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New strategies in the differential diagnostics and rational therapy of lumbosacral radiculopathies

Abstract

Dorsopathies make up the most significant group of chronic pain syndromes in neurology due to their high prevalence and resistance to treatment. It is especially difficult to manage patients with the so-called "failed back surgery syndrome" (FBSS). Unfortunately, nowadays there are no reliable ways to predict the outcomes of the conservative treatment of patients with dorsopathies. Otherwise, we could identify patients that demand intensive treatment including therapeutic blockades. We have studied the effectiveness of conservative treatment among 60 patients with lumbosacral dorsopathies–35 patients manifested with the exacerbation of sciatica, and in 25 we diagnosed FBSS. The results of our work provide evidence that, firstly, there is a definite resistance in the treatment of patients with FBSS and, secondly, the complex management of patients including neuromidin and epidural steroid blockade shows great efficiency and, thirdly, the evaluation of radicular latency duration with neuromidin test turns out to have important prognostic value that could be used to stratify the rational treatment of patients with dorsopathies.

Keywords: radiculopathy, failed back surgery syndrome, radicular latency duration, neuromidin, epidural steroid blockade

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Abbreviations: FBSS, failed back surgery syndrome; LSR, lumbosacral radiculopathy; CT, computed tomography; MRI, magnetic resonance imaging; ENMG, electroneuromyography; SSEP, somatosensory evoked the potential; TMS, Transcranial magnetic stimulation; RDAM, root delay assessment method; IMR, induced motor responses; RDI, root delay indicators

Introduction

Lumbosacral dorsopathy is the most outstanding pathology among nosologic forms, accompanied by chronic pain syndrome. The formation of the clinical patterns of dorsopathy is based on: microtraumatization of various elements of the spine (lifting heavy objects, prolonged uncomfortable posture, etc.) – in 70% of cases; age-related degenerative-dystrophic changes in the discs and facet joints – 10%; disc hernias – 4%; spinal stenosis – 3%; compression vertebral fractures caused by osteoporosis – 4%; spondylolisthesis – 2%; traumatic vertebral fractures–less than 1%; congenital abnormality of the spine (pronounced kyphosis, scoliosis, etc.) – also less than 1%. So the term "dorsopathy" covers a rather large heterogeneous group of diseases, where vertebrogenic or discogenic lumbosacral radiculopathy, despite its relatively low occurrence, plays a key role, as it is often accompanied by "unbearable" pain, results in significant neurological disorders (flaccid paresis or paralyses in the lower extremities) and requires surgical intervention. Apart from vertebrogenic origin, compression of the spinal nerve roots can be caused by developmental anomalies (arteriovenous malformations, arachnoid cysts), neoplasms (neuromas, primary and metastatic vertebral tumors, etc.), infectious (herpes zoster, Lyme disease) and inflammatory diseases (sarcoidosis), endocrine disorders (hypothyroidism, diabetes mellitus), which is less than 1% of all cases of radiculopathies.

In medical practice, if patients have pain in their back and extremities, they are most often diagnosed with "radiculitis", as the clinical differential diagnosis of various pathogenetic forms of dorsopathies is complicated due to nonspecific pattern of pain sensation ("pain cloud"), although the classical pattern of radiculopathy is a combination of radiating pain, paresthesia, hypesthesia, allodynia, flaccid paresis, and reduction (loss) of the respective tendon reflex in the innervation zone of the affected root. Moreover, the aforesaid symptoms worsen when coughing, sneezing, in the vertical position, and decrease in the horizontal position; the positive Lasègue symptom has a sensitivity of 80% and a specificity of 40% in case of disc protrusions.[1] Furthermore, as the root compression can occur in the root canal (between the posterolateral surface of the vertebral body and the superior articular process) and in the intervertebral foramen, the clinical pattern of radiculopathy has its own peculiarities: unlike the root compression by the disc hernia in case of the radicular canal stenosis or narrowing of the intervertebral foramen, the symptoms develop gradually. Moreover, such patients usually have the Spurling's sign of "intervertebral foramen": when the torso flexion to the affected side causes paresthesia, or pain develops and radiates to the innervation zone of the root compressed in the intervertebral foramen.[2]

Modern diagnostic strategy of lumbosacral radiculopathy (LSR) focuses mostly on neuroimaging examination methods. Furthermore, despite the fact that computed tomography (CT) and magnetic resonance imaging (MRI) have a similar sensitivity (70-90 and 60-100%, respectively) for the detection of intervertebral disc protrusions, the frequency of their application in clinical practice varies significantly in favor of MRI.[3] At the same time, CT, especially CT myelography, ensures a better differentiation of radiculopathy caused by facet joint pathology or narrowing of intervertebral foramina.

