BG Pulse Journal USA
ISSN:1947-2536 http://www.bgpulseusa.com/index.php/bg
BG PULSE JOURNAL

UDC: 616.831-009.11-053: 616-073: 616-085

DIAGNOSIS AND THERAPY OF PEDIATRIC MUSCLE SPASTICITY

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Doi: https://doi.org/10.5281/zenodo.21840446

Abstract: pasticity is one of the most common motor disorders in children with central nervous system lesions. It is characterized by a velocity-dependent increase in muscle tone, leading to functional limitations and reduced quality of life. Despite advances in treatment, challenges remain in early diagnosis and in selecting the most appropriate therapeutic strategy. The study included 180 children aged 3–16 years: 150 patients with spastic syndrome of various etiologies and 30 conditionally healthy children (control group). Clinical and neurological examination, assessment scales (Ashworth Scale, Barthel Index, GMFCS, MACS, Wechsler Scale), and instrumental methods (electroneuromyography and brain MRI) were applied. A differentiated approach to the diagnosis and treatment of spasticity in children improves therapeutic outcomes and should be considered a basis for personalized rehabilitation.

Keywords: spasticity, children, cerebral palsy, rehabilitation, GMFCS, Ashworth, neuromuscular disorders

INTRODUCTION

Relevance. Spastic syndrome is one of the most significant and frequent manifestations of upper motor neuron lesions in children and is characterized by a velocity-dependent increase in muscle tone, hyperreflexia, and impaired voluntary motor control. According to the classic definition proposed by James W. Lance [1], spasticity represents a component of the pyramidal tract lesion syndrome associated with dysregulation of stretch reflexes. Contemporary neurophysiological studies have shown that this condition is underlain by a complex imbalance between excitatory and inhibitory influences at the spinal cord level, including reduced presynaptic inhibition and altered activity of interneuronal networks [2].

Spasticity is widely prevalent in pediatric neurology and is observed across various nosological forms. According to epidemiological data, it is detected in more than 90% of children with cerebral palsy, approximately 35% of patients after stroke, about 50% of children after traumatic brain injury, and up to 70% of those with hereditary spastic paraplegia [3]. Despite the common pathophysiological mechanism, the clinical manifestations of spastic syndrome vary substantially depending on the level and nature of the central nervous system lesion, the patient's age, and disease duration. In recent years, particular attention has been devoted to studying the neurophysiological mechanisms underlying the formation of spasticity. Experimental studies have demonstrated that key roles are played by changes in α-motoneuron excitability, impaired

descending inhibitory influences, and plastic reorganization at the level of spinal reflex arcs [4]. Additional importance is attributed to changes in musculotendinous properties, including the development of fibrosis and muscle shortening, which intensify the clinical manifestations of spasticity and complicate treatment [5]. Despite a considerable body of research, the diagnosis of spastic syndrome in clinical practice remains predominantly clinical and often subjective. The most widely used Ashworth Scale has limitations related to insufficient sensitivity and inter-rater dependence [6]. At the same time, instrumental methods such as electroneuromyography and neuroimaging are not used systematically enough, which limits the possibility of objective patient stratification.

The choice of therapeutic tactics also remains a substantial problem. Various approaches are currently applied, including pharmacotherapy (baclofen, botulinum toxin), physiotherapy, and surgical methods. However, treatment effectiveness largely depends on the accurate assessment of the degree of spasticity, functional status, and the involvement of various levels of the nervous system. The absence of clear differentiated algorithms leads to fragmented and often insufficiently effective treatment [7].

Thus, at the present stage there is a discrepancy between the accumulated knowledge of the pathogenesis of spasticity and its practical application in clinical neurology. Insufficient integration of clinical, functional, and instrumental data complicates the development of personalized approaches to the management of pediatric patients. In this regard, the development of a differentiated approach to the diagnosis and treatment of muscle spastic syndrome in children, based on a comprehensive assessment of neurological status, functional capacity, and instrumental findings, is of particular relevance.

The aim of this study was to develop and scientifically substantiate a differentiated approach to the diagnosis and treatment of muscle spastic syndrome in children, taking into account the severity of neurological impairment and the functional status of patients.

