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Evoked Brain Potentials in the Preoperative Diagnosis of Type 1 Chiari Malformation

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I. Introduction

The rapid development of computer technology predetermined a new stage in the formation of clinical neurophysiology. Improvement of the system equipment makes it possible to adequately assess various functional parameters of cerebral and spinal structures, to conduct their dynamic observation (6, 8, 14).

Evoked brain potentials - acoustic stem evoked potentials and somatosensory evoked potentials have the greatest diagnostic value in determining the functional state of the brain stem structures and spinal conducting systems (10, 12, 13). In modern publications, there is no consensus on the neurophysiological aspects of clinical syndromes of Chiari malformation 1 - cerebellar, bulbar, pyramidal, syringomyelitis (2, 4, 5, 7). Also, we did not find definite data on the choice of treatment tactics for Chiari malformation of type 1 caused by neurophysiological changes. Currently, neurologists and neurosurgeons, when choosing a therapy for patients with Chiari malformation of type 1, rely mainly on the data of subjective complaints, neurological examination and the degree of tonsil ectopia by MRI (1,3,9,11). However, this whole complex does not fully give an objective picture of the functional state of the stem structures, especially in case of subclinical forms of pathology. This circumstance was the reason for us to conduct clinical and neurophysiological comparisons of the indicated syndromes with type 1 Chiari malformation and the development of neurophysiological characteristics of various clinical syndromes of Chiari 1 malformation according to the data of acoustic stem, somatosensory and motor evoked potentials.

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II. Materials and Methods

Examined 207 patients with Chiari malformation 1 according to MRI studies, who are on outpatient and inpatient treatment at the Republican Specialized Scientific and Practical Medical Center of Neurosurgery, Ministry of Health of the Republic of Uzbekistan. The standard in determining the degree of omission of the cerebellar amygdala in Chiari malformations was the Chamberlain line, which runs from the hard palate to Opistion (2,8,9). We considered the displacement of the cerebellar tonsils beyond the Chamberlain line up to 5 mm admissible. In our studies, we used exactly the Chamberlain line to guide the anatomical anomalies of the craniovertebral junction and the degree of ectopia of the cerebellar tonsils (Fig. 1, 2).

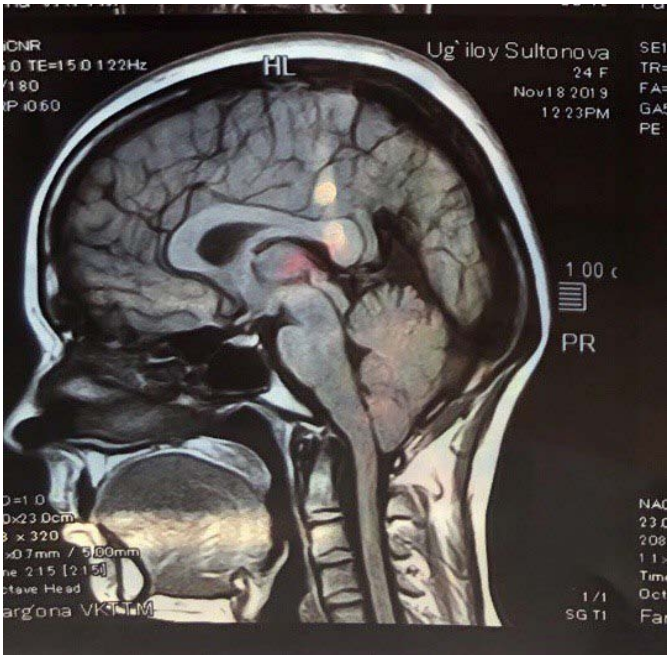

Figure 1: An example of MRI of patient S., with Chiari malformation of type 1 with ectopia of cerebellar tonsils 14mm on the right and 12mm on the left

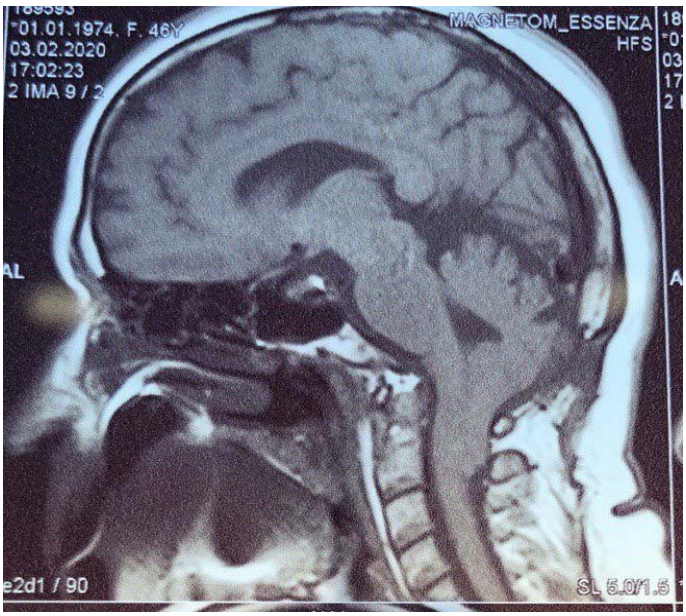

Figure 2: MRI of patient Z., 34 years old, with an anomaly of the craniovertebral junction with displacement of the cerebellar tonsils below the Chamberlain line up to 24 mm from 2 sides

We analyzed clinical symptoms in 207 patients with Chiari malformation of type 1 according to MRI data. Of these, 73 are men and 134 are women between the ages of 14 and 62. In the structure of neurological syndromes, 82 patients with cerebellar syndrome, 26 patients with manifestations of bulbar syndrome, 21 patients with pyramidal syndrome and 78 patients with clinical symptoms of syringomyelitis syndrome were examined.

