International Journal of Psychosocial Rehabilitation, Vol. 24, Issue 02, 2020
ISSN: 1475-7192

EXPRESSION OF NEUROLOGICAL SYMPTOMATICS IN PATIENTS DEPENDING ON THE DEGREE OF SPINE DEFORMATION

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Abstract—*A survey of 337 patients with axial spinal deformities was performed. The following neurological syndromes were identified: vegetative, reflex, radicular and spinal. As the degree of spinal deformity increases, the severity of neurological changes increases. The operated patients showed a regression of neurological manifestations.*

Keywords— *axial deformities of the spine, neurological changes, spinal disorders, neurophysiology.*

I. INTRODUCTION

Progressive axial deformities of the spine (PAD) lead to early disability and social maladaptation of patients and dysfunctions of the musculoskeletal, cardio-respiratory, neuromuscular and other body systems, as well as due to the development of secondary depressive and anxiety-phobic disorders, symptoms of autonomic dysfunctions [5, 8, 10, 14, 16]. The prevalence of axial spinal deformity (ASD) in the population reaches 15.3% [8, 10]. To date, there are no clear criteria for the diagnosis of neurological syndromes in scoliosis [12]. The lack of proper neurological assessment of neurological syndromes associated with scoliosis does not allow us to develop optimal rehabilitation and therapeutic measures aimed at the speedy restoration of functions and quality of life. Systematic studies of neurological impairment in cases of axial spinal deformity (ASD) are insufficient in the available domestic literature. Neurological disorders during gross spinal deformities remain one of the most severe and poorly studied areas. [1, 6, 7].

The aim of the study is to study neurological symptoms in patients with axial spinal deformities.

II. MATERIAL AND RESEARCH METHODS

A total of 337 patients with ASD aged 10 years to 32 years (mean age 21.4 ± 4.6 years) were examined. Among them, 229 patients without surgery with varying degrees of spinal deformity and 108 operated patients with an early postoperative period (EPOP) - up to 1 year and late postoperative period (LPOP) - more than 5 years.

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DOI: 10.37200/IJPR/V24I2/PR200332
Received: 15 Dec 2019 | Revised: 02 Jan 2020 | Accepted: 15 Jan 2020

The neurological status was studied traditionally - clinical and neurological examination and on scales: assessment of pain intensity on a visual analogue scale (VAS); Beck Depression Inventory, adapted at the Research Institute named after V. Bekhterev. Assessment: Depression Level(DL) ≤50 points - no depression; DL = 51 ÷ 59 points – sub depressive state or masked depression of situational or neurotic genesis; DL = 60 ÷ 69 points - mild depression; DL ≥70 - a truly depressive state. A decrease in DL by more than 5 points is considered clinically significant [13].

Electroneuromyography (ENMG) and the study of somatosensory evoked potentials (SSEP) were carried out according to the standard method by stimulating the tibial nerve on both sides using the Neurosoft MVP-4 apparatus.

Electrical stimulation of the tibial and peroneal nerves was performed in the popliteal fossa, the speed of the efferent impulse were recorded [9].

In the study of SSEP, latency and amplitude of the N22 peak (sensory response measured from the tibial nerve at the level of the cauda equina and spinal cone) and the interval N22-P38 (interval from the lumbar level to the cortical centers) were measured [2, 9].

The cognitive component of pain was determined by the study of cognitive evoked potentials (CEP) on the Neuronspektr-5 apparatus (Neurosoft) and the study of the autonomic nervous system, revealing skin-galvonic evoked potentials (SGEP) - VNS metry using the VNS-Spectrum (Neurosoft) apparatus [2].

CEP was investigated by isolating P300 complexes at the expense of significant sound stimuli. To evaluate the motor component, the P300 technique was performed by pressing a button when recognizing significant auditory stimuli. For further analysis, the averaged latency and amplitude values of the P300 potential were taken [2, 3, 15].

Skingalvonic evoked potentials (SGEP) is a change in electrodermal activity in response to a stimulus (in particular, electric current). SGEP is a suprasegmental somatovegetative reflex, the sweat glands are the effector organ, and the posterior hypothalamus is the "generator" of the response. The predominance of sympathetic or parasympathetic effects leads to an increase or decrease in sweating. This is manifested in the deviation of SGEP in the negative or positive direction.

