

# Clinical Evaluation of Treatment Plans for some of Foot Deformity forms in Patients with Neurological Diseases

Vladimir A. Frolov

***Abstract---** The rationale for the present work is in the fact that diagnostics and rehabilitation treatment for nervous and musculoskeletal systems diseases remain acute among issues of theoretical and clinical medicine. Hence, the present article describes the prospects of diagnostics and rehabilitation treatment improvement in patients with feet deformities associated with central and peripheral nervous system diseases. The study was based on clinical neurophysiological analysis.*

*The main study methods were plantography examination and clinical-neurological and psychological testing performed for 128 patients (75 females and 53 males) with clinical picture of neurological disorders in feet. This approach allowed the authors to evaluate the recommendations on non-specific orthopedic therapy for different neurogenic feet deformities and to optimize the complex of therapeutic measures based on the results of clinical questionnaire. This approach contributes to the improvement of primarily diagnostics of feet functional disorders associated with nervous system diseases.*

*In the present article the importance of peripheral neuromotor functional and peripheral maintaining factors insufficiency for diagnostics, disease prognosis and improvement of foot dystonia treatment efficiency is shown. The materials presented in the article are of practical value to neurologists, chiropractics, osteopathic physicians, podiatric specialists and recreation therapists that rely on complex approach in diagnostics and treatment of older patients.*

***Keywords---** Afferent-efferent Relations, Developed Guidelines, Feet Deformities, Complex Questionnaire, Plantography, Complex Approach, Neurologic Disorders.*

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## I. INTRODUCTION

According to the data of Ministry of Healthcare and Social Development of the Russian Federation, the rate of nervous and musculoskeletal systems diseases in the RF tend to increase constantly. It should be noted that a number of nervous system diseases (including peripheral nervous system) lead to the development of musculoskeletal system and connective tissue pathologies, wherein feet deformities are quite widespread [1]. Feet functional disorders associated with neurological diseases (neurogenic feet dysfunction) lead to the development of neurogenic feet deformities.

Despite clinical similarity, foot deformity can be caused by impairments at different levels of central and peripheral nervous system [1]. The present study revealed more than 100 neurological symptoms and syndromes associated with feet.

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Published literature contains descriptions of feet deformities developed after strokes [2], [3], under neuromuscular diseases [4], [5], [6] and at congenital anomalies of nervous system development [7], [8].

It is known that static and dynamic foot deformity, in its turn, can provoke the development of lower extremity compression ischemic neuropathy [9], as well as manifestation of myofascial pain syndrome [10].

A number of studies were dedicated to foot dystonia (FD) as a complication associated with Parkinson's disease pharmacotherapy [11], [12] or multifocal dystonia [13], [14], [15].

It is suggested that pathogenesis of focal forms of dystonia, in particular, foot dystonia, can include neural dysfunction at the level of basal ganglia and thalamus that could lead to impairments of cortex afference [16]. However, the role of peripheral and segmental parts of nervous system in the pathogenesis of FD and their ratio to central afferent mechanisms remain unclear. There is still no clear understanding on the causes of central afferent system functional change as well as on their relation to the main mechanisms of dystonia manifestations.

Clinical picture of neurogenic feet deformities is understudied and require further investigations.

The reasons of neurologic diseases influence on the development of certain forms of foot deformity are not clear. Foot deformity influence on the development of neurological diseases (tunnel neuropathies, myofascial pain syndromes) is also understudied. There were no studies on the alterations, associated with unilateral lesions, in the contralateral ("relatively healthy") foot.

Literature review showed that there are few publications on clinical-neurophysiological analysis of feet deformities associated with central and peripheral nervous system diseases and on their treatment.

In spite of the fact that this pathology is widespread and worsens quality of life of patients with feet dysfunction and deformity associated with central and peripheral nervous system disorders, there is lack of research data on the most efficient methods of rehabilitation treatment in this category of patients.

### ***The Study Tasks***

1. To investigate clinical manifestations and peculiarities of feet deformities associated with neurological syndromes: central and peripheral paresis, foot dystonia, neurogenic plantalgia and psychogenic impairments.
2. To evaluate alterations, associated with unilateral lesions, in the contralateral ("relatively healthy") feet.
3. To assess functional status of corticospinal pathways and afferent systems associated with feet neurological diseases.
4. To assess functional status of peripheral neuromotor apparatus, associated with feet functional disorders, that provides low extremities muscle functioning.
5. To identify etiological factors of foot tunnel syndromes development.
6. To develop complex etiological factors research based guidelines for improvement of diagnostics and rehabilitation treatment for neurogenic feet deformities.

The authors conducted a complex study on feet deformities associated with nervous system diseases. Orthopedic peculiarities and plantographic characteristics of the deformed and relatively healthy (contralateral) foot were investigated. Afferent-efferent relations and the status of neuromuscular apparatus associated with neurogenic feet deformities were evaluated. The obtained research data contributed to the understanding of clinical picture of foot dystonia associated with Parkinson's disease, multifocal dystonia and foot tunnel syndrome. Comparative analysis of orthoses for correction of neurogenic feet deformities was performed. Research-based guidelines were developed for non-pharmaceutical methods of treatment for neurological feet disorders.