The next instrumental technique in terms of validity for radiculopathy diagnostics is electroneuromyography (ENMG), which enables the detection of abnormal neural changes in the muscles innervated by a single root (positive acute waves, fibrillation potentials, etc.), and impaired conductivity in the peripheral nerves formed with the participation of the affected spinal root. Some ENMG methods are considered routine; e.g. changes in the H reflex when stimulating the tibial nerve in the popliteal fossa area of marker

S1 radiculopathy, and the additional use of S1 root stimulation to provoke H reflex significantly increases the diagnostic sensitivity of the method. The clinical application of the somatosensory evoked the potential (SSEP) assessment method sometimes causes contradictory results, and its significance for radiculopathy diagnostics has not been defined until now.[4] Transcranial magnetic stimulation (TMS) with root delay time assessment has not found its application in neurological and neurosurgical practice, despite the studies carried out by a number of authors.[5,6] Besides for the diagnostic component, the assessment of the functional state of the compressed root have fundamental importance for the selection of treatment tactics (surgical or conservative), as the detection of disc protrusion with the dural sac compression based on the data of neuroimaging examination methods is possible in persons without signs of dorsopathy.[7]

In view of the aforesaid, the aim of this work was the approbation and modification of the root delay assessment method (RDAM) in patients with lumbosacral radiculopathy to identify the diagnostic significance and the possibility of applying RDAM in monitoring the effectiveness of conservative and operative therapy in patients of this profile.

Materials and methods

60 patients with L5-S1 LSR exacerbation (40 males and 20 females) aged 22 to 68 years with a duration of disease of 2-25 years were examined. The patients were divided into two groups comparable in sex and age: the first group consisted of 35 persons who had previously undergone conservative treatment for radiculopathy; the second group consisted of 25 persons who had undergone disc hernia surgery 3-6 months beforehand in various neurosurgical hospitals (standard discectomy with interlaminectomy) with FBSS. The control group for electrophysiological data analysis consisted of 15 persons with lumbosacral dorsopathy with pain syndrome of the radicular type in their clinical pattern (back pain irradiating in the leg); but sensory, reflex or motor disorders typical for radiculopathy were not identified.

All patients in the first and second groups underwent a thorough dynamic neurologic examination, ENMG with root delay assessment method (screening, one week after the completion of the treatment course), CT or MRI of the lumbar spine (once). Clinical neurologic examination was supplemented by testing patients using the numerical rating scale for pain (NRS).

A standard ENMG was performed using the myograph by Neirosoft Company (Russia); the amplitude of M-response, latency indicators and electrical impulse conductivity rate over the motor fibers of the peroneal and tibial nerves from two sides, F-wave study (identification of the minimum and average latencies) of the corresponding nerves were identified to assess the status of the peripheral nerves and roots. The magnetic stimulator Magstim (UK) generating biphasic rectangular impulse fed through a circular coil was applied to carry out magnetic stimulation and identify the root delay indicators at the L4, L5, S1 level. The registration device was a myograph by Neirosoft company (Russia). The stimulation of L4, L5, and S1 roots was performed according to B. Hecht's method[8] with the circular coil at the level of the spinous processes of the corresponding vertebrae, so that the examined motor roots ware stimulated in the area of their outlet from the intervertebral foramen. The induced motor responses (IMR) were obtained during the study, followed by an analysis of the range, shape and root delay indicators (RDI). The IMR range was calculated as a distance from the isoline to the negative peak. The RDI calculation was carried out by comparing the excitation time with the magnetic stimulation of the root outlet areas at the level of the corresponding vertebrae and the estimated time of F-wave excitation conduction when stimulating the corresponding nerve at a distal point. The values RDI below 3.8ms were considered as normal values.[8] A modified RDAM applied in the work as follows: the background indicators of radicular delay were first obtained according to the standard procedure, then 15mg of Neuromidin were administered intramuscularly and the check control recording of root delay was performed 30 minutes later.

