

# CHANGES OF THE PRODUCTION FACTORS' COMPLEX ON THE STATE OF AFFERENT SOMATOSENSORY CONDUCT WAYS IN VERTEBROGENIC SPINE PATHOLOGY IN MINERS

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## INTRODUCTION

Pathology of the lumbosacral spine takes about 30% of the overall morbidity, and 20–30% of all diseases of the nervous system and more than 80% of the peripheral nervous system. About 80% of all healthcare system costs accounts for the treatment of back pain [1, 2].

The prevalence of occupational diseases of the peripheral nervous system among the persons of employable age is characterized by high detectability, often with loss of employability and disability. These are the most frequent of the diseases which lead to disability of persons younger than 45 years, exceeded only by diseases of the cardiovascular system and joints. When analyzing the frequency of back pain in patients with vertebral disorders it was revealed that the pain syndrome in the lumbar region was a multifactorial problem, and production factors are risk factors of back pain [3, 4].

There is no doubt that it is important to study the spine stem mechanisms of formation of autonomic motor reflex responses to the adverse effects of the complex environment factors.

Current knowledge on the functioning mechanisms of pain and analgesia are based on anatomical and morphological, neurophysiological and biochemical studies.

Considering the problem in general, it may be noted that pathophysiological mechanisms of lumbosacral radiculopathy with pain syndrome in miners remain insufficiently studied [5, 6]. Characteristics and extent of changes on peripheral, segmental and central levels of the sensor motor system taking into account emotional-effective disorders in occupational vertebrogenic diseases are underestimated that affect the choice of making the examination of work capacity, connection of the disease with the occupation, adequacy and effectiveness of individual treatment and rehabilitation program [7, 8].

The above mentioned issues are of great practical importance, and allow to propose evidence-based

## Abstract

### Background

We carried out a comprehensive analysis of all parts of afferent somatosensory systems in the presence of pain of varying severity in miners. The analysis was based on modern neurophysiological studies.

### Methods

We conducted the symptoms complex analysis of indicators of electrophysiological studies in patients with mild to moderate degrees of pain. The control group consisted of 28 patients.

### Results

Neurophysiological changes are characterized by generalized mixed affect of the peripheral sensory nerves and  $1\alpha$ -afferents of H-reflex arc. The failure of systems of pain control has dominantly deafferentation character. A characteristic difference between them is also the involvement of  $1\beta$  fibers (motor nerves and efferent link of the H-reflex).

### Conclusions

It was found out that the data obtained allow to extend the understanding of the mechanisms of formation of neuropathic pain in the vertebral spine pathology.

### Keywords

pain reception, afferent neurons, proprioception.

differential approaches to treatment and expert issues solutions for introduction into clinical practice, taking into account severity of occupational diseases of musculoskeletal system.

## THE STUDY AIM

is rationale of the influence of production factors on the state of the afferent somatosensory system in the process of miners' work.

## MATERIALS AND METHODS

The studies were conducted within the frameworks of the Applied Research Program of the Ministry of Healthcare of the Republic of Kazakhstan in the Republican State Governmental Enterprise “National Center of Labor Hygiene and Occupational Diseases” (NC LH OD) 0103RK, supervised by the Department of Science and human resources (2006–2009).

We conducted a neurological and electrophysiological examination of miners of LLP Zhezkazgan mining-metallurgical combine “Kazakhmys Corporation” and JSC “Arcelor Mittal” with the pain syndrome of vertebral pathology and lumbosacral spine who were patients in the neurological department of the clinic of the National Center of Labor Hygiene and Occupational Diseases. Control group were miners (28 people) who did not have signs of pain syndrome. According to outpatient medical records these patients were not registered in the dispensary at the vertebral spine pathology.

The miners were divided into 3 groups on the basis of severity of a leading clinical syndrome: group 1 included 83 miners with the pain syndrome (average age is  $41.8 \pm 9.6$  years) – not sharply expressed degree of reflex tonic pain syndrome, group 2 included 46 miners with moderate expressed degree of reflex tonic and radicular-pain syndrome (mean age  $44.6 \pm 11.4$  years), group 3 included 19 miners (average age is  $48.0 \pm 10.9$  years) miners with the severe degree.

Electrophysiological study: definition of somatosensory afferent-efferent pathway, evoked potentials (EP) and brain structures for the objective assessment of functional status at different levels of specific and nonspecific afferent systems.