Complex clinical questionnaire was developed for improvement of primary diagnostics of feet functional disorders associated with nervous system diseases. The obtained neurophysiological examination data and specific patterns of clinical manifestations, associated with feet neurological disorders, can be used for verification of clinical diagnosis considering etiology and pathogenesis of the disease. The importance of peripheral neuromotor apparatus functional insufficiency and peripheral maintaining factors identification for diagnostics, prognosis and improvement of foot dystonia treatment efficiency was shown. The developed recommendations on non-specific orthopedic therapy for different neurogenic feet deformities will allow the clinicians to optimize the complex of treatment measures.

## **II. MATERIALS AND METHODS**

The study material included 128 patients (75 females and 53 males) with clinical picture of neurological feet disorders. Average patients age was  $45 \pm 13$  years old.

### ***The Study Entry Criteria***

- 1) Diagnosed neurologic disease that leads to functional feet disorder;
- 2) Complex of symptoms of upper/lower motoneuron or clinical manifestation of foot dystonia;
- 3) Syndrome of neurogenic plantalgia.

### ***Main Clinical Groups (Table 1)***

1. 30 patients with central foot paresis, wherein 16 patients had complications of acute cerebrovascular event (ACVE), 11 patients had thoracic extramedullary spinal tumor (ST), 3 patients had posttraumatic thoracic myelopathy (TM).
2. 32 patients with peripheral foot paresis, wherein 15 patients had neuropathy of fibular nerve, 4 patients had distal myodystrophy, 7 patients had inherited sensorimotor neuropathy type I (ISMN), 6 patients had complications of poliomyelitis.
3. 30 patients with foot dystonia, wherein 16 patients had Parkinson disease (PD) and 14 patients had multifocal dystonia (MFD).
4. 36 patients with neurogenic plantalgia syndrome, wherein 21 patients had tarsal tunnel syndrome (TTS) and 15 patients had Morton's neuralgia.

Table 1: Patient Groups

Group 1 Central foot paresis (30 people)	16 patients had complications of acute cerebrovascular event (ACVE), 11 patients had thoracic extramedullary spinal tumor (ST), 3 patients had posttraumatic thoracic myelopathy (TM).
Group 2 Peripheral foot paresis (32 people)	15 patients had neuropathy of fibular nerve, 4 patients had distal myodystrophy, 7 patients had inherited sensorimotor neuropathy type I (ISMN), 6 patients had complications of poliomyelitis.
Group 3 Foot dystonia (30 people)	16 patients had Parkinson disease (PD) and 14 patients had multifocal dystonia (MFD)
Group 4 Plantalgia (36 people)	21 patients had tarsal tunnel syndrome (TTS) and 15 patients had Morton's neuralgia
Healthy participants (40 people)	40 healthy participants, similar by sex and age to the patients in the main group

Control group included 40 healthy volunteers that were similar by sex and age to patients in the main group.

Control group entry criteria:

1. No neurological diseases;
2. No complaints on feet pain;
3. No evident deformities of feet and toes during visual examination.

All the patients underwent the following examination: primary examination with the use of special questionnaire for patients with neurological disorders in foot (the scale of dystonia severity and the scale of disability status (due to lower extremity dystonia) were used for patients with foot dystonia), evaluation of patients health status by functional scale of lower extremity (before and after the treatment) and quality of life questionnaire. In 100% of cases, plantographic study and psychological test (State Trait Anxiety Inventory (STAI) and Beck Depression Inventory (BDI)) were performed. ST, TM and ACVE diagnoses were verified by magnetic resonance imaging (MRI) in all the patients. All the patients with foot dystonia, associated with Parkinson disease, underwent neuroimaging study that revealed no focal lesions of brain. Electrophysiological examination included transcranial magnetic stimulation (TMS) and somatosensory evoked potentials study (SSEP). The patients with foot dystonia, peripheral foot paresis and neurogenic plantalgia underwent electroneuromyography (ENMG).

Healthy participants were examined once by all the methods. Dynamic clinical-psychological examination was performed at the study entry, at different stages of treatment, when minimal response to complex therapy was registered and after correction of walk stereotype and orthopedic impairments.

### III. CLINICAL STUDY

Detailed description and analysis of feet functional disorders were performed according to the developed "Complex clinical questionnaire for patients with feet deformities". The questionnaire contained a set of questions for evaluation of neurologic and orthopedic status peculiarities focused on peripheral neuromotor apparatus of the "deformed" and "healthy" foot, character of provoking and maintaining peripheral factors and typical early symptoms and stages of the disease development.

Clinical-neurological examination of lower extremities evaluated lower leg dystrophy, active and passive movements, muscle force (including lower Barre test) and muscle tonus (before and after walk) in proximal and

distal segments of lower extremities. Patellar, Achilles and plantar reflexes were analyzed, as well as the presence or lack of foot pathology signs and foot clonus. The patients were checked for sensory impairments (superficial and deep sensitivity) and presence of Tinel's symptom, Mulder's sign, Grifk sign and Gaenslen's sign.

During the study of local orthopedic status, the authors evaluated the difference in lower extremities length (absolute or relative due to pelvic obliquity (functional sacroiliac block)), lateralized myofascial and muscular tonic syndromes, and scoliosis. Hindfoot and forefoot positions (under loaded and non-loaded condition), foot type (normal, hollow, flat), presence of contractures and toe deformities and hypermobility of foot joints were identified. Feet examination was performed in patients prone position with measurement of movements amplitude in subtalar joint (by Donatelli's test).

Evaluation of dystonia severity was performed by the scale of dystonia severity [17], scale of disability status associated with dystonia [17] and scale of disability status associated with dystonia of lower extremity (percent of normal activity) [18]. The scales of dystonia severity and disability status associated with dystonia allowed the authors to assess the severity of foot dystonia in patients with Parkinson's disease and multifocal dystonia (Group 3) and were used in this group for therapy efficiency evaluation.