MRI was performed in the prone position in the sagittal and frontal planes using a Magnetom Sonata device (Siemens) with the magnetic field intensity 1.5Ts and cut thickness of 3mm. Computed tomography was performed using Siemens Emotion Duo device with a cut thickness of 1.5-2mm. Patients with signs of the central canal stenosis were excluded from the study. Statistical processing of the data obtained was carried out according to the generally accepted methods.[9]

After the screening examination, the patients of the first and second groups gave informed consent and were divided into two subgroups for further treatment for 3 weeks: conservative treatment was carried out according to the generally accepted methods (NSAID, vitamin therapy, physiotherapy, massage) in subgroups Ia (n=17), IIa (n=12). In subgroups Ib (n=18) and IIb (n=13) along with the same conservative therapy, we applied Cutlen epidural blockade[1] with novocaine (40ml), dispropan (1 ampule) and vitamin B12 (500mg), and neuromidin–first intramuscularly 15mg daily for 7 days, then orally 20mg 3 times daily for two weeks. Selective COX-2 inhibitor Arcoxia 90mg daily for two weeks was chosen as the main NSAID; vitamin B therapy consisted of Milgamma, first for 10 days intramuscularly 2ml, and then 1 tablet twice daily. The physical therapy procedure included ultrasound bath with hydrocortisone for the lumbar spine daily; the course consisted of 14 sessions.

Results

The indicators of pain level according to NRS scale were the same before the beginning of the treatment: 1a – 7.70±0.47, 1b – 7.50±0,42; 2a – 8.05±0.69, 2b – 8.2±0.29; ($p>0.1$). The clinical complex of symptoms in the patients of both groups, along with the pain syndrome, included sensitive disorders in the corresponding dermatome, mostly in the form of a prolapse, mild motor disorders in the form of flaccid paresis of foot and toe extensor on the affected side, loss of Achilles tendon reflex on the same side, and restriction of the spine mobility in the lumbar part (Table 1).

Despite the fundamental similarity of neurological status, differences in the groups were observed in patients: firstly, the pain syndrome in the patients of the second group was mostly "multifaceted": radicular (along the root compressed by disc hernia), pseudofaceted (due to nociceptive impulse irrigation from the field of surgical intervention) and multiradical (in patients with reduced cauda equina syndrome); secondly, motor disorders in the leg on the side of the lesion and sensory disorders beyond the dermatome L5-S1 prevailed in the patients of the second group.

In general, the clinical pattern of postoperative recurrent intervertebral disc hernias at the lumbar level was characterized by a more significant neurologic deficit compared with the primary disc hernias. According to the MRI (CT) data, all of the patients had signs of degenerative dystrophic changes in the spinal elements.

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Table 1 Main neurological symptoms among the researched category of patient

Group of patients / Main symptoms	Group 1 (n=35), abs/%				Group 1 (n=25), abs/%			
	1a, (n=17)		1b (n=18)		2a (n=12)		2b (n=13)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
1. Pain:	17/100	5/29*	18/100	2/11 Δ	12/100	6/50 Δ	13/100	3/23 Δ
- local	7/41	1/6		- Δ	8/67	3/25	3/23*	2/15*
- irradiated	7,70±0,47	4,45±0,26*	7,50±0,42	2,05±0,34 Δ	8,05±0,69	5,72±0,55	8,2±0,29	4,04±0,51 Δ*
- NRS (points)								
2. Sensory disorders								
- anaesthesia (hypoesthesia)	12/71	8/67*	10/56	4/22 Δ	10/83	7/58	10/77	6/46
- hyperesthesia	1/6	-	1/6	-	-		1/8	-
- paraesthesia	3/18	1/6	2/11	-	7/58	4/33	5/38	3/23
- hyperpathia	1/6	-	2/11	-	2/17	-	1/8	1/8
4. Motor disorders:								
- paresis (1-2 points)	3/24	1/6	4/22	1/6	5/42	3/25	5/38	2/15
- paresis (3-4 points)	8/47	3/18	9/50	1/6 Δ	5/42	3/25	6/46	3/23
7. Reflex disorders:	6/35	2/12	5/28	1/6	4/20	3/25	5/38	3/23
- absence of reflexes	10/59	7/41	13/72	3/17 Δ	8/62	4/33	7/54	4/31
- hyporeflexia	1/6	-	-	-	-	-	1/8	-
- hyperreflexia								
9. Lasègue's sign	7/41	1/6	5/28	1/6	5/42	2/17	5/38	2/1
- at 45° or less	10/59	6/35	13/72	3/17	7/58	5/42	7/54	4/31
- at more than 45°								