To record somato-sensory evoked potentials (SSEP) we used electromyograph “Neurosoft”, Russia. SSEP was recorded from the surface of the head with conventional disc electrodes with the diameter of 5 mm. When recording the potentials caused by stimulation of the right median nerve, the registration was held at Yerba (over the Brachial Plexus), C7 in the cervical (above the seventh vertebra) Fz in the frontal region, C3 and C4 (zone projection of somatosensory cortex on the left and right). The respective components were identified in trass N9 (brachial plexus response), N11-N13 (cervical segments of the spinal cord), N20-R25 (zone of the cortical projection of arm) (according to the international system of electroencephalographic leads “U-20%) when recording the potentials caused by stimulation of the left and right tibial nerve.

Statistical analysis was performed on IBM-compatible personal computer Pentium using the program Microsoft Excel and statistical analysis program AnalystSoft, StatPlus, 2007 version.

The calculation of basic statistical parameters was carried out using the methods of parametric and nonparametric descriptive statistics. A comparison between different samples of data was performed using Student’s *t*-test for independent samples to define levels of reliability.

## RESULTS AND DISCUSSION

The main clinical manifestations of disease in the patients examined were pain in lumbosacral localization (65 patients) radiating to the lower limbs (65 patients, or 100%) of moderate and severe degrees.

The results of SSEP components in patients with occupational pathology of vertebral pain syndrome, suggest that the main neurophysiological characteristics of chronic pain syndrome in the absence of structural lesions of the brain is hyperexcitability of its afferent systems. The necessary condition for occurrence and continuation of the pain is the relative safety of specific projections carrying information to the cerebral cortex by fast conducting fibers.

In a study of indicators of SSEP it was revealed that an afferent wave of excitation, namely, pain in the ways of general sensitivity, took place in the posterior columns of the spinal cord (component N22), then through the stem sections of the brain (component N30) and later in cortex (a component of P38, R46). These curves reflect the passage of the nerve impulse to the relevant structures and can detect subclinical slowing of the pulse indicating the failure of the conduction system (Table 1).

**Table 1.** Indicators of amplitude characteristics of the SSEP components in patients with vertebral pathology of the lumbar spine depending on the pain syndrome

Component	Amplitude, mCB			
	Control group n=28	Control 1 n=83	Group 2 n=46	Group 3 n=19
N22	1.1±0.5	0.74±0.012	2.54±0.02*	3.45±0.03**
N30	0.8±0.3	0.59±0.06	2.48±0.04*	3.39±0.05**
P38	2.4±1.5	1.94±1.0	3.5±0.09*	4.1±0.08**
P46	2.3±1.3	2.0±0.81	3.7±0.9	4.5±0.6*

**Note:** \* – Significant difference between the indices of the control group and the surveyed group, \* –  $p < 0.05$ , \*\* –  $p < 0.01$

We revealed the increase of amplitude of characteristics depending on the pain, but it is more expressed in mild to moderate degrees of pain. Thus, the amplitude of component N 22 reflecting the activation of neuronal elements of the spinal level, mainly cauda equine and medullary cone increased, with moderate-expression and expressed pain syndrome.

The amplitude of the component N30 representing the activation of the posterior columns of the spinal cord at the border of the cervical spine and medulla oblongata with mild to moderate degree increased.

Indicators of the amplitude component P38 and 46 reflecting the activation of the somatosensory cortex had tendency to increase at a moderate and significant degree of pain.

Thus, increasing the components of SSEP amplitude, interval, latency in patients with apparent pain syndrome, depression and anxiety may indicate the development of central sensitization of the conduction system (N30, P38, P46), realized in nonspecific medio-basal limbic structures of the brain [7, 8].

N10-N20 intervals characterize the conduction on the ascending pathways of the spinal cord, the interval N20-P30 is similar to the central conduction time (CCP).

In patients with the moderate degree of pain there was a significant lengthening of the intervals: N10-N13; N13- N20; N20-N30. These indicators have most significantly increased in the apparent degree of pain (Table 2).

**Table 2.** Parameters of basic intervals of SSEP components in patients with vertebral pathology of the lumbar spine, depending on the pain syndrome

Components	Intervals, ms			
	Control group n = 28	Group 1 n=83	Group 2 n=46	Group 3 n=19
N10-N13	7.65±1.04	12.34±2.44	15.2±2.01*	19.8±2.3**
N13-N20	8.36±1.56	15.63±2.38	18.6±3.2*	20.4±3.1**
N20-N30	16.1±1.55	28.0±3.15	32.3±2.56*	37.1±1.9**

**Note:** \* – Significant difference between the indices of the control group and the group surveyed, \* –  $p < 0.05$ , \*\* –  $p < 0.01$

The increase of the interval N22-P38 with the preservation of CCP indicating a violation of the ascending pathways of the spinal cord, depending on the severity of pain in patients with vertebral lumbar pathology was marked.