Foot pain syndrome scoring was performed by visual analogue scale (VAS) (from 0 – “no pain” to 10 – “unbearable pain”). The scoring results were used for analysis of pain syndrome intensity before and after the treatment.

Quality of life was evaluated by a special questionnaire. The obtained data was considered during evaluation of therapy efficiency.

Lower Extremity Functional Scale (LEFS) was used for assessment of rehabilitation treatment efficiency.

Psychological testing was performed for verification of presence, evaluation of degree and identification of changes in anxiety and depression by the STAI and BDI.

Plantographic study included: 1) visual evaluation of foot arches status, degree of their deformity and shape (straight, underprone, overprone); 2) visual identification of foot overload zones and distribution of pressure in feet; 3) assessment of foot arch and heel bone position during functional tests (“sit test”, “double legs stand”, “single leg stand”, etc.); 4) Plantographic imaging of the disease dynamics and the process of selection and or crafting of individual in-shoe orthoses.

#### **IV. ELECTROPHYSIOLOGICAL METHODS**

##### ***Transcranial Magnetic Stimulation (TMS)***

TMS is a non-invasive method of investigation for evaluation of nerve conduction velocity by means of analysis of central motor conduction time (CMCT) and excitability of interneurons and motoneurons during the study of motor response (MR) thresholds in cortex. The method allows the researchers to evaluate functional status of corticospinal pathways.

### ***Somatosensory Evoked Potential Study (SSEP)***

During SSEP study, the analysis of afferent electric impulses conduction via sensory pathways of posterior column of spinal cord, their associate structures and spinothalamic tract was performed, which allowed the authors to examine somatosensory cortex activation (Left hemisphere peaks N30 and P40 for low extremities studies).

### ***Stimulation Electroneuromyography of Lower Extremity Nerves***

The method is based on identification of excitement conduction velocity in motor and sensory fibers of fibular and tibial nerves of both lower extremities in response to nerve electrical stimulation.

Statistical analysis of the obtained data was performed by the software package STATISTICA 8.0 for Windows. Discriminate and cluster analyses were performed for distribution of patients into groups. Average quantitative parameters values were calculated by means of methods of descriptive statistics. Attributes frequency was identified by frequency tables. Shapiro-Wilk W test was used for analysis of qualitative parameters distribution compliance with the law of normal distribution. Non-parametric distribution analysis was performed by Mann-Whitney-Wilcoxon test, applied to qualitative and quantitative parameters. Spearman correlation analysis was performed between all the studied parameters among all the patients and within each group.

## **V. RESULTS AND DISCUSSION**

### ***Material Clinical Characteristics***

Specific clinical symptoms and foot deformities representation in patients with central and peripheral foot paresis, foot dystonia and neurogenic plantalgia were analyzed.

Positions of “deformed” (160) and “relatively healthy” (contralateral) feet were evaluated in 128 patients with different neurologic diseases as compared to healthy participants (Table 1). The data, obtained for all the patients and healthy participants during visual examination of feet position, is presented in Table 2. Normal position in “deformed” feet was not observed. Paralytic (equinus, equinopolovarus, equinovarus, equinovalgus) deformities were observed in “deformed” feet in 40% of cases, in particular, equinovarus deformity was diagnosed significantly more often ( $p < 0.05$ ) in “deformed” feet (25%) as compared to “relatively” healthy feet and HP. Paralytic feet posture developed in all the cases associated with disbalance of muscle groups that support the feet in normal physiological position and was determined by paresis distribution and muscle dystonia.

Table 2: The number of feet («deformed» and «relatively healthy») unilateral and bilateral disorders rate in the studied groups

	«Deformed» feet (n)	«Relatively healthy» feet (n)	Unilateral disorders (N)	Bilateral disorders (N)
Group 1	44	16	16 (53.3%)	14 (46.7%)
Group 2	42	22	22 (68.8%)	10 (31.2%)
Group 3	32	28	28 (93.3%)	2 (6.7%)
Group 4	42	30	30 (83.3%)	6 (16.7%)
TOTAL:	160	96	104 (56.5%)	40 (43.5%)

N – number of patients, n – number of feet

Among “relatively healthy” feet, varus and vagus positions were registered equally in 47.9% cases (in combination with hyper pronation in 44.7%). Paralytic feet deformities and hollow feet were not observed in healthy participants; 50% of the examined participants had normal feet position in subtalar joint – moderate valgus. In healthy participants in the majority of cases forefoot to hindfoot was varus oriented, in fewer cases it was valgus or neutral oriented.

Table 2: Neurogenic feet deformities in the studied patients

Deformity	«Deformed» feet (124)		«Relatively healthy» feet (96)		Healthy participants (80)	
	Absolute amount	%	Absolute amount	%	Absolute amount	%
Norm ♦*	—	—	7	7.29%	80	50%
Varus	52	32.5%	46	47.9%	40	25%
Valgus	12	7.5%	35	36.4%	40	25%
Hyperpronation	30	18.75%	8	8.33%	—	—
Hollow feet	2	1.25%	—	—	—	—
Equinus	4	2.5 %	—	—	—	—
Equinovarus • ♦	40	25%	—	—	—	—
Equinoplovarus foot	18	11.25%	—	—	—	—
Equinovalgus	2	1.25%	—	—	—	—
• – “deformed” feet is significantly different from “relatively healthy” feet at $p < 0.05$						
♦ – “deformed” feet is significantly different from feet of healthy participants at $p < 0.05$						
* – “relatively healthy” is significantly different from feet of healthy participants at $p < 0.05$						

Thus, at the stage of neuroorthopedic examination the authors identified differences between the patients feet (both “deformed” and “relatively healthy”) and healthy participants feet.

