NEUROPHYSIOLOGICAL CHANGES IN THE AFFERENT SOMATOSENSORY SYSTEM INDICES IN THE CASE OF VERTEBROGENIC SPINE PATHOLOGY IN MINERS

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Abstract
Objectives: The aim of the paper was to prove that job conditions impact the state of the afferent part of the somatosensory system in miners. Materials and Methods: Data analysis of the electrophysiological examination of the syndrome in 148 patients, aged from 28 to 55 years, with a mild, moderate and severe degree of the pain syndrome was performed. The control group included 28 people without any pain symptoms. The method used was that of somatosensory stimulated potential (SSP) with the potentials amplitude and latency main components taken into consideration. Results: It was proven that the true decrease of the somatosensory stimulated potential SSP N22 (p < 0.05) component amplitudes by 41%; N30 component amplitude tend to decrease by 26%. This proves that the true N22 (p < 0.01) component latency increase by 63.8% corresponds to afferent excitation wave conductibility under the pain syndrome of vertebral pathology through sensitivity pathways mainly in the posterior spinal cord columns and then, through the parts of the brain stem, involving the cerebral cortex, which is confirmed by the fact that the P38 and P46 components amplitudes tend to decrease. In addition to this, the proven N10–N13 (p < 0.05), N13–N20 (p < 0.05), N10–N20 (p < 0.05) intervals increases by 43.5–41.8–38.7%, respectively, correspond to the nervous impulse conductibility through the peripheral nervous system structures and allow to reveal the subclinical slowdown of impulse conductibility, which indicates that the conducting system is changed even under a mild pain syndrome. Conclusions: It was found that the data obtained allow for the better understanding of how the neuropathological pain syndrome under vertebral spine pathology is formed.

Key words: Miners, Vertebral pathology, Pain syndrome, Somatosensory Stimulated Potential (SSP), Pain reception, Afferent neurons

INTRODUCTION

Lumbosacral pathology is responsible for 30% of the incidence rate of non-occupational diseases, 20% of the nervous system diseases and more than 80% of the peripheral nervous system diseases [1,2]. Almost 80% of all health care costs account for the back pain treatment. The spread of occupational peripheral nervous system diseases among the people of active working age is highly detectable and often is the cause of temporary or complete disability. These are the most common cases, after cardio-vascular events and pathology of joints, which cause limiting disability in people younger than 45 years old. The analysis of the rate of lumbalgia in patients with vertebral pathology showed that the low back pain syndrome is a problem caused by a set of factors, workplace conditions being the risk factors [3,4].
The study of the way spinal stem mechanisms form reflex motor reactions to a complex of negative factors of working place is, undoubtedly, of big importance. The present day knowledge about pain and analgesic mechanisms is based on the data of anatomical morphological and neurophysiologic examinations. Looking at the problem as a whole, one can state that pathophysiological mechanisms of lumbo-sacral radiculopathy in miners with the pain syndrome have not been adequately studied [5,6].

The characteristics and the degrees of the peripheral, segment and central sensorimotor system level changes teamed with emotional impact disorders under occupational vertebral diseases remain underestimated, which complicates the examination of disability and the identification of the link between the disease and the job; it also influences the choice of an adequate and efficient individual treatment and rehabilitation program [7,8].

The issues stated above, which are of practical importance, call for the introduction of evidence based differentiated approaches into clinical practice, the methods to be used in treatment and expert decision-making taking into consideration the degree of professional locomotorium pathology.

**OBJECTIVES**

The aim of the research was to prove that job conditions impact the state of the afferent part of the somatosensory system in miners.

**MATERIALS AND METHODS**

The patients with the vertebral pain syndrome and those with lumbo-sacral pathology were all miners from the Jezkazgan Mining and Metallurgical Plant, Kazakhmys Inc. and coal miners from JSC Arselor Mittal. There was a control group of 28 patients with no pain syndrome and no vertebral pathology record. All the patients underwent neurological and electrophysiological examinations.

The examined patients were divided into three groups according to the severity degree of the pain syndrome: group 1–83 patients of 41.8±9.6 years of age on average with mild reflex-tonic pain syndrome; group 2–46 patients in the age range of 33.6–56 years with moderate radicular pain syndrome; group 3–19 patients of 48.0±10.9 years of age with severe degree of the pain syndrome.