III. METHODOLOGY

electrodes are superimposed on the index finger of the right hand. Electrical pulses are stimulated with a frequency of 10 Hz for 1 second with a current strength depending on the threshold of the SGEP: 25 μA at a threshold of 12 μA, 50 μA at a threshold of 25 μA, 100 μA at a higher threshold. The following indicators of SGEP are calculated: LP - latent period of SGEP; A1, A2, A3 - the amplitudes of the first, second and third phases; S1, S2, S3 - the duration of the ascending parts of the phases. For further analysis, we focused on A1p and A2p - the amplitudes of the first and second phases, since A1p reflects the parasympathetic response, and A2p - sympathetic [2].

To study the degree of deformation of the spinal column, an X-ray examination was performed. Patients underwent spondylography of the spine in 2 projections, in a straight standing and lateral lying. Depending on the arc of curvature of the spine, several degrees of axial spinal deformities (ASD) are distinguished. I degree - a curvature angle of 10 degrees, II degree - a curvature angle - from 11 to 25 degrees, III degree - a curvature angle from 25 to 40 degrees, IV degree - a curvature angle of more than 40 degrees [11].

DOI: 10.37200/IJPR/V24I2/PR200332
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IV. RESULTS AND DISCUSSIONS

The main neurological complaints in patients with ASD are back pain (in 96% of cases), autonomic disorders (sweating of the hands and feet, chilliness in the limbs and back - in 100%), impaired gait (in 35.3%), headaches (in 29.4%), head tilt (in 27.5%) and dizziness (in 7.8%).

Back pain can be of a different nature: from a feeling of fatigue in the back towards evening, to severe radicular pain, in addition there are local point pains, the so-called trigger point pains, as well as reflex and reflected pains radiating to arms and legs.

Headaches in patients are mostly qualified as tension headaches associated with both concomitant affective disorders (anxiety, depression) and with pericranial muscle tension.

Dizziness in patients often occurs in an orthostatic state, lasts no more than one minute, accompanied by the illusion of movement of one's own body or surrounding objects.

Because of the curvature of the spine and distortion of the pelvis, gait disturbances and head tilts in one direction or another arise [5, 6, 8, 10].

The degree and variety of neurological symptoms in the presence of ASD is largely determined by the type and localization of spinal deformity. Polymorphism of neurological pathology is caused by a common pathological process that captures a large number of roots, which is manifested by a variety of neurological syndromes. Clinical and neurological disorders in the studied patients were characterized by a large polymorphism and manifested themselves in the form of the symptoms shown in table 1.

As can be seen from the above table 1, neurological disorders in the presence of ASD are diverse. The revealed pathology from the side of neurological status was of a secondary nature and, occured due to the presence and severity of spinal deformity. Polymorphism of neurological symptoms occur because of the diversity and prevalence of spinal lesions, leading to the development of these symptoms. In most cases, we observed signs of damage to the peripheral nervous system in the form of radicular sensitivity, paresthesia, on the one hand, and increased tendon reflexes, as well as clonusoids and clonuses, as signs of pyramidal pathway insufficiency, on the other. However, there were cases of a combination of hyperreflexia and loss of abdominal reflexes in 58.4% of cases, which we regarded as a sign of central insufficiency of the pyramidal pathways.

The pain syndrome was different in intensity and localization. By the nature of the pain, they were described as “shooting,” “stitching,” “pulsating” in nature. According to localization, in 51.1% of cases there was torocalgia, in 74.24% there was lumbalgia, in 15.72% of cases lumbar ischialgia. The intensity of the pain was evaluated on the VAS scale. The severity of pain according to VAS data is shown in table 1, and with the increase in the degree of spinal deformity, the severity of pain also increased. In the operated patients, the pain syndrome on the VAS scale was lower than in patients with III and IY stage and decreased over time - in patients with LPOP (late postoperative period) it was lower than in patients with EPOP(early postoperative period).

When analyzing the level of depression on the Beck scale, it was revealed that patients generally did not have depressive manifestations, only patients with severe deformity (IY stage) had a mild depression (Table 1).