The most typical neurogenic foot deformities associated with upper motoneuron lesion was equinovarus (45.5%) and varus (31.8%) feet positions combined with pyramid signs muscle hypertonia (hypotonia).

The analysis of the obtained results and comparison of pathologic feet positions associated with cerebral and spinal level spastic paresis of lower extremities showed that among the most typical deformities in the subgroups there were different walk patters (spastic-paretic and hemiparetic), paresis distribution (muscle force decrease in distal segments of lower extremities was more often registered in patients with cerebral level lesions), prevalence of synkinesis associated with cerebral lesions and prevalence of clonuses and adduction reflexes associated with spinal lesions. Cerebral lesions primarily caused (86.7%) ankle joint contractures correlated and correlated with high spasticity. Spinal level lesions were clinically characterized by foot function failure, valgus deformities and higher rate of disability status development.

Spastic foot drop was observed at both cerebral and spinal level lesions.

Peripheral paralysis (Group 2) was associated with reduction of sensitivity and reflexes, deficit of muscle force and their tonus, hypotrophy and atrophy, limitation or absence of voluntary and reflectory movements. All the disorders were characterized by the reduction of muscle force, which simplified evaluation of patients kinetic status. Development of neurogenic foot deformities was assessed by the degree of force disbalance of antagonist muscles. Different variations of peripheral equinovagus were registered in the group of patients with peripheral foot paresis. Formation of equinovarus or equinoplovarus deformity was associated only with the duration of the disease.

Foot dystonia (Group 3) included two main elements: plantar toe flexion and/or foot inversion (patients stepped on lateral border of foot) that were noticed only during walking. According to the classification by C. Pacchetti [19], the authors divided foot dystonia in patients into simple and complicated types.

Simple type was characterized by foot inversion or hallux extension (66.6%), and complicated type was characterized by foot inversion and toe plantar flexion or plantar flexion combined with toe flexion (33.3%) (Figure 1).

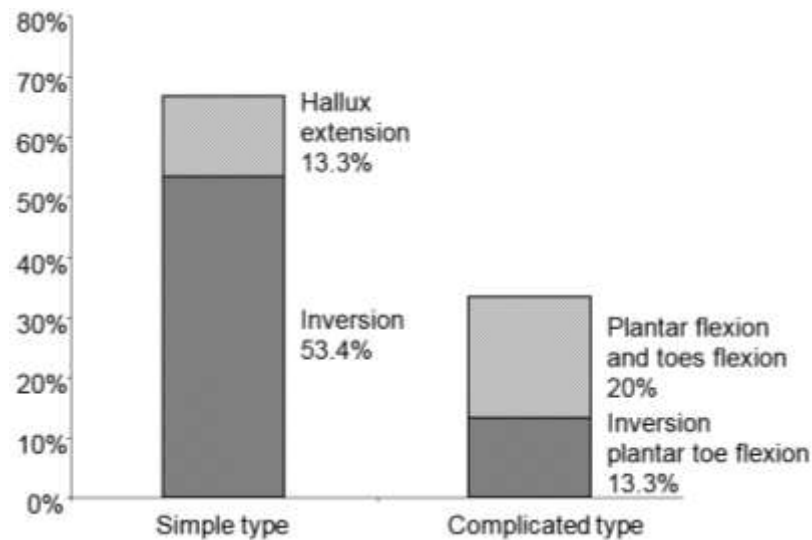


Figure 1: Foot Dystonia Type

Severity of dystonia score, assessed by Burke scale (0–120 points), was equal to  $7.5 \pm 14.8$  (1–15) points, which corresponded to moderate severity of dystonia. Severity of dystonia, assessed by the Fahn scale of disability status (0–30 points), was equal to  $4.5 \pm 2.3$  points, and also corresponded to moderate severity of dystonia. The share of remaining normal activity in patients with foot dystonia was within the range of 1 – 79% (Table 3).

Table 3: Evaluation of dystonia severity (mean values)

<i>Evaluated parameter</i>	<i>DF</i>	<i>HP</i>
Burke scale (score)	* $7.5 \pm 14.8$	0
Fahn scale of disability status (score)	* $4.5 \pm 2.3$	0
Percent of remaining normal activity (%)	* $62.5 \pm 18.6$	100

\* – significant difference as compared to healthy participants,  $p < 0.05$ .

The majority of patients with foot dystonia (87.5%) had varus feet position.

Foot dystonia associated with MFD was characterized by ( $p < 0.05$ ) sensory signs at the disease onset, other hyperkinases manifested at the disease onset, higher Burke scale score, corrective actions and prevalence of complicated forms.

The other type of foot dystonia developed during Parkinson’s disease. Simple dystonia types prevailed. Foot dystonia was one of the first manifestations only in one patient. More often dystonia developed as a variant of drug-



induced dyskinesia in response to long-term Levodopa therapy (“off” dystonia, in particular, “early morning dystonia” – 53.3% and “late night dystonia” – 46.7%). “Early morning dystonia” manifested in the morning when the patient got up from bed, before first morning Levodopa dose.