*significant difference (p<0,05) between the corresponding subgroups of the main groups
Δ significant difference (p<0,05) between the subgroup "a" and "b" within the main groups

Single and multiple protrusions, disc hernias mostly at L4-L5, L5-S1 levels were observed. The size of protrusions and hernias varied from 3 mm to 8 mm (mean 5.5 mm). Schmorl's nodes were found in 34 patients. The signs of the dural sac compression were observed in 48 cases, impaired liquor dynamics – in 25 cases (42%), discitis – in 4 patients (7%), spondylitis – in 2 patients (3%), epidural fibrosis – in 20 patients of the second group. 9 patients (15%) were diagnosed with hemangiomas in the lower thoracic and lumbar vertebrae.

A significant decrease in the nerve impulse conductivity rate along the sensory and motor fibers, a decrease in the amplitude of the M-response and F-wave, an increase in the F-wave latency were observed in patients of both the first and second groups compared with the control group ($p<0.05$). However, a reliable intergroup difference between the ENMG parameters studied was not identified (Table 2).

At the same time, the amplitude of the M-response, both proximal and distal, was significantly lower in patients with paresis compared with patients without paresis; moreover, an increase in the latent period of the F wave, a decrease in its amplitude, and polyphasia were observed. In patients without paresis, the distal and proximal amplitude indicators did not differ significantly from the standard indicators, the amplitude of F-wave was slightly reduced, and the latent period was slightly increased, there were few cases of polyphasia. The amplitude of distal M-responses was reduced compared with standard indicators by 54.7% in patients with paresis and by 24.2% in patients without paresis, which suggested axonal damage of the peripheral nerves. A decrease in SPI along the motor fibers was 59.1% in patients with paresis and 69.8% in the group without paresis, it was decreased by 32.4% and 23.1%, respectively, compared with the control group. There was no significant difference of SPI in the sensory fibers between the groups of patients with and without paresis, and the SPI was reduced by 52.8% and 53.4%, respectively, compared with the standard indicators. The amplitude of the F wave in the presence of paresis was reduced by 14.5%, and it decreased by 8.7% in the group without paresis, which confirmed the axonal lesion of the spinal nerves. The latency of the F wave was increased by 27.1% in patients with paresis and by 12.1% in patients without paresis ($p<0.05$), which suggested the demyelinating type of lesion of the spinal nerves. In all of the examined subgroups, a significant increase in the time of root delay was found before treatment, compared with the control group, which decreased after the neuromidin test (positive test) but did not reach the standard values (Table 1 & Table 3).

Thus, according to the ENMG and RDAM data, axonal demyelinating lesion of proximal and distal fibers of the lumbosacral plexus with a prevalence of demyelination in the proximal parts (spinal root) and axonal damage in the distal parts were found in patients with LSR. Comparison of clinical and electrophysiological data showed that in case of lumbosacral radiculopathy, sensory fibers are always involved in the process, which clinically manifests as radiculitis of the radicular neural type. Motor fibers are also involved in the process, clinical manifestations in the form of peripheral paresis occur in 75% of cases. The damage of sensory fibers seems to be mostly demyelinating, the damage of motor fibers is axonopathy.

The study according to the program planned was completed by all of the patients, and the compliance coefficient was 100%. The treatment results were analyzed taking into consideration the dynamics of neurological disorders and the data of neurophysiological studies.

Significant improvement in the indicators in patients of all subgroups as compared to baseline values was observed during dynamic evaluation of the radicular pain syndrome severity (according to the NRS scale) (Table 1). These changes were most pronounced in patients who underwent Cutlen epidural blockade and took Neuromidin during treatment. The same trend of changes was found when assessing the indicators of the motor activity and the severity of sensory disorders over time (Table 2). It should be noted that the pain syndrome in patients with FBSS was rather resistant to treatment: by the end of the 3-week therapy, the pain intensity according to the NRS scale in this group of patients was significantly higher than in patients diagnosed with LSR exacerbation.