In the study of peripheral nerves by ENMG, in particular, the tibial and fibular nerves, it was found that the speed of the impulse along these nerves tends to decrease with an increase in the degree of spinal deformity, and in the tested patients, the speed of the impulse was gradually restored (Table 2).

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A similar trend was observed in the study of SSEP in the peripheral nerves (N22 latency) and the spinal cord (interval N22-P38), which revealed an increase in latency of the SSEP with an increase in the degree of spinal deformity of the SSEP and an improvement in these parameters in the operated patients (Table 2).

An analysis of the autonomic nervous system research on SGEP revealed an increase in parasympathicotonia (A1p amplitude) and sympathicotonia (A2p amplitude), but with a large prevalence of sympathicotonia. Moreover, sympathicotonia tended to increase with increasing degree of spinal deformity, and parasympathetic did not have such a dependence on the degree of deformation. In the treated patients, there was a decrease in both parasympathicotonia and sympathicotonia over time (Table 2).

In the study of cognitive functions (CEP lat P300) in patients with ASD, no significant changes were detected. However, with an increase in the degree of spinal deformity, P300 latency tended to increase. This is possibly associated with chronic pain syndrome with tension suprasegmental vegetative centers. In the operated patients, there was a slight increase in P300 latency (in patients with EPOP), possibly associated with getting anesthesia during surgery, and an improvement in cognitive functions in patients with LPOP (Table 2).

Thus, the studies of the nervous system according to scales and neurophysiological studies allow us to conclude that with an increase in the degree of defect of the spine in patients, the pain syndrome according to the VAS scale increases, signs of mild depression appear, conductivity along the peripheral nerves and spinal cord worsens, and parasympathicotonia increases, so and sympathicotonia with a predominance of sympathicotonia and minor cognitive changes are detected. In operative patients, a gradual improvement in all neurological changes is noted over time.

In order to systematize the neurological symptoms encountered in our patients, we used clinical syndromes based on the classification principles proposed by I.P. Antonov (1985), as well as D.K. Bogorodinsky and A.A. Skoromets (2002), where reflex, radicular, and spinal syndromes were detected. In addition, due to the fact that some patients with I tbsp. spinal deformities revealed only vegetative manifestations, we included the fourth autonomic syndrome.

Vegetative syndrome in frequency of occurrence took the first place (from 83.7% to 100% of patients) and was manifested by sweating or cooling of the distal extremities, de - or hyperpigmentation of the skin of the trunk, grease and hyperesthesia of it, and a violation of dermographism. According to SGEP, sympaticotonia (an increase in the amplitude of A2p) was manifested in some patients (an increase in the amplitude of A2p), in some patients, parasymaticotonia increased the amplitude of A1p).

Reflex syndrome was found in most patients and was manifested by pain and fatigue in the back, in the lumbar region under static and physical exertion, in some cases (25.8%) of girdle pain, in the chest, fatigue of the legs, pain in the legs. During examination of patients, we determined the pain of paravertebral points, the defansion of the long back muscles.

Radicular syndrome was detected slightly less frequently than reflex and was clinically expressed by a radicular sensitivity disorder, more from the deformity side, tendon anisoreflexia. Soreness of paravertebral points, positive symptoms of tension: Neri, Lassega, Wasserman, Matskevich. On ENMG and SSEP, a slowdown in the speed of the impulse along the motor and sensory nerve fibers was detected.

Spinal syndrome was observed in a small part of patients and neurological changes were expressed in the addition of pathological signs: Babinsky, Rossolimo, Oppenheim, foot clonuses, sensory disturbances in the

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conductor type, as well as disorders of the pelvic organs. In SSEP, a slowdown in the spinal cord is detected (interval N22-P38).

Vegetative syndrome in an isolated form was observed only in 45 patients with a single-stage arthritis. In patients with reflex syndrome, all patients had vegetative manifestations. In patients with radicular syndrome, autonomic and reflex manifestations were also present. And in patients with spinal syndrome in the neurological clinic there were signs of autonomic, reflex and radicular syndromes.

From table 3 it is clearly seen that among the neurological manifestations of ASD, syndromes indicating a predominant lesion of the peripheral nervous system and a violation of autonomic innervation predominate.