All patients were examined according to a multimodal neurophysiological protocol, including acoustic stem evoked potentials, somatosensory evoked potentials, and motor evoked potentials (7). The studies were carried out on a 4-channel "Synapsis" complex (Neurotech, Russia) with computer data processing.

For the included acoustic stem evoked potentials, a standard vertex-mastoidal lead (M1-Cz,

M2-Cz) was used; stimulation was performed through headphones with acoustic clicks of 0.1 ms duration biurally with a feed frequency of 20 Hz and a sound of 70 dB.

When carrying out somatosensory evoked potentials, the discharge electrodes were installed according to the standard technique (see Chapter 2) C4-Fz - with n. medianus S stimulation C3-Fz- with n. medianus D stimulation. Stimulation was carried out with electric impulses in the projection of the median nerve at the level of the wrist by current 15-20 m A, frequency 2 Hz.

We performed stimulation EMG by default for n.glossopharyngeus et n.accessorius with the setting of recording electrodes in accordance with the muscle innervation. If necessary, we supplemented the studied nerves based on the clinical syndrome.

III. Results and Discussion

a) Cerebellar syndrome in patients with Chiari malformation type 1

We studied the data of acoustic stem evoked potentials, somatosensory evoked potentials and motor evoked potentials in 82 patients - 52 women and 30 men with clinical manifestations of cerebellar syndrome and Chiari 1 anomaly. The control group consisted of 30 healthy individuals.

The obtained data, including acoustic brainstem evoked potentials - studies in patients with cerebellar syndrome are presented in Table 1. It was revealed that in all examined patients the latent periods of and peaks were extended bilaterally with significant differences compared to healthy individuals. The mean values of the latencies of the remaining components - I. II, IV, were unchanged compared with the results of the control group. The amplitude indices of the and peaks were significantly increased relative to the control values, which dissociated with the general ideas about the depression of amplitude indices with the inclusion of acoustic stem evoked potentials in patients with pathology of stem structures. In our opinion, an increase in the amplitudes of the III components in patients with cerebellar syndrome indicated functional irritation of the stem structures at the level of the superior olivary complex. Analysis of the mean values of the peak-to-peak intervals showed an insignificant delay in III, IV and I-in the study group with significant differences from the control individuals, which indicated a slowdown in conduction at the pontomesencephalic level. Peak intervals I- were preserved in comparison with the control group, which can be explained by the intactness of the peripheral portion of the auditory analyzer.

Table 1: Indicators of acoustic stem evoked potentials - latency period, peak amplitudes and inter-peak intervals in healthy individuals of the control group (n=30) and patients with cerebellar syndrome Chiari malformation 1 (n= 82)

Latent period, ms

Control group (n=30)	PI	PII	PIII	PIV	PV
S	1.79± 0.16	2.95 ± 0.18	3.94 ± 0.24	5.06 ± 0.22	5.97 ±0.25
D	1.72 ± 0.17	2.98 ± 0.19	3.92 ± 0.22	5.13 ± 0.20	6.02± 0.25
Cerebellar Syndrome (n=82)					
S	1.74 ± 0.18	2.96 ± 0.17	4.25 ± 0.25	5.25± 0.21	6.55± 0.22*
D	1.68 ± 0.16	3.02 ± 0.19	4.25 ± 0.21*	5.38± 0.19	6.70 ±0.24*

Amplitude, μV

Control group(n=30)	PI	PIII	PV
S	0.286± 0.05	0.262± 0.04	0.368 ±0.06
D	0.282± 0.04	0.265± 0.06	0.338± 0.08
Cerebellar Syndrome (n=82)			
S	0.348± 0.03	0.370± 0.03**	0.375 ± 0.05*
D	0.340± 0.04	0.372± 0.05**	0.380 ± 0.07*

Peak intervals, ms

Control group (n=30)	PI-PIII	PIII-PV	PI-PV
S	2.19± 0.16	2.06± 0.18	4.38± 0.22
D	2.24± 0.18	2.08± 0.22	4.46± 0.24
Cerebellar Syndrome (n=82)			
S	2.56± 0.15	2.52± 0.14**	4.90±0.21*
D	2.88± 0.17	2.60± 0.18**	4.82±0.20*

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

We analyzed the data of somatosensory evoked potentials in 82 patients with clinical manifestations of cerebellar syndrome and Chiari 1 anomaly. Registration of somatosensory evoked potentials was carried out with stimulation of the median and tibial nerves from 2 sides, the average values of somatosensory evoked potentials were compared with the values in the control group. The results of the somatosensory evoked potentials of the study in cerebellar syndrome are presented in Table 2.