Table 2 Changes of the ENMG parameters among the researched groups of patients

groups / nerves	1a		1b		2a		2b		control
	before treatment	after treatment	before treatment	after treatment	before treatment	after treatment	before treatment	after treatment	
	NCV in sensory fibres, m/s								
n. peroneus	22,4±5,7*	42,2±4,6* μ	23,8±3,7*	59,1±4,1 Δ μ	20,2±5,1*	33,7±4,4*	19,1±4,2*	46,5±45 Δ	56,3±4,7
n. tibialis	23,6±4,9*	41,1±5,3* μ	24,7±4,1*	58,2±3,8 Δ μ	19,6±4,2*	34,5±4,7*	18,8±3,9*	44,8±4,1 Δ	55,8±5,4
	NCV in motor fibres, m/s								
n. peroneus	27,7±4,7*	37,6±4,5 μ	28,1±3,5*	40,2±3,1 Δ μ	26,8±5,5*	31,1±3,9	28,6±5,2*	35,2±4,1	42,9±5,6
n. tibialis	28,5±5,1*	37,4±3,8 μ	29,6±4,9*	41,3±4,4 Δ μ	27,2±4,8*	30,2±3,8	29,3±5,1*	36,9±3,7	44,3±6,1
	Distal amplitude of the M-response, mV								
n. peroneus	2,1±0,7*	3,3±0,5	2,2±0,3*	4,8±0,4 Δ μ	2,5±0,3*	3,1±0,2	2,1±0,7*	3,5±0,6	4,3±0,5
n. tibialis	3,4±0,6*	4,6±0,8 μ	3,3±0,5*	5,9±0,7 Δ μ	3,4±-0,5*	3,8±0,3	3,5±0,3*	4,2±0,4	5,2±0,8
	Latent period of the F-wave, m/s								
n. peroneus	56,1±4,3*	50,8±4,2 μ	57,2±4,8*	48,2±4,5 Δ μ	56,3±4,3*	51,7±4,5	59,5±4,5*	53,7±4,1	47,6±3,9
n. tibialis	55,3±4,5*	48,9±4,3 μ	54,1±4,2*	47,4±3,9 Δ μ	55,4±4,7*	52,5±4,1	54,8±4,9*	51,6±3,8	46,5±4,7
	Amplitude of the F-wave, mV								
n. peroneus	260,5±23,4*	288,6±20,3	273,8±16,6*	307,8±24,3 Δ μ	259,4±19,3*	277,2±21,5	278,1±19,5*	289,2±19,8	308,7±15,2
n. tibialis	283,4±20,2*	293,5±18,4 μ	257±24,5*	310,6±17,1 Δμ	270,3±22,6*	284,4±22,1	268,3±20,8*	291,3±23,6	312,1±17,7

*significant difference (P<0,05) versus the control group

Δ significant difference (P<0,05) between subgroups "a" and "b" within the main groups

μ –significant difference (P<0,05) between corresponding subgroups of the main groups

Table 3 Changes of *the root delay indicators* among the researched groups of patients.

Root delay indicators / Groups	1a, ms		1b, ms		2a, ms		2b, ms	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Before "Neuromidin" (control group – 3,5±0,3 ms)	6,5±0,6*	4,8±0,4*	6,6±0,7 *	3,6±0,3 Δ μ	6,4±0,5*Δ	5,3±0,7	6,5±0,7 *	4,3±0,5* Δ
After "Neuromidin"	4,5±0,3*	4,0±0,4	4,3±0,5 μ	2,8±0,5 Δ μ	5,0±0,6*	4,7±0,5	4,7±0,8*	3,9±0,7 Δ

*significant difference (P<0,05) versus the control group

Δ significant difference (P<0,05) between subgroups "a" and "b" within the main groups

μ – significant difference (P<0,05) between corresponding subgroups of the main groups

According to the data of the clinical studies, radicular pain syndrome and sensitivity disorders decreased to a greater extent in patients of the group I, in particular in subgroup 1b. The same trend, but less pronounced, was observed in the assessment of motor disorders. A more significant increase in the impulse conductivity rate along the sensory (by 1.6 times on average) and motor fiber (by 1.3 times on average) was observed after the use of Neuromidin; the amplitude of voluntary muscle contraction also increased – by 1.5 times on average. All of this suggested a significant improvement in nerve conductivity along the affected roots and descending nerves when Neuromidin was included in the complex of conservative treatment.