To make an objective assessment of the function of the specific and non-specific afferent systems on various levels of cerebral structures, the somatosensory stimulated potentials (SSP) of the somatosensory afferent-efferent pathway were detected.

NeuroSoft electromiograph, Russia, was used to record SSP. Somatosensory stimulated potentials were recorded through usual 5 mm disc electrodes placed on the head of the subject. When stimulating the right median nerve, SSP were recorded at Erb’s point (above brachial plexus), C7, in cervical part (above the seventh vertebra), Fz in frontal part, C3 and C4 (right and left projection areas of the somatosensory cerebral cortex). The corresponding tracks revealed component N9 – brachial plexus response, N11–N13 – spinal cord cervical segments, N20–P25 – cortex projection area of a hand (according to the Encephalographic Abduction International System U – 20%) while recording SSP under the stimulation of left and right tibial nerve.

The statistical analysis was conducted using 2007 Microsoft Excel, AnalystSoft and StatPlus program versions on IBM-compliant Pentium PC.

The main statistical parameters were calculated using parametric and nonparametric descriptive statistical methods. The comparison of the average results between the samples was made via independent sample Student t-test with the defined difference reliability level.
RESULTS AND DISCUSSION

General clinical manifestation in the examined patients had the form of ambient to severe lumbar-sacral pains, 65 patients, and pains radiating to the lower limbs, 65 patients (100%).

The results of the SSP components examination allow to hypothesize that the main neurophysiology characteristic for the chronic pain syndrome in patients with occupational vertebral pathology in the case of no structural cerebral affection is hyperexcitability of the cerebral afferent systems. In order for the algesis to appear and sustain, the necessary condition is the relative preservation of specific projections transporting information to the cerebral cortex via rapidly conducting fibers.

The analysis of the SSP data revealed that afferent wave excitation, namely the pain syndrome conduct, through the general sensitivity pathways was through the posterior columns of the spinal cord (component N30) and further, to the cortex (components P38, P46). The resulting graphs reflect the nervous impulse passage through the relevant structures, which helps to reveal the subclinical slowdown of the impulse conduction, thus detecting the damage of the conducting system (Table 1).

The amplitude characteristics increase was found to depend on the degree of the pain syndrome, being most expressed in the case of moderate and severe degrees. So were the characteristics of the component N22 range under the moderate and severe pain syndromes, which corresponds to neuronal elements of the spinal cord level affection, mainly those of cauda equina and medullary cone, respectively. The component N30 range, reflecting the affection of the posterior columns of the spinal cord at the boundary of the cervical spine and medulla under moderate and severe degrees, increased.

The range characteristics of the P38 and P46 components, which correspond to cerebral cortex somatosensory area activation, only tended to increase under the moderate and severe pain syndromes.

Thus, an increase in the SSP amplitude components, those of interval and latency in patients with the severe pain syndrome, depression and anxiety may signal the development of the central conductive system sensitization (N30, P38, P46), which manifests itself at nonspecific mediodasal limbic cerebral structures [7,8].

The N10–N20 interval characterizes the conduction through ascending spinal paths, N20–P30 interval is similar to the time of the central impulse conduction.

In the patients with the moderate degree of the pain syndrome, a true N10–N13, N13–N20, N20–N30 interval broadening was indicated. In the case of the severe pain syndrome, the indices increased (Table 2).

The N22–P38 interval showed an increase while the central impulse conduction remained unaffected, which indicates ascending spinal paths conduction failure that

Table 1. The SSP component range indices in the patients with vertebral lumbar pathology according to the degree of the pain syndrome

<table>
<thead>
<tr>
<th>Components</th>
<th>control group (N = 28)</th>
<th>group 1 (N = 83)</th>
<th>group 2 (N = 46)</th>
<th>group 3 (N = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N22</td>
<td>1.1 ±0.5</td>
<td>0.74±0.012</td>
<td>2.54±0.02*</td>
<td>3.45±0.03**</td>
</tr>
<tr>
<td>N30</td>
<td>0.8 ±0.3</td>
<td>0.59±0.06</td>
<td>2.48±0.04*</td>
<td>3.39±0.05**</td>
</tr>
<tr>
<td>P38</td>
<td>2.4 ±1.5</td>
<td>1.94±1.00</td>
<td>3.50±0.09*</td>
<td>4.10±0.08**</td>
</tr>
<tr>
<td>P46</td>
<td>2.3 ±1.3</td>
<td>2.00±0.81</td>
<td>3.70±0.90</td>
<td>4.50±0.60*</td>
</tr>
</tbody>
</table>

SSP – somatosensory stimulated potential.
Reference: index difference reliability between the control group and the patients under the examination; * p < 0.05, ** p < 0.01.
indicating exhaustion and desynchronization of the higher antinociceptive centers occurred.