The analysis of etiologic factors of low extremity compression neuropathy (LECN) development in different localizations (TTS, Morton’s syndrome) showed that static and dynamic overload (long-time standing, high-heeled shoes) and syndrome of benign hypermobility syndrome prevailed. Besides, one third of the patients had body weight gained, which was an additional overloading factor. Overload was mostly significant in development of foot and ankle compression neuropathy.

Static foot deformities were observed in 66.7% of patients with neurogenic plantalgia. Arthritis and arthroses of lower extremities joints were observed in 50% of patients with LECM. Ankle joint arthroses were associated with compression lesions of tibial nerve in tarsal canal. Arthroses of metatarsophalangeal articulations led to plantar nerves neuropathy development.

In group with plantalgia valgus (hypertonus) feet position significantly prevailed (85.7%) in sublar joint.

Other orthopedic peculiarities in the studied groups were alterations of low extremities length, primarily due to relative extension of equinovarus or equinus foot, hyperpronation, sometimes due to formation of oppositely directed deformities (varus and valgus) and low extremity shortening as a result of poliomyelitis. Change of deformed extremity length led to non-fixed pelvic obliquity, development of static S-shaped or C-shaped scoliosis, pain syndrome at the lumbar level. Toe deformities were associated with I toe valgus deformity, clasp or claw-shaped toe deformity. Fixed or dynamic contractures were observed in all the groups at different rates (12.5% – 86.7%).

The analysis of quality of life (QoL) showed that low extremity functional scale (LEFS) and visual analogue scale (VAS) score was significantly different ( $p<0.05$ ) from the score of HP. QoL in Groups 1 and 3 was similar and significantly different ( $p<0.05$ ) from other Groups and HP. It correlated with the degree of paresis, presence of contractures in ankle joints and inversely correlates with the muscle tonus. Significant ( $p<0.05$ ) inverse correlation between LEFS score and patients age, degree of paresis, muscle tonus impairment, difference in legs length, Tunnel symptom, hyperpronation and transverse platypodia on the deformed foot. Probably, it is explained by more favorable biomechanical characteristics of valgus (hyperpronated) foot as compared to other deformities. Maximal pain syndrome score by VAS scale was observed in the group with plantalgia. VAS score significantly correlated with the degree of walk impairment

### ***Results of Plantographic Examination***

Comparison of “deformed” feet, regardless of nosology, and feet of healthy participants during plantographic study showed significant ( $p<0.05$ ) prevalence of longitudinal (23.9%) and combined platypodia (21.7%) in “deformed” feet as compared to HP.

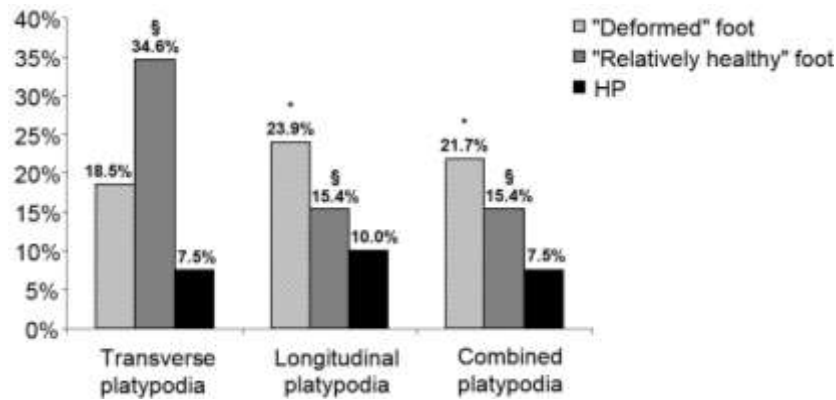


Figure 2: Plantographic characteristics of the deformed and contralateral (“relatively healthy”) feet in the studied groups

§ - “relatively healthy” foot is significantly different from healthy participants at  $p < 0.05$ .

\* - “deformed” foot is significantly different from healthy participants at  $p < 0.05$ .

The analysis of plantographic characteristics of contralateral (“relatively healthy”) feet and feet of healthy participants showed significant ( $p < 0.05$ ) prevalence of all platypodia types in “relatively healthy” feet as compared to HP. The degree of longitudinal platypodia in relatively healthy foot significantly ( $p < 0.05$ ) correlated with the disease duration. In all the groups longitudinal and transverse platypodia significantly correlated ( $p < 0.05$ ) with pains in lumbar spine (65%).

Evaluation of plantographic parameters in the studied groups showed that the group with plantalgia significantly ( $p < 0.05$ ) differed from the other groups by the rate of platypodia (85.7%), primarily, of transverse platypodia (47.6%). In the group with peripheral paresis hollow foot was observed significantly more often (within equinoplovarus deformity). The presence of transverse platypodia in groups with central and peripheral paresis of feet significantly ( $p < 0.05$ ) correlated with functional extension of lower extremity, change of leg length in 71.4% and 60% of cases, respectively.

## VI. RESULTS OF ELECTROPHYSIOLOGICAL STUDIES

### *Transcranial Magnetic Stimulation (TMS)*

Neurophysiological parameters, localized at cerebral level, were characterized by reduction of interneuron and motoneuron excitability at cortical level (MR and TMS threshold rise). TMS results at isolated lesion of spinal cord indicated on significant reduction of motor pathways conduction. Probably, this can be explained by topically compact location of descending neurons at the spinal level and involvement of a number of these motor pathways under abnormal focus at the thoracic level.

