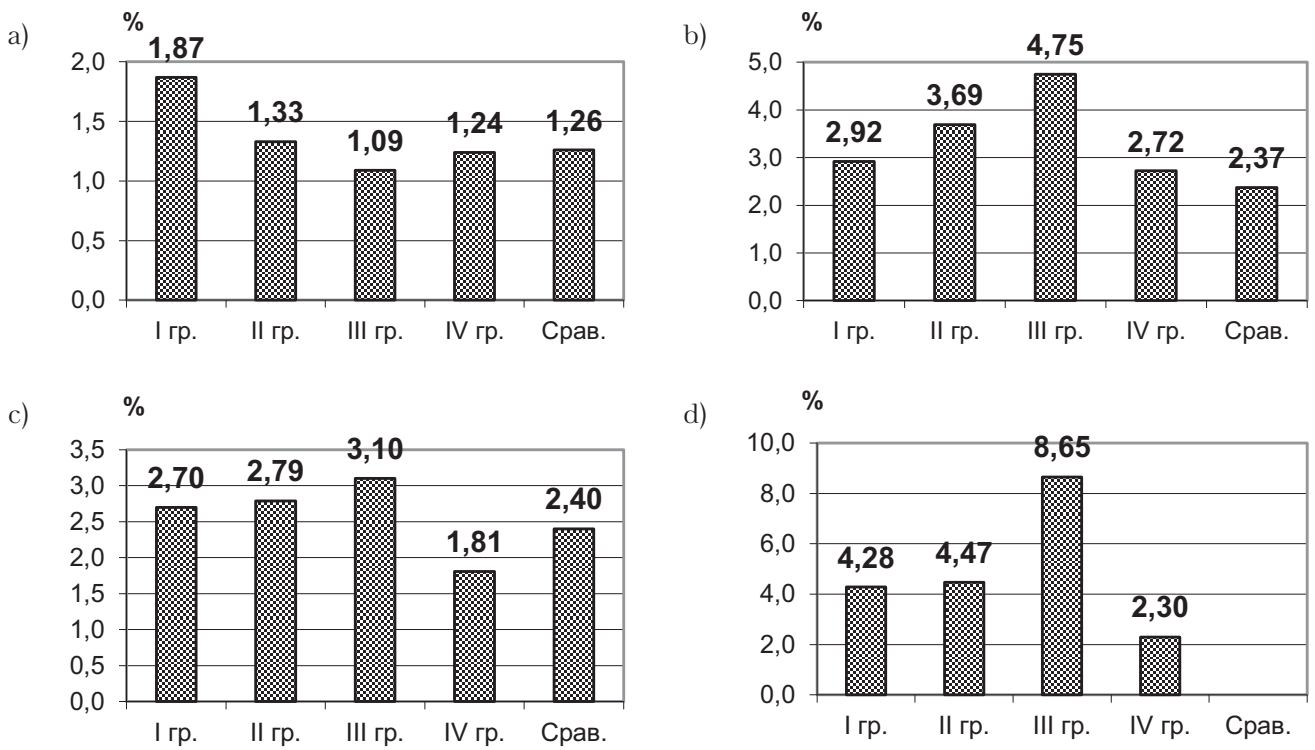

Figure 1. *Peak amplitudes of short-latency somatosensory evoked potentials, ms: a – peak N13, b – peak P17, c – peak N20, d – peak Px*

A study of short-latency somatosensory evoked potentials (SSEPs) allowed us to determine the preserved conductivity of central sections of deep sensitivity pathways in patients from Groups 1, 2, and 3.

Amplitude characteristics of SSEP peaks reflected both the conditions of impulse conduction and the excitability of structures generating these peaks. The amplitude of N20 peak was higher in Groups 1, 2, and 3 than in the comparison group (healthy volunteers). The amplitude of N20 peak in Group 1 was 2.70 ± 0.36 μV, in Group 2 — 2.79 ± 0.40 μV, in Group 3 — 3.1 ± 0.37 μV. In the control group, the amplitude of N20 peak was 1.81 ± 0.44 μs, and in the comparison group — 2.40 ± 0.38 μV (Fig. 1c). Increased amplitude of cortical peaks has been described in clinical studies and experiments as a result of acute and chronic pain.

The amplitude of the thalamic peak P17 was increased in Group 3 in comparison with the other groups (Fig. 1b). The amplitude of P17 peak in Group 1 was 2.92 ± 0.24 μV, in Group 2 — 3.69 ± 0.23 μV, in Group 3 — 4.75 ± 0.57 μV.

In the control group, the amplitude of P17 peak was 2.72 ± 0.26 μV — significantly lower than in Groups 2 and 3 and not different from the value obtained for Group 1. In the comparison group, the mean amplitude of P17 was 2.37 ± 0.26 μV. In patients from Group 3, the amplitude of P17 exceeded values obtained for other groups. The obtained data allowed us to hypothesize that pains and paresthesias induced by tunnel and traumatic neuropathy differed in their pathogenesis and the locus of the development of a pathologically enhanced excitation generator.

The analysis of SSEP peaks in patients from Groups 1, 2, 3, and 4 revealed Px peak in the CVI-Fpz lead with a latency of 21 to 35 ms (Fig. 2).

The analysis of SSEP in healthy volunteers showed no such peak despite identical experimental conditions. The analysis of Px peak parameters revealed the following.