Table 2: Indices of somatosensory evoked potentials upon stimulation of the median nerve - latency period, peak amplitudes and inter-peak intervals in healthy controls (n=30) and patients with cerebellar Chiari malformation 1 (n=82)

Latency, ms	Controlgroup(n=30)	Cerebellarsyndrome(n = 82)
N9 Erba	9.6±0.7	9.4±0.7
N13 neck	13.2±0.8	14.5±0.7*
N20 cortex	18.8±1.0	18.9±1.2
Amplitude,μV		
N9 Erba	5.4±2.5	5.6±2.2
N13 neck	2.9±1.3	2.7±1.2
N20 cortex	2.8±1.6	2.9±1.5
Peakintervals, ms		
N9-N13	3.5±0.4	3.2±0.3
N13-N20	5.8±0.5	6.9±0.2*
N9-N20	9.2±0.5	8.8±0.7

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

As can be seen from the above proposed data, in the group of patients with cerebellar syndrome, there was a significantly significant increase in the latency of the N13 component to 14.5 ms compared to the control group, which was more often symmetric bilateral (84% of observations). The amplitude indices of all components of the somatosensory evoked potentials were preserved relative to healthy individuals. The extension of the peak-to-peak intervals N13-N20 to 6.9 ms was isolated in the group of patients with Chiari malformation 1; the parameters of the peak-to-peak intervals N9-N13 and N9-N20 were unchanged compared to the control values.

When analyzing these indicators somatosensory evoked potentials for stimulation of the tibial nerve, shown in Table 3, a significant extension of the latent period of the N30 component to 38.1 ms was determined in patients with cerebellar syndrome relative to the control group. Changes in the amplitudes of the components N22, N30, P37 in the studied group of patients were not recorded. The N30-P37 peak-to-peak interval was moderately extended to 12.5 ms in most cases (68%) with cerebellar syndrome compared with healthy individuals; the N22-N30, N22-P37 peak latencies corresponded to the control group.

Table 3: Indices of somatosensory evoked potentials during stimulation of the tibial nerve - latent period, peak amplitudes and inter-peak intervals in healthy controls (n=30) and patients with cerebellar Chiari malformation 1 (n= 82)

Latency, ms	Controlgroup(n=30)	Cerebellarsyndrome (n = 82)
N 22 lumbar	23.6±1.9	23.2±1.6
N 30 cervical	30.6±2.5	38.1±1.2**
P37 cortex	37.5±3.4	36.±3.0
Amplitude, μV		
N 22 lumbar	1.3±0.5	1.7±0.3*
N 30 cervical	0.9±0.3	1.1±0.2
P37 cortex	2.6±1.5	2.9±1.5

Peakintervals, ms		
N22-N30	7.62±1.14	7.86±1.07
N30-P37	8.05±1.32	12.5±1.54*
N22-P37	15.7±1.65	16.9±1.35

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

Thus, the analysis of the data somatosensory evoked potentials upon stimulation of the median and tibial nerves revealed an increase in the latency of the N13, N30 components in patients with cerebellar Chiari malformation syndrome 1 in a predominant number of cases was combined with an expansion of the inter-peak intervals N13-N20 (64% of patients) and N30-P37 (55% of patients), which indicated a slowdown in afferentation at the level of the cervical spinal cord and then the medulla oblongata - thalamus cortex with a tendency to decrease the postsynaptic activation of the medulla oblongata nuclei.

We analyzed the electroneuromyography of the data obtained during stimulation of the oculomotor, facial, glossopharyngeal nerves, as well as the median and tibial nerves in the group of patients with cerebellar disorders and Chiari 1 anomaly. As follows from Table 4 below, the values of the speed of motor conduction of the SPI eff were insignificant. decreased in the facial and glossopharyngeal nerves with significant differences from the control group. The efferent velocity along the oculomotor nerve in the study group was preserved relative to the control. Indicators of the speed of conduction of the impulse SPI eff along the nerves of the upper and lower extremities were unchanged in comparison with healthy individuals. Also, we did not register significant deviations in the A max of the M-response amplitudes for all studied nerves in the group of patients. However, after stimulation, pathological waves along the facial nerve were observed in 27% of patients with cerebellar syndrome, whereas in the group of healthy individuals, such a phenomenon was not recorded.

Table 4: Electroneuromyography indices of oculomotor, facial, glossopharyngeal, median and tibial nerves in healthy controls (n = 30) and patients with cerebellar Chiari malformation syndrome 1 (n = 82)

Control group(n=30)	SPI, m / s	Amax, μV	Additionalpathologicalwaves
Oculomotornerve	29.4±2.2	1080±105.5	-
Facialnerve	39.5±1.8	1235±126.3	-
Glossopharyngealnerve	42.6±2.0	1860±164.0	-
Mediannerve	61.0±1.7	6254±267.0	-
Tibialnerve	49.6±2.1	7125±745.5	-
Cerebellarsyndrome (n = 82)			
Oculomotornerve	29.1±2.0	1072±105.8	
Facialnerve	34.8±1.6**	1130±138.0*	+
Glossopharyngealnerve	39.2±1.4**	1851±170.5	
Mediannerve	60.4±1.5	6158±245.6	
Tibialnerve	48.3±1.9	7245±760.8	

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

In our opinion, small deviations of the SPI eff indices towards a decrease in the facial and glossopharyngeal nerves against the background of relatively unchanged values of the M-response amplitudes testified to the functional involvement of the structures of the pons pons and medulla oblongata in cerebellar syndrome. Pathological waves along the facial nerve may correspond to irritative disorders at the cerebellopontine level. Unchanged parameters of SPI eff and amplitudes of muscle responses during stimulation of the median and tibial nerves in the group of patients with Chiari 1 malformation indicated the absence of dysfunctions of the segmental apparatus in cerebellar disorders.

b) Bulbar syndrome in patients with Chiari malformation type 1

We analyzed the neurophysiological data of 26 patients with clinical manifestations of bulbar syndrome with Chiari malformation 1 at the age of 18 to 65 years, the number of men was 9 cases, women - 17 cases. The control group consisted of 30 healthy individuals.