The analysis of separate motor symptoms in patients with central paretic foot dysfunction by TNS showed that there was direct correlation between foot paresis and central conduction time (CCT), and inverse correlation – between tonus and CCT (i.e. the lower the tonus, the higher is the CCT). Besides, intensification of tendon reflex

and manifestation of clonus directly correlated with MR thresholds (all the coefficients were significant at  $p < 0.05$ ). Direct correlation between hyperreflexia and increased cortex thresholds were observed in patients after ACVE.

Comparison of patients with lesions at spinal and cerebral levels showed more significant neuron conduction reduction rate in motor pathways (increase of CCT) in patients with lower paraparesis (Table 4).

Dissection of ST, that compressed motor pathways, led to increase of descending impulses and, as a result, to more extensive special and time impulse summation, which decreased CCT during TMS.

Table 4: TMS results in patients with cerebral and spinal level lesions (Group 1) in lower extremities

Groups Parameter	Level of lesion		
	Cerebral (ACVE) N=16	Spinal (TM, ST) N=14	Healthy participants N=40
CCT, ms	♦ 35.5±11.2	♦ 40.2±12.6•	14.4±0.2
F CCT, ms	♦ 22.3±8.7	♦ 23.5±7.1	13.2±0.5
MR thresholds, %	♦ 75.3±4.9•	66.3±4.3	65.3±2.3
F MR thresholds, %	♦ 64.3±2.6•	58.7±8.2	59.5±6.0
Amplitude index	♦ 2.46±3.29•	1.18±1.03	0.78±0.37

- ♦ – significant difference between HP and patients ( $p < 0.05$ ).
- – significant difference between patients ( $p < 0.05$ ).

Comparative analysis of TMS in patients with Parkinson's disease (PD) and multifocal dystonia (MFD) (Table 5) identified different neurophysiological patterns. At PD significantly lower CCT ( $p < 0.05$ ) was observed, as well as increase of amplitude index and decrease of motor response threshold at cortex stimulation. The patients with MFD were characterized by less expressed changes in CCT and motor response threshold. TMS in patients with MFD showed significant decrease (as compared to HP) of magnetic field intensity threshold that exerted motor effect, which indicated on increased reflectory readiness to muscle contraction.

Shortening of latency and increase of amplitude in patients with PD suggested that hyperexcitability of cortical and spinal motor neurons led to increase of MR amplitude due to greater amount of neurons that were excited by external stimulus. Probably, change in latency is caused by activation of pathways that are not stimulated in healthy people and, probably, by fast conduction or shorter pathways.

Table 5: Comparative analysis of lower extremities TMS parameters in patients with PD and MFD (Group 3)

Groups Parameter	PD N=16	MFD N=14	Healthy participants
CCT, ms	♦ 13.0±0.2	13.5±0.3•	14.4±0.2
Amplitude index	♦ 2.66±2.02•	0.64±0.44	0.78±0.37
MR threshold, %	♦ 54.3±2.5	♦ 55.3±4.1	65.3±2.3

- – significant difference between the patients in groups ( $p < 0.05$ ),
- ♦ – significant difference between the patients and healthy participants ( $p < 0.05$ ).

Table 6: Comparative analysis of lower extremities TMS parameters in patients with central foot paresis and foot dystonia (Groups 1 and 3).

Groups Parameter	Central foot paresis N=15	Foot dystonia N=15	Healthy participants N=20
CCT, ms	♦37.7±11.7•	♦13.2±0.4	14.4±0.2
Amplitude index	♦1.84±2.48	♦1.72±1.28	0.78±0.37
MR threshold, %	♦69.5±2.4•	♦54.7±3.2	65.3±2.3

- – significant difference between the patients in groups ( $p < 0.05$ ).
- ♦ – significant difference between the patients and healthy participants ( $p < 0.05$ ).

Comparative analysis of TMS parameters in patients with central foot paresis and foot dystonia (Table 6) showed that during the diseases associated with primary lesion of pyramid system, motor evoked potentials under magnetic stimulation were characterized by the tendency ( $p < 0.1$ ) towards increase of CCT via corticospinal pathways and rise of cortical response threshold. During the diseases associated with extrapyramidal system pathology (PD and MFD), neurophysiological parameters of corticospinal pathways functional activity impairment were identified, like decrease of CCT and threshold of magnetic motor response.

The specified changes allowed the authors to identify four TMS patters (cerebral, spinal, dystonic, parkinsonian) that can be used in differential diagnostics (Figure 3).

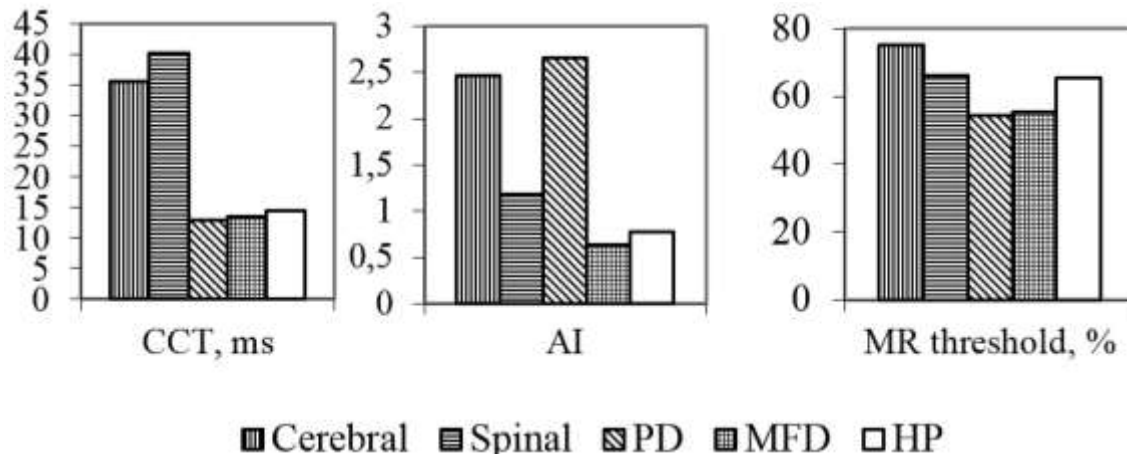


Figure 3: Main TMS parameters at different nosological entities.