Thus, under the influence of the complex factors of production (lesion of spinal roots L5-S1) there is the reaction of myelinated  $1\alpha$ -sensitive and motor guides. This pain syndrome occurs by stimulation of low-threshold mechanoreceptors  $1\beta$ -fibers not destroyed stimuli on the background of central sensitization.

In patients with vertebral pathology with mild pain syndrome there was a significant increase in latency of N30 with a tendency to increase the component P38, P 46, and in patients with mild to moderate

degree of pain there was a significant lengthening of the latency of components N22, N30, P38, P 46 (Table 3).

**Table 3.** Indicators of latency of components of somatosensory evoked potentials of patients with vertebral pathology of lumbar spine depending on the pain

Component	Latency, ms			
	Control group n=28	Group 1 n=83	Group 2 n=46	Group 3 n=19
N22	22.1±2.2	36.2±6.1	42.1±4.9*	51.3±5.4**
N30	29.8±2.8	41.2±3.9*	49.6±4.1*	60.5±5.7**
P38	38.3±3.3	43.8±4.8	52.6±3.7*	63.9±4.2**
P46	46.4±3.2	49.1±4.0	54.3±3.4*	71.2±6.7**

**Note:** \* – differences reliability between the indices of the control group and the group of the patients, \* –  $p < 0.05$ , \*\* –  $p < 0.01$

In patients with vertebral pathology with moderate and severe pain syndrome there was a significant increase in latency of components N22, N30, P38, P46, respectively, characterizing the intensification of the processes of inhibition of neural structures of the spinal cord.

Also, in patients with apparent pain syndrome there was a significant increase in the duration of intervals of absolutely all the components, indicating exhaustion and desynchronization of the higher antinociceptive centers.

As it is seen from Table 3, in patients with mild to apparent degree of pain we revealed a significant lengthening of N22 latency indices up to  $42.1 \pm 4.9$  ms,  $P < 0.05$  and  $51.3 \pm 5.4$  ms,  $P < 0.01$  (control  $22.1 \pm 2.2$  ms); N 30 to  $49.6 \pm 4.1$  ms,  $P < 0.05$  and  $60.5 \pm 5.7$  ms,  $P < 0.01$  (control  $29.8 \pm 2.8$  ms); P38 to  $52.6 \pm 3.7$  ms,  $P < 0.05$  and  $63.9 \pm 4.2$  ms,  $P < 0.01$  (control  $38.3 \pm 3.3$ ); R46 up to  $54.3 \pm 3.4$  ms,  $P < 0.05$  and  $71.2 \pm 6.7$  ms,  $P < 0.01$  (control  $38.1 \pm 3.3$  ms), respectively, that is characterizing the affect of fibers of peripheral sensory fibers (decrease in SSEP) of 1a-afferents of the H-reflex arc and L3-L4 afferents demyelination on a spinal level (data of SSEP study with stimulation of n. tibialis).

Thus, the idea of understanding the mechanisms of the production factors influence on the severity of clinical manifestations with pain syndrome in vertebral pathology in miners at the level of the afferent somatosensory system was enhanced in the scientific work.

## CONCLUSIONS

1. Found out changes of SSEP in vertebrogenic spine pathology in miners is the criteria of the pain syndrome degree diagnostics.

2. Developed criteria for diagnosing disorders of pain sensitivity make it possible to carry out differential diagnosis of disorders in clinically ambiguous cases.

## REFERENCES

1. Neurophysiological studies in clinics. Ed. by Schekutev G.A. Moscow.: Antidor 2001.
2. **BROWN M.J., MARTIN I.R. ET AL.** Painful diabetic neuropathy. A morphometric study. Arch Neurol 2006; 33. – P. 164–171.
3. **SOKOLOV A.A., LIVSHITTS A.V.** Comparative evaluation of the EP in response to pain and somatosensory stimulation in man. Human Physiology. – 1985. – P. 734–742.
4. **DYCK P.J., LAMBERT E.H., O'BRIEN P.C.** Pain in peripheral neuropathy, related to rate and kind of fiber degeneration. Neurology.– 2005, 26. – P. 466–472.
5. **BIESCNBACH G., EICHBAUER-STRUM G., GRAFIGER P., ZAZGORNIK G.** Cerebrolisin in treatment of painful diabetic neuropathy. Eurorehab. – 2004. – P. 97–103.
6. **BONICA J.J.** The management of pain. Philadelphia: Lea and Febiger. – 2006.
7. **FIELDS H.L.** Painful dysfunction of the nervous system. In: H.L. Fields, Pain. McGraw-Hill Book Company. – New York. – 2005. – P. 133–169.
8. **FIELDS H.I. BESSON J.-M. (EDS.).** Pain modulation. In: Progress in Brain Research, Elsevier Science Publishers. – Amsterdam. – 2007.