All patients of this group underwent an analysis including acoustic stem evoked potentials of the data, which revealed significant differences with the control group in terms of the latency parameters and amplitudes of the components presented in Table 4.5. Thus, the latency of the PIII and PV components was

moderately extended from 2 sides compared to the control group. There was a reduction in the amplitude values of the peaks PV and PV, often bilaterally, while the asymmetry of changes including acoustic brainstem evoked potentials-indicators was recorded in 76% of the examined patients, the complete absence of PIII and PV components was noted in 24% of cases in comparison with healthy individuals. Noteworthy is the significant expansion of the peak-to-peak intervals PIII-PV and PI-PV by almost two times in the group of patients with bulbar manifestations compared to the control. In 24% of patients, it was impossible to analyze the peak-to-peak intervals due to the absence of the formation of PIII and PV components. Thus, the analysis of the above changes in the parameters of acoustic brainstem evoked potentials objectively indicated gross conduction disturbances in the clinical manifestations of bulbar syndrome in patients with Chiari 1 malformation at the level of the inferior bridge and midbrain, often asymmetrically with dissociated functional involvement of pontomesencephalic structures.

Table 5: Indicators of acoustic stem evoked potentials - latency period, peak amplitudes and inter-peak intervals in healthy individuals of the control group (n = 30) and patients with bulbar Chiari malformation syndrome 1 (n = 26)

Latent period, ms

Control group (n=30)	PI	PII	PIII	PIV	PV
S	1.79± 0.16	2.95 ± 0.18	3.94 ± 0.24	5.06 ± 0.22	5.97 ±0.25
D	1.72 ± 0.17	2.98 ± 0.19	3.92 ± 0.22	5.13 ± 0.20	6.02± 0.25
Bulbar syndrome (n=26)					
S	1.80 ± 0.18	2.98 ± 0.17	4.35 ± 0.25	5.30± 0.21	7.05± 0.22**
D	1.76± 0.16	3.01 ± 0.20	4.70 ± 0.21**	5.65± 0.19	8.01 ±0.24**

Amplitude, μV

Control group (n=30)	PI	PIII	PV
S	0.286± 0.05	0.262± 0.04	0.368 ±0.06
D	0.282± 0.04	0.265± 0.06	0.338± 0.08
Bulbar syndrome (n=26)			
S	0.348± 0.03	0.050± 0.01**	0.050 ±0.02**
D	0.340± 0.04	0.180± 0.02*	0.220± 0.04*

Peakintervals, ms

Controlgroup (n = 30)	PI-PIII	PIII-PV	PI-PV
S	2.19± 0.16	2.06± 0.18	4.38± 0.22
D	2.24± 0.18	2.08± 0.22	4.46± 0.24
Bulbar syndrome (n=26)			
S	2.36± 0.15	3.96± 0.15**	6.05±0.20*
D	2.48± 0.17	3.65± 0.20**	6.35±0.21**

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - $P< 0.05$, ** - $P < 0.01$*

We analyzed the data of somatosensory evoked potentials in 26 patients with clinical manifestations of bulbar syndrome, Chiari 1 anomaly. Registration of somatosensory evoked potentials was carried out with stimulation of the median and tibial nerves from 2 sides, the mean values of somatosensory evoked potentials were compared with the values in the control group. The results of somatosensory evoked potentials of the study in bulbar syndrome are presented in table 6.

Table 6: Indices of somatosensory evoked potentials during median nerve stimulation - latency period, peak amplitudes and inter-peak intervals in healthy controls (n = 30) and patients with bulbar Chiari malformation syndrome 1 (n = 26)

Latency, ms	Controlgroup (n = 30)	Bulbarsyndrome (n = 26)
N9 Erba	9.6±0.7	10.1±0.8
N13 neck	13.2±0.8	18.4±1.2*
N20 cortex	18.8±1.0	18.7±1.5
Amplitude,μV		
N9 Erba	5.4±2.5	5.1±2.0
N13 neck	2.9±1.3	1.1±0.5**
N20 cortex	2.8±1.6	1.2±0.4**
Peakintervals, ms		
N9-N13	3.5±0.4	3.9±0.5
N13-N20	5.8±0.5	8.0±0.7**
N9-N20	9.2±0.5	9.8±0.6*

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

As can be seen from the above proposed data, in the group of patients with bulbar syndrome, there was a significant increase in the latencies of the N13 components up to 18.4 ms in comparison with the control group. Also, in the group of these patients, a statistically significant decrease in the amplitudes of the N13 and N20 components was recorded, often bilateral with an asymmetry in 61% of observations relative to healthy individuals. The values of the N13-N20 peak-to-peak intervals were significantly increased in the majority of patients in this group up to 8.0 ms, however, the parameters of the N9-N13, N9-N20 intervals were slightly changed relative to normal values.

Further, we studied the data of somatosensory evoked potentials obtained on stimulation of the tibial nerve in patients with bulbar syndrome with Chiari malformation 1, presented in Table 7.