**Somatosensory Evoked Potential.**

Cortex somatosensory activation reduction was identified in patients with ACVE complications (Table 7). Probably, increase of LH N30 and P40 peaks during SSEP examination can be explained by expressed impairment of afferent impulses conduction via fast sensory pathways of posterior columns of the spinal cord and associated brain stem and spinal-thalamic structures [20], [21], [22], [23]. Compact located motoneurons lesions at the level of spinal cord are characterized by lower reserve capacity and, as a result, by greater motor capacity deficit. Besides, somatosensory afferent pathway impairment at isolated spinal cord lesion could aggravate the existing motor defects.

Table 7: SSEP examination results in patients with cerebral and spinal CNS lesions in lower extremities (Group 1).

		Level of lesion		Healthy participants N=20
		Cerebral (ACVE) N=8	Spinal (TM, ST) N=7	
Latency	R	◆32.0±0.9	◆38.3±0.7●	29.5±1.2
N30	L	◆32.1±1.1	◆38.4±0.6●	29.4±1.2
Latency	R	◆47.3±1.9●	◆43.1±2.2	39.3±1.0
P40	L	◆47.5±1.8●	◆43.3±2.1	39.4±1.1
Latency	R	22.3±2.3	22.2±2.3	22.1±2.2
N22	L	22.2±2.2	22.1±2.2	22.0±2.1

- ◆ – significant difference between the patients and healthy participants (p<0.05)
- – significant difference between the patients in groups (p<0.05).

During SSEP study in 60% of patients with foot dystonia (Table 8) changes in P40 component was observed under stimulation of tibial nerve, which manifested as elongation of latent time and reduction of amplitude (at PD) or shortening of latent time (at MFD).

Table 8: Changes of SSEP parameters in patients with foot dystonia associated at PD and MFD (Group 3)

Latency		PD	MFD	HP
N30	R	29.6±1.0	29.4±1.4	29.5±1.2
	L	29.7±0.9	28.9±1.6	29.4±1.2
P40	R	◆40.8±1.4●	◆37.1±1.9	39.3±1.0
	L	◆41.0±1.5●	◆36.8±2.1	39.4±1.1

- ◆ – significant difference between the patients and healthy participants (p<0.05).

Comparative analysis revealed direct correlation between elongation of latent time, severity of walk impairment and duration of the disease, which indicated on spreading of degenerative process on afferent pathways. Shortening of latent time (at MFD) can be explained by extension of afferent pathway to cortex or by compensatory increase of afferent system role due to postural impairments, including hyperkinesia in foot.

### **Electroneuromyographical Study**

Average parameters of impulse conduction rate via motor pathways were below the norm at compensatory neuropathy of common peroneal nerve in lower leg, which corresponded with the results of clinical examination – paresis of foot and toes extensors.

In the group with foot dystonia, decrease of NCV in peripheral motor and sensory pathways was identified in 16 patients (53.3%) with foot dystonia associated with MFD and PD, in all the cases it was combined with expressed varus deformity of both feet. It can be suggested that functional denervation hypersensitivity can develop in patients with foot dystonia at lower extremity neuropathy.

The results, obtained by the method of stimulation ELMN at neurogenic plantalgia, confirmed the involvement of motor and sensory neurons into compression lesion and correlated with the results of clinical study. The most sensitive markers of compression foot nerve lesion were latent times of S- and M-responses.

### ***Results of Non-specific Orthopedic Therapy***

Rehabilitation treatment was aimed at changing movements pattern, in particular, at correcting “defect” walk stereotype with shoes and orthoses.

The treatment included two stages. The first stage (pre-orthosis) included procedures until the time when permanent orthosis was crafted. This stage treatment was aimed at complete relief or reduction of pain by means of manual therapy of feet and ankles as an integrated structure of supporting-motor apparatus and cross bandage taping of ankle, which provided heel fixation in inversed position and fixation of I metatarsal bone in plantar flexion position for 10 days. At the same time, it was recommended to limit the time of walking and standing. The second stage of treatment started when the orthosis was ready. It was aimed at orthopedic correction of foot deformation for prevention of secondary alterations in bones and joints of feet. The patients were offered to perform a complex of physical exercises for recreation of feet muscles.

The results of non-specific orthopedic therapy were evaluated in 46 patients, including 14 patients with peripheral foot paresis (Group 2), 16 patients with foot dystonia (Group 3) and 16 patients with plantalgia (Group 4). The rest patients in Groups 2, 3 and 4 (44 people) underwent conventional treatment without orthosis application. The therapy efficiency was evaluated based on patients subjective perception of changes, clinical examination and scoring by VAS and LEFS scale repeated in 6 months. The results of psychological testing showed that there were significant ( $p<0.05$ ) changes in patients psychic sphere (reduction of depression tendencies) that did not meet the parameters of healthy participants. Probably, this was associated with insufficient duration of treatment in some patients or overlaying of psycho-traumatic situations.