The normalization of the root delay time was observed after 3 weeks of conservative treatment only in the subgroup Ib. Significant improvement in the nerve impulse conductivity was observed in other subgroups, but significant differences from the norm were still present (Figure 1). It should be noted that the parameters obtained when studying the root delay time correlated with other results of the treatment in all subgroups of patients ($p<0.05$). Therefore, a significant increase in the time of root delay found before the beginning of the treatment, was an important prognostic marker for the insufficient effectiveness of conservative therapy. Moreover, this correlation was observed not only with electrophysiological parameters, but also

with subjective and objective clinical manifestations of the disease – severity of weakness in the extremities, intensity of pain syndrome, paresthesia, hyperpathy, etc. (Table 3). Side effects (hypersalivation, bradycardia, dizziness, nausea, vomiting) were not observed during administration of Neuromidin.

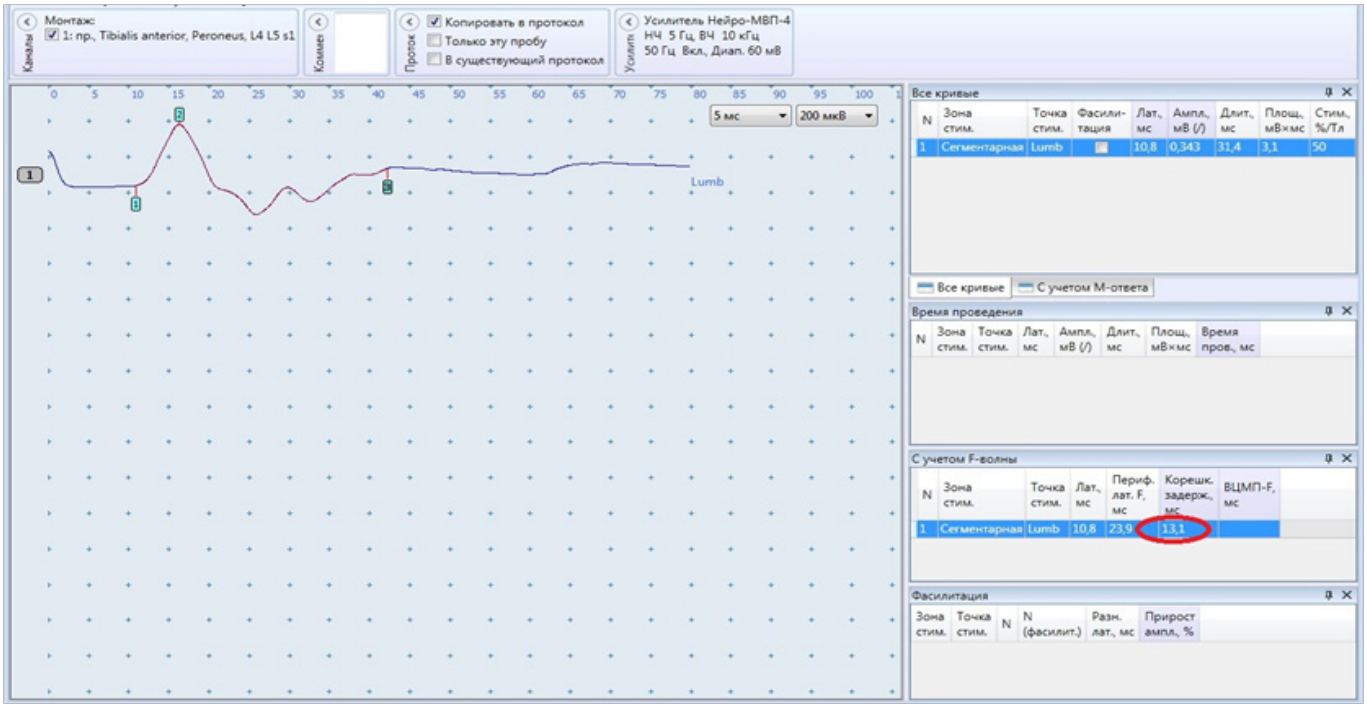

Figure 1 The root delay time, 13,1ms.