Table 7: Indices of somatosensory evoked potentials during stimulation of the tibial nerve - latency period, peak amplitudes and inter-peak intervals in healthy controls (n = 30) and patients with bulbar Chiari malformation syndrome 1 (n = 26)

Latency, ms	Controlgroup(n = 30)	Bulbarsyndrome(n = 26)
N 22 lumbar	23.6±1.9	23.9±1.6
N 30 cervical	30.6±2.5	42.8±1.26**
P37 cortex	37.5±3.4	38.4±3.0
Amplitude,μV		
N 22 lumbar	1.3±0.5	1.65±0.3*
N 30 cervical	0.9±0.3	0.28±0.1**
P37 cortex	2.6±1.5	2.85±1.6
Peakintervals, ms		
N22-N30	7.62±1.14	7.80±1.05
N30-P37	8.05±1.32	17.8±1.52**
N22-P37	15.7±1.65	17.0±1.25

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

We found a statistically significant isolated extension of the N30 component latency to 42.8 ms in the group of patients with bulbar syndrome compared to the control group, while the latencies of the N22 and P37 components were relatively preserved. Also, these patients showed a reduction in the amplitude of the N30 component to 0.28 μV against the background of unchanged values of the amplitudes of the N22 and P37 components in comparison with normal values. A significant expansion of the N30-P37 peak-to-peak intervals up to 17.8 ms was recorded in the group of patients with bulbar syndrome, often had a pronounced asymmetric character (in 61% of cases) compared with the control group, although the N22-N30 and N22-P37 peak-to-peak intervals had slight deviations from the norm ...

So, the analysis of changes in the parameters of somatosensory evoked potentials for the stimulation of the median and tibial nerves in patients with clinical manifestations of bulbar syndrome indicated a pronounced slowdown in conduction at the presynaptic level of the medulla oblongata nuclei with a decrease in their activation. A pronounced retardation of afferent conduction at the pontomedullary level in bulbar disorders was combined with moderate disturbances in thalamo-cortical conduction.

Characteristics of electroneuromyo graphy - data for bulbar syndrome with Chiari 1 anomaly was

analyzed in 26 patients. The results of the motor evoked potentials obtained by stimulation of the oculomotor, facial, glossopharyngeal nerves, as well as the median and tibial nerves are presented in Table 8.

Table 8: Electroneuromyography indices for the oculomotor, facial, glossopharyngeal, median and tibial nerves in healthy controls (n = 30) and patients with bulbar Chiari malformation syndrome 1 (n = 26)

Control group (n = 30)	SPI m / s	Amax, μV	Additional pathological waves
Oculomotor nerve	29.4±2.2	1080±105.5	-
Facial nerve	39.5±1.8	1235±126.3	-
Glossopharyngealnerve	42.6±2.0	1860±164.0	-
Mediannerve	61.0±1.7	6254±267.0	-
Tibialnerve	49.6±2.1	7125±745.5	-
Bulbar syndrome (n = 26)			
Oculomotor nerve	28.5±2.0	1072±124.8	
Facial nerve	34.1±1.6*	1180±122.0*	+
Glossopharyngeal nerve	20.8±2.6**	788±182.0**	+++
Mediannerve	54.5±1.8*	5011±256.5	
Tibialnerve	42.7±1.7	6450±628.5	

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - $P< 0.05$, ** - $P < 0.01$*

When analyzing the obtained indicators, a significantly significant decrease in the speed of the efferent impulse was revealed during stimulation of the glossopharyngeal nerve in the group of patients with bulbar syndrome in a relatively healthy group, while in 60% of the examined the parameters of SPIEff were reduced by more than 2 times compared with the control. It should be noted that even with mild bulbar symptoms, the efferent STI indices significantly differed from normal in the direction of decrease, which, possibly, reflected subclinical functional disorders. The rate of efferent conduction along the facial nerve decreased by less than 25%, and along the oculomotor nerve was relatively unchanged from the initial parameters. Along with changes in speed indicators, there was a significant decrease in the amplitudes of the M-response along the glossopharyngeal nerve, more than 2 times relative to the indicators of healthy individuals. Amplitude values of the M-response of the facial and oculomotor nerves with a tendency to decrease in the group of patients compared with the control group. Attention is drawn to the presence of pathological waves of fibrillation in 30% of patients with stimulation of the glossopharyngeal nerve, which indicated the involvement of the medulla oblongata nuclei in the pathological process. When analyzing the electroneuromyography of the data obtained during the stimulation of the median and tibial nerves, a tendency towards a decrease in the rate of conduction of the efferent impulse was recorded in the group of patients compared with healthy individuals. At the same time, the indicators of the maximum amplitude of the M-response of the median and tibial nerves were practically unchanged in comparison with the control group. This phenomenon, in our opinion, is associated with reactive involvement of the efferent pathways in patients with bulbar syndrome with the development of bilateral pyramidal insufficiency.

Thus, in the study of electroneuromyography, the bulbar syndrome was characterized by a pronounced conduction disorder at the level of the medulla oblongata nuclei, often with the capture of the intersection of the pyramidal tract. electroneuromyography data made it possible to objectively assess the condition of patients with bulbar syndrome Chiari malformation 1, even in the subclinical phase of the disease.

c) Pyramidal syndrome in patients with Chiari malformation type 1

We studied the neurophysiological characteristics of the pyramidal syndrome in 21 patients with Chiari malformation 1, including 13 women and 8 men aged 16 to 45 years. Acoustic stem evoked potentials were performed in all patients of this group. The data obtained by us in comparison with the control group are presented in table 9.