Changes in the central nervous system in the form of tension of higher autonomic centers (SGEP), with a predominance of sympathicotonia, mild depressive changes on the Beck scale, a slight slowdown in cognitive functions according to CEP, are most likely a consequence of the effects of chronic pain syndrome [4] and long-term progression of spinal deformity.

The frequency of occurrence of neurological syndromes in case of ASD is shown in Table 3. In Fig. 1 it is clearly seen that in non-operated patients, neurological symptoms tend to increase and aggravate as the degree of spinal deformity increases. And in the operated patients there is a regression of the severity and severity of neurological syndromes with increasing time spent after surgery. In this case, it is possible that there are sanogenetic mechanisms for the restoration of neurological deficit over time in patients with a straightened spinal column.

Thus, on the basis of our studies, it can be concluded that the severity and polymorphism of neurological symptoms is largely determined by the degree of curvature of the spinal column and the secondary interest of the spinal roots, ganglia and spinal cord, as well as some interest in the central nervous system due to chronic pain and long-term existence and progression of spinal deformity.

V. FINDINGS

1. In patients with axial spinal deformities, there is a polymorphic neurological symptomatology, manifested in the form of 4 main syndromes: autonomic, reflex, radicular and spinal.
2. In patients with axial spinal deformities, peripheral nervous system lesions predominate. Minor changes in the central nervous system are a consequence of the effects of chronic pain and long-term spinal deformity.
3. With an increase in the degree of spinal deformity, the severity and polymorphism of neurological symptoms increase, and in the operated patients, neurological symptoms improve over time over spinal deformity. All this indicates the relationship between the severity of the angle of deformation of the spinal column and involvement in the pathological process of the spinal cord and peripheral nervous system.

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Received: 15 Dec 2019 | Revised: 02 Jan 2020 | Accepted: 15 Jan 2020

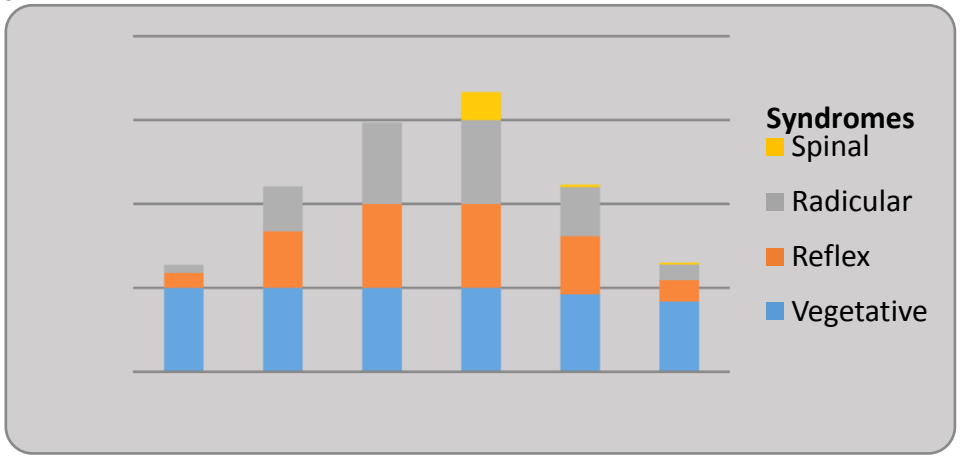

Fig. 1. Neurological syndromes in patients depending on the severity of spinal deformity

Table 1. Neurological manifestations in patients depending on the severity of spinal deformity