MATERIALS AND METHODS

A total of 180 children aged 3 to 16 years were included in the study. The main group comprised 150 patients with spastic syndrome of various origins, and the control group comprised 30 conditionally healthy children matched for age and sex. Within the main group, patients with cerebral palsy predominated (60%), along with children with sequelae of traumatic brain injury (15%), stroke (10%), hereditary spastic paraplegia (10%), and multiple sclerosis (5%), reflecting the clinical heterogeneity of spastic syndrome in pediatric practice. The study was conducted in several stages: initial clinical and neurological examination, functional assessment using standardized scales, instrumental verification, and subsequent patient stratification with the formation of a differentiated treatment strategy.

Research methods.

Clinical and neurological examination: All patients underwent a standard clinical, anamnestic, and neurological examination assessing muscle tone, tendon reflexes, motor activity, symmetry of the lesion, and the presence of pathological reflexes characteristic of upper motor neuron syndrome.

Functional assessment: To objectify the degree of spasticity, the level of motor impairment, and functional status, standardized and validated scales were used: muscle tone was assessed using the Modified Ashworth Scale [8, 6]; independence using the Barthel Index [9]; gross motor function level using the GMFCS system [10]; manual abilities using the MACS system [11]; and cognitive function using the Wechsler Scale [12]. Assessments were conducted in accordance with the original protocols for these instruments.

Instrumental methods: To clarify the nature and level of the nervous system lesion, the following were used: electroneuromyography (ENMG) with analysis of conduction and muscle activity [13]; brain magnetic resonance imaging (MRI) using standard protocols [14]. The use of instrumental methods supplemented the clinical assessment and increased the objectivity of patient stratification.

Patient stratification: Based on the combined clinical, functional, and instrumental data, patients in the main group were classified according to the severity of spastic syndrome and functional deficit into three categories: mild, moderate, and severe. Stratification took into account Ashworth Scale scores, GMFCS and MACS levels, the Barthel Index, and ENMG and MRI findings.

Treatment principles: The study applied a differentiated therapeutic approach based on the severity of spasticity and functional deficit. Treatment measures included rehabilitation programs, pharmacotherapy (including muscle relaxants), botulinum toxin therapy, and, when necessary, consultations with related specialists.

Statistical analysis: Statistical processing of the data was performed using standard methods of variation statistics. Means and standard deviations were calculated. Corresponding parametric and non-parametric tests were used to assess differences between groups. Differences were considered statistically significant at $p < 0.05$.

RESULTS

Distribution of patients by degree of spasticity.

Analysis of baseline clinical data showed that children with a moderate degree of spasticity predominated in the study cohort (n = 150). The distribution is presented in Table 1.

Table 1. Distribution of patients by degree of spasticity (n = 150)

Degree of spasticity	n	%
Mild	48	32%
Moderate	69	46%
Severe	33	22%

The mean Ashworth Scale score in the overall group was 2.6 ± 0.8 points.

Thus, nearly half of the patients (46%) had a moderate degree of spasticity, indicating the presence of clinically significant but potentially reversible pathology.

Clinical and functional characteristics of the groups.

Based on an integrated assessment of clinical, functional, and instrumental parameters, all patients in the main group were stratified into three clinical-functional groups according to the severity of spasticity and functional deficit: Group I – mild degree (n = 48), Group II – moderate degree (n = 69), Group III – severe degree (n = 33). A comparative characterization of the groups is presented in Table 2.

Table 2. Clinical and functional characteristics of patients according to degree of spasticity

Parameter	Group I (mild, n=48)	Group II (moderate, n=69)	Group III (severe, n=33)
Ashworth, points	1.3 ± 0.2	2.4 ± 0.3	3.5 ± 0.4
GMFCS	I–II	II–III	IV–V
Barthel, points	85 ± 10	58 ± 12	32 ± 11
GMFM, %	72 ± 8	48 ± 9	24 ± 7
Clinical characteristics	minimal limitations, independent ambulation preserved	gait and coordination disturbances, reduced endurance	contractures, pronounced motor limitations, dependence on assistance

As shown by the data presented, motor and functional status deteriorated progressively with increasing severity of spastic syndrome. Patients in Group I showed minimal clinical manifestations, a comparatively high level of independence, and preserved gross motor function. In Group II, a significant decrease in motor activity was recorded compared with Group I, accompanied by gait disturbances and reduced coordination and endurance. The most pronounced changes were observed in Group III patients, who were characterized by high Ashworth scores, low GMFM and Barthel scores, and marked limitations in independent mobility and self-care.

Results of instrumental examinations (ENMG)

Quantitative parameters.