Table 9: Indicators of acoustic stem evoked potentials - latency period, peak amplitudes and inter-peak intervals in healthy individuals of the control group (n = 30) and patients with pyramidal syndrome Chiari malformation 1 (n=21)

Latent period, ms

Control group (n = 30)	PI	PII	PIII	PIV	PV
S	1.79± 0.16	2.95 ± 0.18	3.94 ± 0.24	5.06 ± 0.22	5.97 ±0.25
D	1.72 ± 0.17	2.98 ± 0.19	3.92 ± 0.22	5.13 ± 0.20	6.02± 0.25
Pyramidalsyndrome (n =21)					
S	1.80 ± 0.16	2.90 ± 0.17	4.41 ± 0.25**	5.25± 0.21	6.55±0.22*
D	1.82 ± 0.16	2.86 ± 0.15	4.48 ± 0.21**	5.38± 0.19	6.52±0.24*

Amplitude, μV

Control group (n = 30)	PI	PIII	PV
S	0.286± 0.05	0.262± 0.04	0.368 ±0.06
D	0.282± 0.04	0.265± 0.06	0.338± 0.08
Pyramidal syndrome (n=21)			
S	0.290±0.05	0.375± 0.03*	0.380 ± 0.05*
D	0.284±0.04	0.360± 0.05*	0.385 ± 0.04*

Peak intervals, ms

Control group (n = 30)	PI-PIII	PIII-PV	PI-PV
S	2.19± 0.16	2.06± 0.18	4.38± 0.22
D	2.24± 0.18	2.08± 0.22	4.46± 0.24
Pyramidalsyndrome (n=21)			
S	2.32± 0.14	3.48± 0.14**	4.75±0.20*
D	2.29± 0.12	3.52± 0.16**	4.84±0.18*

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

As can be seen from the above proposed table, when the study included acoustic stem evoked potentials in patients with pyramidal syndrome Chiari 1 malformation, the most variable were the latencies of components and inter-peak latencies. Thus, in the group of patients, there was an increase in the latency of the P and PV peaks, often symmetric in 85% of cases, relative to the control values. The latencies of the components PI, PII, PIV were unchanged in comparison with normal values. Attention is drawn to the phenomenon of an increase in the amplitude indices of the peaks PIII and PV from 2 sides in the group of patients with pyramidal syndrome with Chiari malformation 1 relative to the control group, which, in our opinion, is caused by irritative disorders of the motor tract against the background of concomitant hypertensive-hydrocephalic symptoms. Typical disorders involving the acoustic brainstem evoked potentials of the pyramidal syndrome indicators were manifested in the protraction and expansion of the inter-peak intervals PIII-PV and PI-PV, which was significantly different in comparison with the group of healthy individuals. Moreover, in more than 80% of cases, these changes were bilateral. Thus, with the inclusion of acoustic stem evoked potentials in patients with clinical manifestations of pyramidal syndrome, a widespread deceleration of conduction at the pontomedullary level is recorded, which has a bilateral nature. The phenomena of irritation of the motor pathways can also correspond to the symptoms of pyramidal insufficiency, which in most cases developed against the background of hypertensive-hydrocephalic syndrome.

Next, we analyzed the indicators of somatosensory evoked potentials in patients with clinical manifestations of pyramidal syndrome and Chiari 1 anomaly. The resulting changes in the indicators of somatosensory evoked potentials during stimulation of the median nerve are presented in Table 10.

Table 10: Indices of somatosensory evoked potentials during median nerve stimulation - latency period, peak amplitudes and inter-peak intervals in healthy controls (n = 30) and patients with pyramidal syndrome Chiari malformation 1 (n = 21)

Latency, ms	Controlgroup (n = 30)	Pyramidalsyndrome (n = 21)
N9 Erba	9.6±0.7	9.2±0.7
N13 neck	13.2±0.8	14.1±0.6*
N20 cortex	18.8±1.0	20.2±0.8*
Amplitude,μV		
N9 Erba	5.4±2.5	5.6±2.0
N13 neck	2.9±1.3	2.8±1.1
N20 cortex	2.8±1.6	2.0±1.3
Peakintervals, ms		
N9-N13	3.5±0.4	3.6±0.3
N13-N20	5.8±0.5	6.4±0.4*
N9-N20	9.2±0.5	10.8±0.6*

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

So, in pyramidal syndrome, a slight increase in the latencies of the N13 and N20 components was recorded as compared with the control group. The increase in the latencies of N13 and N20 was symmetrical in most patients in this group, while the latency of the N9 component was relatively unchanged. The amplitude parameters N9, N13, N20 in the group of patients with Chiari malformation 1 were significantly unchanged in comparison with the group of healthy individuals. There was a tendency to a prolongation of the peak-to-peak intervals N13-N20 and N9-N20 in the group of patients with pyramidal syndrome reliably relative to the control. The peak-to-peak interval N9-N13 remained unchanged in the group of patients in comparison with healthy individuals.

Table 11 shows the indicators of somatosensory evoked potentials during stimulation of the tibial nerve in patients with clinical manifestations of pyramidal syndrome.