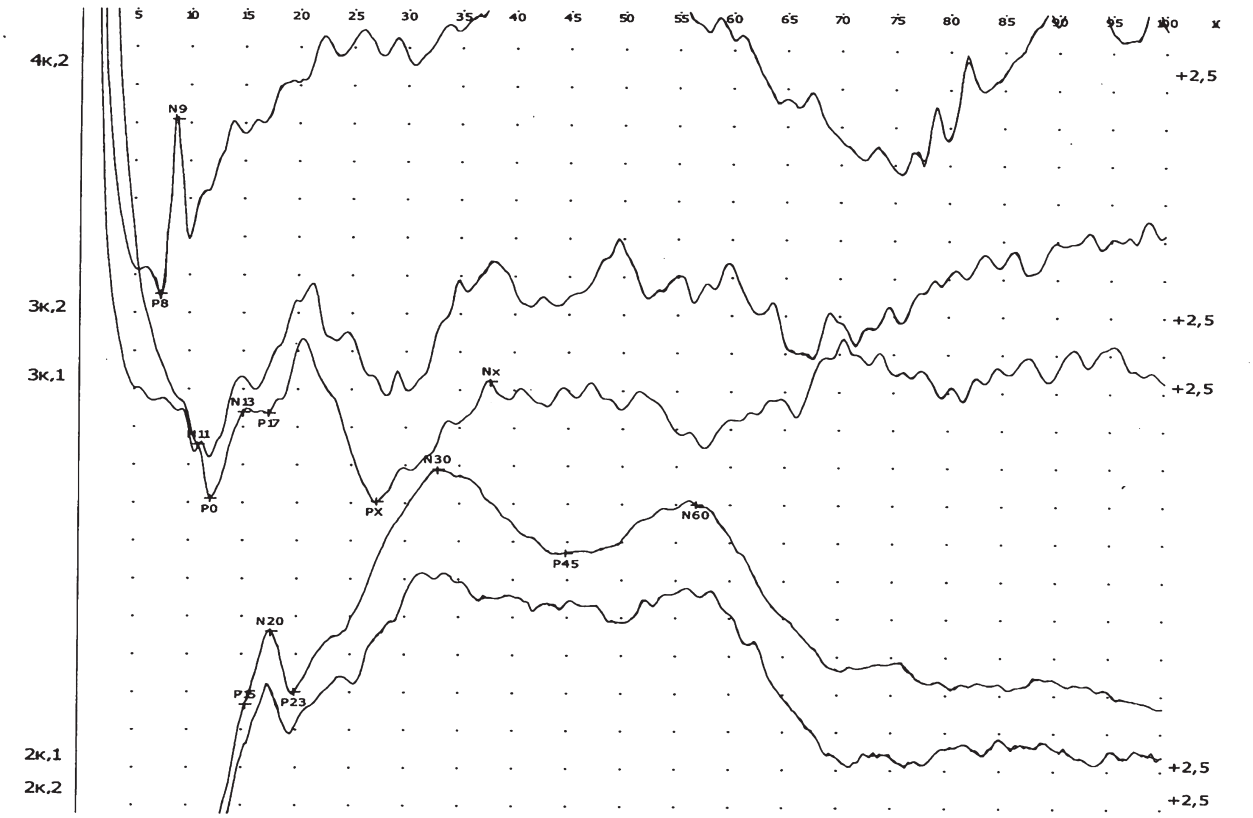

Figure 2. *Short-latency somatosensory evoked potentials with stimulation of the right median nerve in a patient with carpal tunnel syndrome and active MCPF in the brachial muscle and pronator teres*

The amplitude of Px peak in Group 1 was 4.28 ± 0.56 μV, in Group 2 — 4.47 ± 0.68 μV, in Group 3 — 8.65 ± 1.54 μV, in the control group — 2.30 ± 0.85 μV. In the comparison group, no Px peaks of SSEP were observed. It can be seen that the amplitude of Px peak in Group 3 was higher than in other groups ($P_{I-III} < 0.01$, $P_{II-III} < 0.05$, $P_{III-IV} < 0.01$).

The diagram shows that Px peak with a latency of 26.8 ms was registered at Channel 3 (CVII-Fpz). The latency of Px peak in patients from the comparison group had the following values: Group 1 — 21.98 ± 1.31 ms, in Group 2 — 27.46 ± 1.04 ms, in Group 3 — 26.49 ± 1.14 ms, in the control group — 22.63 ± 1.49 ms. The obtained data show that the latency of Px peak in patients from Groups 2 and 3 was higher than in patients from Group 1 and from the control group ($P_{I-II} < 0.01$, $P_{I-IV} < 0.05$, $P_{I-III} < 0.01$).

It was impossible to find its source; however, CVI-Fpz lead and the peak's positivity indicated that it was thalamic in nature. It is known that thalamic damage due to a hemorrhage or ischemia can result in excruciating burning pains (causalgia) in a certain part of the body. This indicated the possibility that the origin of the pain was in the thalamus.

Appearance of a previously undescribed peak may mean the development of a pathologically enhanced excitation generator that induces painful paresthesias. This peak may be connected both to the pain syndrome and to paresthesias observed in the examined patients.

Conclusions

Active myofascial trigger points were the root cause of nocturnal painful paresthesias in patients with myofascial pain syndrome of the shoulder girdle and upper limbs. In patients with painful hand paresthesias induced by tunnel mononeuropathies of the upper limbs, myofascial trigger points had no effect on the clinical course. The intensity of painful paresthesias was higher in patients with tunnel neuropathies than in patients with myofascial pain syndrome.

Therefore, the syndrome of nocturnal painful paresthesias was mixed in etiology and had, as an initial link, myofascial pain syndrome. The detection of a pathologically enhanced excitation generator in the suprasegmental sections of the sensitive pathway when short-latency somatosensory evoked potentials are recorded provided an adequate method for diagnosing painful paresthesias.

Conflict of Interests

The authors declare no conflict of interests.

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