Types of Disorders	**I st. n=62**	**II st. n=58**	**III st. n=64**	**IY st. n=45**	**EPOP n=65**	**LPOP n=43**
VAS	3,1±0,1	4,5±0,3	6,1±0,2	7,0±0,3	4,2±0,3	2,8±0,4
Beck Scale	41,5±0,84	44,7±0,84	49,5±0,54	53,49±1,02	48,3±0,5	40,1±0,6
The back muscles	11(17,8)	39(67,3)	64(100)	45(100)	40,1 (61,7)	14 (32,6)
Root-type sensitivity disorders	6 (9,7)	31 (53,6)	62 (96,9)	45 (100)	50 (76,9)	10 (23,3)
Conductivity Sensitivity Disorders	0	0	7 (10,9)	8 (17,8)	6 (9,23)	2 (4,7)
Paresthesia on the back	0	0	19 (29,7)	22 (48,9)	65 (100)	22 (51,2)
Vegetative-trophic disorders	62 (100)	58 (100)	64 (100)	45 (100)	60 (92,3)	20 (46,5)
Pelvic disorders in the form of urinary incontinence	0	0	0	9 (20,0)	1 (1,5)	0
Symptoms of tension Neri, Lasseg, Wasserman,	2 (3,2)	5 (8,6)	19 (29,7)	20 (44,5)	48 (73,8)	16 (37,2)
Tendon reflex revitalization (PR and AR)	16 (25,8)	21 (36,2)	54 (84,4)	33 (73,3)	50 (76,9)	22 (51,2)
Tendon reflex reduction (PR and AR)	0	0	10 (15,6)	12 (26,7)	36 (55,4)	10 (23,3)
Prolapse of the abdominal reflexes	15 (24,2)	17 (29,3)	25 (39,1)	40 (88,9)	42 (64,6)	12 (27,9)
Pathological reflexes (Babinsky and Rossolimo)	0	0	5 (7,8)	10 (22,2)	1 (1,5)	1 (2,33)
Clonus or clonoid of the foot	0	0	2 (3,1)	7 (15,6)	2 (3,08)	1 (2,33)
Gait Disorders (scoliotic)	3 (4,8)	49 (8,5)	64 (100)	45 (100)	35 (53,8)	4 (9,3)

Note: In parentheses are percentages

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Received: 15 Dec 2019 | Revised: 02 Jan 2020 | Accepted: 15 Jan 2020

Table 2. Neurophysiological parameters in patients depending on the severity of spinal deformity

Types of Disorders	**I st. n=62**	**II st. n=58**	**III st. n=64**	**IY st. n=45**	**EPOP n=65**	**LPOP n=43**
ENMG TN SI eff.	49,1±2,1	48,5±1,8	45,8±1,4	44,6±1,5	47,14±1,1	48,66±1,4
ENMG PN SI eff.	49,8±1,9	48,4±1,7	46,4±1,6	44,8±1,5	47,52±1,0	48,43±1,5
SSEP latency N22	21,6±0,4	22,3±0,41	22,8±0,3	23,5±0,5	22,3±0,4	21,48±0,4
SSEP interval N22-P38	17,2±0,3	17,8±0,3	18,3±0,3	19,3±0,3	17,7±0,3	17,4±0,3
SGEP amplitude A1p	1,3±0,2	1,2±0,2	1,3±0,3	1,4±0,4	1,29±0,2	1,02±0,4
SGEP amplitude A2p	3,6±0,3	3,7±0,4	4,0±0,5	4,2±0,6	3,76±0,3	2,21±0,4
CEP latency P300	318,51±2,2	327,12±1,9	335,18±2,0	347,55±2,4	338,06±1,0	326,1±2,5

Note: TN – tibial nerve; PN – peroneal nerve; SI eff. - the speed of the efferent impulse.

Table 3. Neurological syndromes in patients depending on the severity of spinal deformity

Syndromes	**I st. n=62 (%)**	**II st. n=58(%)**	**III st. n=64(%)**	**IY st. n=45(%)**	**EPOP n=65 (%)**	**LPOP n=43(%)**
Vegetative	62 (100)	58 (100)	64 (100)	45 (100)	60 (92,3)	36 (83,7)
Reflex	11 (17,8)	39 (67,3)	64 (100)	45 (100)	45 (69,2)	11 (25,6)
Radicular	6 (9,7)	31 (53,5)	62 (96,9)	45 (100)	38 (58,5)	8 (18,6)
Spinal	-	-	-	15 (33,3)	2 (3,1%)	1 (2,3%)

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DOI: 10.37200/IJPR/V24I2/PR200332
Received: 15 Dec 2019 | Revised: 02 Jan 2020 | Accepted: 15 Jan 2020

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DOI: 10.37200/IJPR/V24I2/PR200332
Received: 15 Dec 2019 | Revised: 02 Jan 2020 | Accepted: 15 Jan 2020