To objectify neurophysiological disturbances and clarify the nature of functional changes in the peripheral neuromuscular apparatus, electroneuromyography was performed in all patients of the main group, with assessment of conduction and reflex activity parameters. The data obtained revealed consistent changes in ENMG parameters depending on the severity of spastic syndrome. Comparative analysis was performed between the control group and the three clinical-functional patient groups. As the severity of spasticity increased, a consistent change was observed in all analyzed electrophysiological parameters. The most pronounced deviations were recorded in patients with severe spastic syndrome. Motor nerve conduction velocity progressively decreased from 52 ± 3 m/s in the control group to 49 ± 4 m/s in patients with mild spasticity, 44 ± 5 m/s in moderate spasticity, and 39 ± 6 m/s in severe spasticity. Thus, the maximum decrease in conduction velocity in Group III patients was approximately 25% compared with the control group ($p < 0.01$). Already at the stage of moderately severe spastic syndrome, a noticeable deviation of this parameter from normal values was observed. In contrast, M-wave amplitude increased progressively with worsening clinical severity. Whereas it was 6.5 ± 1.2 mV in the control group, it was 7.8 ± 1.4 mV in patients with mild spasticity, 9.2 ± 1.6 mV in moderate spasticity, and reached 11.0 ± 2.1 mV in the severe spasticity group. The increase in M-wave amplitude relative to the control group was most pronounced in Group III, reaching approximately 69% ($p < 0.01$).

A similar trend was observed for latency. This parameter increased from 3.2 ± 0.4 ms in the control group to 4.4 ± 0.7 ms in patients with severe spasticity. The progressive prolongation of latency reflected increasing functional impairment of neuromuscular transmission and paralleled the increasing clinical severity of spastic syndrome. Of particular significance was the dynamics of the H/M ratio, which characterizes the level of reflex excitability. In control group patients, this parameter was 0.35 ± 0.08, whereas in mild spasticity it increased to 0.48 ± 0.10, in moderate spasticity to 0.62 ± 0.12, and in severe spasticity to 0.79 ± 0.15. Thus, in patients with severe spasticity, the H/M ratio was more than twice that of the control group ($p < 0.01$), indicating a pronounced enhancement of reflex activity.

Treatment efficacy

Overall dynamics.

The efficacy of the therapy administered was assessed on the basis of a comparative analysis of clinical and functional parameters before and after treatment. The data obtained are presented in Table 3.

Table 3. Changes in parameters before and after treatment

Parameter	Before treatment	After treatment	p
Ashworth	3.1 ± 0.6	1.9 ± 0.5	<0.001
GMFM (%)	42 ± 8	60 ± 10	<0.01
Barthel	55 ± 12	67 ± 14	<0.01

As shown by the data presented, following treatment there was a significant reduction in the degree of spasticity on the Ashworth Scale, from 3.1 ± 0.6 to 1.9 ± 0.5 points ($p < 0.001$). At the same time, an increase in gross motor function scores on the GMFM scale was observed, from 42 ± 8% to 60 ± 10% ($p < 0.01$), along with an increase in the level of functional independence on the Barthel Index, from 55 ± 12 to 67 ± 14 points ($p < 0.01$).

Dynamics by group.

Comparative analysis of treatment outcomes according to the baseline severity of spastic syndrome revealed differences in the magnitude of positive dynamics (Table 4).

Table 4. Changes in spasticity, gross motor function, and functional independence parameters in patients with varying severity of spastic syndrome

Group	Δ Ashworth	Δ GMFM	Δ Barthel
Mild	−22%	+10%	+12%
Moderate	−45%	+24%	+28%
Severe	−28%	+12%	+15%

In patients with mild spasticity, the reduction in Ashworth Scale scores was 22%, the increase in GMFM was 10%, and the increase in the Barthel Index was 12%.

In the moderate spasticity group, the most pronounced positive dynamics were observed: a 45% reduction in spasticity level, a 24% improvement in motor function, and a 28% increase in the level of independence.

In patients with severe spasticity, the reduction in Ashworth scores was 28%, the increase in GMFM was 12%, and the increase in Barthel was 15%.

Thus, the most pronounced positive changes were recorded in the group of children with moderate spasticity ($p < 0.01$).

Efficacy of the differentiated approach

An overall assessment of the efficacy of the differentiated approach showed positive dynamics across all major parameters analyzed (Table 5).