Discussion

The modern interpretation of pain phenomena in clinical practice is far from perfect, and sometimes is not scientific and, therefore, their differentiated therapy either is not carried out, or is formed empirically. In particular, the trend to emphasize the neuropathic component in the chronic pain syndrome of any origin contradicts the obvious scientific data: firstly, pain cannot exist outside the nervous system and, therefore, the latter is somehow involved in the pathological process; secondly, any chronic pain syndrome is a signature of stable pathological associations formed at different levels of the central nervous system within the framework of maladaptive neuroplasticity; thirdly, despite the globalization of the "pain cloud" in case of chronic course of the disease, it is associated with the source of pain.

The study of individual nosological forms accompanying pain syndrome enables to solve not only topical clinical problems, but also fundamental questions. From this point of view, LSR is the optimal model because at present their diagnostics and the range of the treatment methods applied are well developed, which allows monitoring not only pain traffic but also the efficacy of its modulation. In particular, it is known that the vast majority of patients with primary LSR recover within 3 months. This circumstance argues for the predominantly "inflammatory phase" of root compression and explains the high efficacy of NSAIDs or local administration of glucocorticoids.[10–12] Moreover, epidural GC administration ensures their high local concentration and minimizes the systemic side effects. The prognostic factors determining the favorable outcome of LSR conservative treatment are: absence of the spinal canal stenosis, good effect of epidural GC administration, positive Neuromidin test when assessing RDI, and patient's high motivation to participate in motor rehabilitation.

If the subsequent exacerbation lasts more than 6 months, the severity of clinical manifestations of the disease remains at the same level for the next 2-5 years; especially in case of foraminal root compression by abnormally modified facet joints, when the effectiveness of conservative therapy is relatively low, and the intensity of the pain syndrome directly depends on the degree of root compression. Therefore, in these cases, especially in case of increasing neurological disorders, the decision about surgical treatment should be made. Furthermore, as our studies have shown, the effectiveness of root compression diagnostics increases when summarizing the data of neuroimaging and neurophysiological examinations (ENMG and RDAM with Neuromidin test). The study of root delay by magnetic stimulation enables the increase of the percentage of detection of acute root pathology and true exacerbations of radiculopathy and, therefore, it is a necessary supplement to the electrophysiological study of patients with LSR. This method enables the identification of compression of the spinal roots (in case of acute radiculopathy–in 65.8% of cases, in case of exacerbation–in 59.3%), manifested only or mostly by demyelination.

It is well known that the effectiveness of standard discectomy and microdiscectomy is the same in case of compression-ischemic radiculopathy of the lumbosacral roots.[13] A comparative analysis of the efficacy of standard discectomy and conservative treatment in patients with persistent symptoms of LSR after 6-week conservative therapy showed that the results of surgical and conservative treatment were similar in terms of the main indicators of pain and incapacitation. However, the FBSS is often observed in the group of patients who underwent surgery (1-48% of cases), which is associated with recurrent disc hernia, cicatrical adhesion, epiduritis, arachnoiditis, formation of cerebrospinal cysts in the surgery area, occurrence or increase of

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instability in the operated "vertebral segment", which, according to our data, often aggravates neurological symptoms and requires the combined use of NSAIDs, vitamins of group B, physiotherapeutic treatment, remedial exercise, and use of Neuromidin for the restoration of root and neural conductivity.

Conclusion

To ensure the effective treatment of chronic back pain, it is necessary to strive to localize the source of painful impulses as accurately as possible (e.g. compressed root in case of LSR), and to consider the possible mechanisms of compression radiculopathy exacerbation (repeated spinal root trauma or postoperative epidural fibrosis). The exacerbation of LSR may be manifested by varying degrees of the spinal root conductivity disorders, signs of active denervation in muscles innervated by this root depending on the type of neural lesion. However, we should not ignore the fact that prolonged nociceptive afferentation causes neuroplastic changes resulting in peripheral and central sensitization. This partly explains the fact that surgical intervention did not become a panacea in the treatment of neurological complications of lumbar osteochondrosis. Multidisciplinary approach and combined multipurpose treatment can provide an effective influence on the course of LSR. In particular, the use of Neuromidin in the combined treatment of patients with dorsopathies can effectively influence the development of denervation syndrome and reduce the severity of pain syndrome.

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None.

Conflicts of interest

The author declares that there is no conflicts of interest.

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