As can be seen from Table 3, patients with the moderate and severe pain syndrome had a true increase of latency indices: N22 to 42.1±4 ms, p < 0.05 and 51.3±5.4 ms, p < 0.01 (control: 22.1±2.2 ms); N30 to 49.6±4.1 ms, p < 0.05 and 60.5±5.7 ms, p < 0.01 (control: 29.8±2.0 ms); P38 to 52.6±3.7 ms, p < 0.05 and 63.9±4.2 ms, p < 0.01 (control: 38.3±3.3 ms); P46 to 54.3±3.4 ms, p < 0.05 and 71.2±6.7 ms, p < 0.01 (control: 38.1±3.3 ms), which signaled damage of the peripheral sensitive fibers (SSP decrease) of H-reflex 1α-afferent arc and rapidly conductive L3–L4 1β-afferents demyelination on the spinal level (SSP data under n. tibialis stimulation).

Thus, the present work broadens the understanding of the way in which occupational factors impact the severity of the clinical manifestation of the pain syndrome on the

It, thus, can be concluded that under the influence of a set of job factors (damage of L5–S1 radicles myelinated 1α-sensitive and motor conductors are found to be affected. This pain syndrome occurs under tissue damaging 1β-stimuli affecting low-threshold mechanoreceptors under central sensitization.

In the patients with vertebral pathology under the mild pain syndrome, a true increase of N30 latency and P38, P46 components (tendency - trend) trend to increase were indicated, while in the case of the moderate and severe pain syndrome, a true increase of N22, N30, P38, P46 component latency was detected (Table 3), the latter characterizing the increased processes of the spinal cord neural structures inhibition.

It was also found that under the severe pain syndrome, a true increase of absolutely all components’ length depends on the degree of pain syndrome in patients with lumbar pathology of vertebral genesis.

Table 2. The SSP main components interval parameters in the patients with lumbar vertebral pathology with varying degrees of the pain syndrome

<table>
<thead>
<tr>
<th>Components</th>
<th>control group (N = 28)</th>
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<th>group 2 (N = 46)</th>
<th>group 3 (N = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N10–N13</td>
<td>7.65±1.04</td>
<td>12.34±2.44</td>
<td>15.2±2.01*</td>
<td>19.8±2.3**</td>
</tr>
<tr>
<td>N13–N20</td>
<td>8.36±1.56</td>
<td>15.63±2.38</td>
<td>18.6±3.20*</td>
<td>20.4±3.1**</td>
</tr>
<tr>
<td>N20–N30</td>
<td>16.10±1.55</td>
<td>28.00±3.15</td>
<td>32.3±2.56*</td>
<td>37.1±1.9**</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1.

Table 3. The SSP components latency indices in the patients with lumbar vertebral pathology with varying degrees of the pain syndrome.

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</thead>
<tbody>
<tr>
<td>N22</td>
<td>22.1±2.2</td>
<td>36.2±6.1</td>
<td>4.1±4.9*</td>
<td>51.3±5.4**</td>
</tr>
<tr>
<td>N30</td>
<td>29.8±2.8</td>
<td>41.2±3.9*</td>
<td>49.6±4.1*</td>
<td>60.5±5.7**</td>
</tr>
<tr>
<td>P38</td>
<td>38.3±3.3</td>
<td>43.8±4.8</td>
<td>52.6±3.7*</td>
<td>63.9±4.2**</td>
</tr>
<tr>
<td>P46</td>
<td>46.4±3.2</td>
<td>49.1±4.0</td>
<td>54.3±3.4*</td>
<td>71.2±6.7**</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1.
level of the afferent somatosensory system under vertebral pathology in miners.

CONCLUSIONS

1. The detected SSP changes in miners with vertebral pathology allow to elaborate the pain syndrome degree diagnostic criteria.
2. The criteria help in differential diagnostics of diseases in clinical practice.

REFERENCES