Table 11: Indices of somatosensory evoked potentials during stimulation of the tibial nerve - latency period, peak amplitudes and inter-peak intervals in healthy individuals of the control group (n = 30) and patients with pyramidal syndrome Chiari malformation 1 (n = 21)

Latency, ms	Controlgroup (n = 30)	Pyramidalsyndrome (n = 21)
N 22 lumbar	23.6±1.9	2.42±1.7
N 30 cervical	30.6±2.5	32.3±2.2**
P37 cortex	37.5±3.4	39.7±3.1*
Amplitude, μV		
N 22 lumbar	1.3±0.5	1.4±0.4
N 30 cervical	0.9±0.3	1.2±0.4*
P37 cortex	2.6±1.5	2.6±1.8*
Peakintervals, ms		
N22-N30	7.62±1.14	7.80±1.05
N30-P37	8.05±1.32	9.5±1.40*
N22-P37	15.7±1.65	16.4±1.5*

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

The latency parameters of the components N22, N30, P37 in the group of patients with Chiari malformation 1 were practically unchanged in comparison with the control group. An isolated expansion of the P37 component (cortex) was noted in 20% of individuals in this group, which can be explained by reactive involvement of cortical structures. The amplitudes of the components N22, N30, P37 in the group of patients did not differ from the normal values. The peak-to-peak interval N22-P37 (lumbar-cortex) was slightly widened relative to the control group. The MPI values N22-N30, N30-P37 were significantly unchanged compared to the control group. From the above, it follows that pyramidal syndrome with Chiari

malformation 1, according to somatosensory evoked potentials, is characterized by a slowdown in conduction in the central parts of the somatosensory system of the brain. Delayed afferentation at the pontomedullary level in pyramidal syndrome was a little expected fact in combination with movement disorders, which, in our opinion, is due to widespread functional disorders of the conducting systems at the level of the medulla oblongata.

We conducted a study of electroneuromyography of the data obtained during stimulation of the oculomotor, facial and glossopharyngeal nerves, as well as the median and tibial nerves in patients with clinical manifestations of pyramidal syndrome, which are presented in Table 12.

Table 12: Electroneuromyography indices for the oculomotor, facial, glossopharyngeal, median and tibial nerves in healthy individuals of the control group (n = 30) and patients with pyramidal syndrome Chiari malformation 1 (n = 21)

Control group (n = 30)	SPI, m/s	Amax, μV	Additional pathological waves
Oculomotor nerve	29.4±2.2	1080±105.5	-
Facial nerve	39.5±1.8	1235±126.3	-
Glossopharyngealnerve	42.6±2.0	1860±164.0	-
Mediannerve	61.0±1.7	6254±267.0	-
Tibialnerve	49.6±2.1	7125±745.5	-
Pyramidalsyndrome (n = 21)			
Oculomotornerve	27.1±2.1	1052±104.8	
Facialnerve	36.8±1.75*	1126±120.8*	
Glossopharyngealnerve	40.8±2.4*	1635±158.4*	
Mediannerve	42.6±1.4**	3825±253.9*	
Tibialnerve	30.5±2.5**	4905±462.5*	

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - $P< 0.05$, ** - $P < 0.01$*

As follows from the table above, in the group of patients with pyramidal syndrome, there was a significant tendency towards a decrease in PII in the facial and glossopharyngeal nerves. The velocity parameters of the oculomotor nerve were practically unchanged relative to the control group. The amplitudes of the M-responses obtained during the stimulation of the cranial nerves slightly decreased in the group of patients as compared with the normal values. Such changes were symmetrical in most of the subjects (in 80% of cases) and were caused, in our opinion, by bilateral corticonuclear insufficiency. Attention is drawn to the decrease in efferent SPI parameters when stimulating the nerves of the upper and lower extremities. The values of the speed of motor behavior were significantly reduced in the median and tibial nerves from 2 sides in comparison with the control group. All patients with pyramidal disorders showed a reduction in the maximum amplitude of muscle responses along the median and tibial nerves with significant differences from the group of healthy individuals. No additional pathological waves were recorded during stimulation electroneuromyography from the nerves of the upper and lower extremities.

Thus, electroneuromyography data in pyramidal syndrome in patients with Chiari 1 malformation indicated impaired efferent conduction at the suprasegmental level with a predominant involvement of motor pathways at the level of the inferior bridge and medulla oblongata.

d) Syringomyelitis syndrome in patients with Chiari malformation type 1

We have studied in a comparative aspect the neurophysiological features of the syringomyelitis clinical syndrome in 78 patients with Chiari malformation 1, of whom 52 are women and 26 are men aged 14 to 55 years.

Acoustic stem evoked potentials were performed in all patients of this group, the examination results in comparison with the control group are presented in Table 13.

Table 13: Indices of acoustic stem evoked potentials - latency period, peak amplitudes and inter-peak intervals in healthy individuals of the control group (n = 30) and patients with syringomyelitis syndrome Chiari malformation 1 (n = 78)

Latent period, ms

Control group (n = 30)	PI	PII	PIII	PIV	PV
S	1.79± 0.16	2.95 ± 0.18	3.94 ± 0.24	5.06 ± 0.22	5.97 ±0.25
D	1.72 ± 0.17	2.98 ± 0.19	3.92 ± 0.22	5.13 ± 0.20	6.02± 0.25
Syringomyelitis syndrome (n = 78)					
S	1.80 ± 0.16	2.94 ± 0.17	4.20 ± 0.21*	5.10± 0.20	6.25± 0.22*
D	1.78 ± 0.16	2.96 ± 0.18	4.24 ± 0.19*	5.14± 0.19	6.30 ±0.24*