In 19.56% of patients that used orthoses, the long-term therapeutic effect was evaluated by the authors as “very good”: complete restoration of function, discomfort and spasms relief at walking, increase of lower extremity muscle force, significant changes in main clinical parameters. In 32.6% of patients the therapeutic effect was evaluated as “good” and in 43.4% - “satisfactory”.

The results of treatment were characterized primarily as “satisfactory” in the group of patients with peripheral foot paresis, which was associated with longer disease duration and fixed ankle joint contractures.

“Very good” score treatment results were not achieved in the group of patients with foot dystonia regardless of orthoses application, which significantly ( $p<0.05$ ) correlated with lower parameter changes during LEFS scoring. Long-term “unsatisfactory” effect rate was significantly lower ( $p<0.05$ ) in patients that used orthoses.

25% of patients with neurogenic plantalgia that used orthoses had treatment effect evaluated as “very good”.

«Unsatisfactory» results throughout all the period of observation were identified in 37.5% of patients that did not use orthoses.

Taking into account a diversity of nosological entities, the authors introduced such parameters as delta-VAS and delta-LEFS for objectivization of the results that reflected changes in scoring after the treatment (Table 9).

Table 9: Changes in LEFS and VAS scoring in groups after the treatment

Groups	Group 2	Group 3	Group 4
Parameter changes			
Δ LEFS	5.0±3.8	●3.4±3.2	6.5±2.8
Δ VAS	0.6±1.0	1.0±1.0	●4.3±1.5

- – significant difference between the patients in groups ( $p < 0.05$ ).

The analysis of changes in LEFS and VAS ( $\Delta$ ) scoring revealed maximum pain reduction in the group of patients with plantalgia. Minimal pain reduction was observed in the group of patients with MFD by LEFS scoring.

Evaluation of treatment results by LEFS scale in patients that used orthoses showed that they were insignificantly different from those that did not used orthoses.  $\Delta$ VAS results indicated on significant differences ( $p < 0.05$ ) in Group 4 ( $\Delta$ VAS was  $4.3 \pm 1.5$  and  $1.9 \pm 1.3$ , respectively).

The patients with the disease duration longer than one year had significantly ( $p < 0.05$ ) lower changes in LEFS and VAS scoring as compared to the patients with shorter disease duration.

Positive therapeutic effect was observed in more than 90% of patients who underwent foot function correction therapy. Subjective evaluation of the treatment results, regardless of nosologic entity, showed that “unsatisfactory” effect rate at early and late stages of treatment was significantly ( $p < 0.05$ ) higher in patients that refused to use orthoses (27.3%). The share of lowest score increased in all the patients with expressed static loads and duration of the disease.

## VII. CONCLUSION

1. Neurologic syndromes associated with foot function impairment are characterized by certain neurogenic foot deformities: spastic foot drop at central paresis and any deformity at peripheral paresis regarding the affected lower extremity peripheral nerve and involved muscles.
2. The analysis of plantographic characteristics of contralateral (“relatively healthy”) foot and feet of healthy participants showed significant ( $p < 0.05$ ) prevalence of all types of platypodia in “relatively healthy” foot. The degree of contralateral foot deformity, associated with central lesions, depended on the degree of the deformed foot paresis and spasticity. Deformities, associated with peripheral lesions, depended on the duration of the disease and premorbid constitutional changes.
3. Transcranial magnetic stimulation (TSM) study revealed four electrophysiological patterns: cerebral pyramidal, spinal pyramidal, parkinsonian and dystonic, which allows the authors to recommend this method for patients with neurogenic feet deformities for specification of the level of CNS impairment. The study of short latency somatosensory evoked potentials in patients with neurogenic feet deformities showed that in the majority of patients with central foot paresis, there was function reduction of fast sensory pathways and primary cortex activation impairment of somatosensory postcentral brain regions.
4. The most sensitive markers of low extremity nerve compression lesion were latent times at S- and M-responses. Stimulation ELNG study in patients with foot dystonia allows the specialists to identify

compression lesion of motor and sensory pathways in lower extremities, which is an additional peripheral maintaining factor.

5. The analysis of etiologic factors of compression neuropathy development in foot and ankle joint showed that platypodia, static and dynamic overload, weight gain (additional overload factor) and foot joints diseases were the most widespread ones.
6. Individual complex rehabilitation treatment for foot deformities in patients, based on clinical-neuropsychological peculiarities of the disease, showed positive effect in the majority of cases (90%).

### ***List of Abbreviations***

AI – amplitude index

VAS – visual analogue scale

CCT – central conduction time

FD – foot dystonia

HP – healthy participants

CA - corrective actions

QoL – quality of life

LECN - lower extremity compression neuropathy

CRPS - complex regional pain syndrome

LT - latent time

TA - trait anxiety

MR - motor response

MFD - multifocal dystonia

ISMD - inherited sensorimotor dystonia

ACVE - acute cerebrovascular event

ST - spinal tumor

PK - paradoxical kinesia

PF - peripheral factors

SA - state anxiety

BHMS - benign hypermobility syndrome

NCV - nerve conduction velocity

SSEP - somatosensory evoked potential

TM - thoracic myelopathy

TMS - transcranial magnetic stimulation

ELMG - electroneuromyography

NMR - nuclear magnetic resonance

LEFS – low extremity functional scale



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