Table 5. Overall efficacy of the differentiated approach

Parameter	Change (%)
Reduction in spasticity (Ashworth)	−38%
Improvement in motor function (GMFM)	+18%
Increase in independence (Barthel)	+22%

The most pronounced change was observed for the degree of spasticity: the reduction in Ashworth Scale scores was 38%. At the same time, an improvement in gross motor function on the GMFM scale of 18% and an increase in the level of functional independence on the Barthel Index of 22% were observed. These data reflect the overall clinical effect of the therapy administered and confirm the effectiveness of the differentiated approach to the treatment of children with spastic syndrome.

Relationship between ENMG and clinical findings

Correlation analysis was performed to assess the relationship between neurophysiological and clinical characteristics (Table 6).

Table 6. Correlations between ENMG parameters and clinical characteristics

Parameter	r	p
ENMG ↔ GMFCS	0.47	<0.05
ENMG ↔ Ashworth	0.52	<0.01

As shown in Table 6, a moderate direct correlation was found between ENMG parameters and the level of motor impairment on the GMFCS scale ($r = 0.47$; $p < 0.05$). Similarly, a positive correlation was established between ENMG parameters and the severity of spasticity on the Ashworth Scale ($r = 0.52$; $p < 0.01$). These findings indicate that as the clinical severity of spastic syndrome increases, the neurophysiological signs of pathological reflex activity also intensify.

Discussion

The findings obtained confirm that spastic syndrome in children is a clinically and pathophysiologically heterogeneous condition requiring a differentiated approach to diagnosis and treatment. The present study demonstrated that the distribution of patients by degree of spasticity is not uniform: the predominance of moderate forms (46%) indicates the presence of a substantial group of patients who are potentially the most responsive to therapeutic intervention. The

relationship identified between Ashworth, GMFCS, and Barthel scores confirms the concept of a close association between neurophysiological impairment and the patient's functional status. The correlation coefficients obtained ($r = 0.68$ and $r = -0.59$) are consistent with previous research indicating that the degree of spasticity is one of the key factors determining the level of motor activity and independence of patients. The ENMG results demonstrated increased reflex activity and signs of motoneuron hyperexcitability, confirming the leading role of spinal mechanisms in the formation of spasticity. These findings are consistent with current concepts of the pathogenesis of spastic syndrome, according to which impaired descending inhibitory control and reorganization of segmental reflex arcs are of key importance. The observed decrease in nerve conduction velocity is of particular significance, as it may reflect secondary changes in the neuromuscular system, including structural and functional reorganization. Thus, spasticity should be considered not only as a neuronal phenomenon but also as a complex condition encompassing peripheral changes in muscle tissue.

The application of a differentiated treatment approach made it possible to achieve statistically significant improvement in all key parameters. A 38% reduction in the level of spasticity was accompanied by a clinically significant improvement in motor function (+18%) and level of independence (+22%). These results indicate the high efficacy of personalized therapy compared with a traditional standardized approach. The most pronounced therapeutic effect was observed in patients with moderate spasticity. This finding may be explained by the preserved plastic potential of the central nervous system and the lesser severity of secondary structural muscle changes. At the same time, in patients with severe spasticity, treatment efficacy was limited, which is likely related to the formation of contractures and persistent morphological changes.

The data obtained confirm that a key problem in current clinical practice is insufficient patient stratification. The lack of integration of clinical, functional, and instrumental data reduces treatment efficacy, as was also demonstrated in the present study. The implementation of a comprehensive approach incorporating the GMFCS, MACS, and Ashworth scales together with instrumental methods makes it possible to increase diagnostic accuracy and optimize treatment selection.

Thus, the results of the present study demonstrate that the differentiated approach is not only theoretically justified but also a clinically effective tool for the management of patients with spastic syndrome. It enables a transition from symptomatic correction to personalized neurorehabilitation oriented toward specific pathophysiological mechanisms and the functional needs of the patient.

Conclusion

The present study showed that spastic syndrome in children is a clinically heterogeneous condition, distinguished by considerable variability in the severity of motor and functional impairment. The use of standardized clinical and functional scales, including Ashworth, GMFCS, and MACS, makes it possible to objectify the severity of spastic syndrome and improve the accuracy of patient stratification. The findings indicate that a differentiated approach to diagnosis and treatment, based on a comprehensive assessment of clinical, functional, and neurophysiological parameters, provides a reliable reduction in the severity of spasticity, improvement in motor function, and an increase in the level of functional independence. The most pronounced therapeutic effect was observed in patients who began treatment earlier and for whom a personalized management strategy was selected in a timely manner.

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