Amplitude, μV

Control group (n = 30)	PI	PIII	PV
S	0.286± 0.05	0.262± 0.04	0.368 ±0.06
D	0.282± 0.04	0.265± 0.06	0.338± 0.08
Syringomyelitis syndrome (n = 78)			
S	0.280± 0.05	0.310± 0.04**	0.370 ± 0.04*
D	0.286± 0.04	0.325± 0.05**	0.382 ± 0.06*

Peakintervals, ms

Control group (n = 30)	PI-PIII	PIII-PIV	PI-PV
S	2.19± 0.16	2.06± 0.18	4.38± 0.22
D	2.24± 0.18	2.08± 0.22	4.46± 0.24
Syringomyelitis syndrome (n = 78)			
S	2.30± 0.15	2.36± 0.12*	4.56±0.22**
D	2.84± 0.15	2.42± 0.14*	4.61±0.21**

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

As follows from the above data, in the group of patients with syringomyelitis syndrome, there was a significant tendency to the expansion of the latency of the PIII and PV peaks from 2 sides compared to the control group. Bilateral changes in the latent parameters of PIII and PV were observed in 58 (75%) patients. The latencies of the PI, PII and PII peaks were unchanged relative to normal values. When analyzing the amplitude parameters, attention is drawn to the phenomenon of an increase in the PIII and PV peaks with significant differences with the group of healthy individuals. The increase in amplitudes was symmetrical in 45% of cases and asymmetric in 55% of cases, which often correlated with the asymmetric degree of ectopia of the cerebellar tonsils. In all our observations with syringomyelitis syndrome, a significant expansion of the inter-peak intervals PIII-PIII and PI-PV compared with the control group was noted, and the interval PI-PV changed to a greater extent. Violations of the parameters of MPI PI-PIII in patients of this group were not registered. Thus, the predominant symmetric expansion of the MIP PI-PV in patients with syringomyelitis syndrome indicated a widespread deceleration of conduction at the level of pontomesencephalic structures. The increase in the amplitudes of the peaks PIII and PV, in our opinion, were signs of irritative disturbances of the upper olivary complex and mesencephalic structures.

Somatosensory evoked potentials are of great importance in the diagnosis of syringomyelitis syndrome in patients with Chiari malformation 1. We analyzed the changes in somatosensory evoked potentials in patients of this group, obtained by stimulating the median and tibial nerves. Table 14. shows the results of our studies of somatosensory evoked potentials in syringomyelitis syndrome to stimulation of n.medianus.

Table 14: Indices of somatosensory evoked potentials during stimulation of the median nerve - latency period, peak amplitudes and inter-peak intervals in healthy controls (n = 30) and patients with syringomyelitis syndrome Chiari malformation 1 (n = 78)

Latency, ms	Controlgroup (n = 30)	Syringomyelitissyndrome (n = 78)
N9 Erba	9.6±0.7	14.4±0.6**
N13 neck	13.2±0.8	20.8±0.8**
N20 cortex	18.8±1.0	21.7±1.1*
Amplitude, μV		
N9 Erba	5.4±2.5	2.0-1.1*
N13 neck	2.9±1.3	1.7±0.8*
N20 cortex	2.8±1.6	2.9±1.5
Peakintervals, ms		
N9-N13	3.5±0.4	6.2±0.5**
N13-N20	5.8±0.5	8.9±1.1**
N9-N20	9.2±0.5	16.8±0.8**

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

As can be seen from the data shown, in patients with Chiari 1 anomaly with syringomyelitis syndrome, reliably significant deviations from the norm in latency indices and amplitudes of components N9, N13, N20 were recorded. The latencies of the N9 and N13 components were significantly increased compared to the control group. In 83% of cases (65 patients), such deviations were asymmetric and did not depend on the degree of ectopia of the cerebellar tonsils. Depression of the amplitudes of the N9 and N13 components was significant in the group of patients with relatively healthy individuals, while the N20 values were practically unchanged compared to normal values. Noteworthy is the significantly significant expansion of the peak-to-peak intervals N9-N13, N13-N20, N9-N20 in the group of patients with syringomyelitis manifestations in relatively healthy individuals. Moreover, MPI N9-N13 and N9-N20 were tightened almost twice as much - up to 6.2ms, 8.9ms, 16.8ms, respectively, from the control values. The expansion of the peak latencies was also asymmetric in 75% of cases.

In table 15. we present the results of somatosensory evoked potentials in patients with syringomyelitis syndrome, Chiari malformation 1, obtained by stimulation of n.tibialis.

Table 15: Indices of somatosensory evoked potentials during stimulation of the tibial nerve - latency period, peak amplitudes and inter-peak intervals in healthy controls (n = 30) and patients with syringomyelitis syndrome Chiari malformation 1 (n = 78)

Latency, ms	Control group (n = 30)	Syringomyelitis syndrome (n = 78)
N 22 lumbar	23.6±1.9	30.4±1.8**
N 30 cervical	30.6±2.5	39.8±1.4**
P37 cortex	37.5±3.4	39.5±1.1*
Amplitude, μV		
N 22 lumbar	1.3±0.5	0.6-0.2**
N 30 cervical	0.9±0.3	0.3-0.2**
P37 cortex	2.6±1.5	0.9-0.1
Peakintervals, ms		
N22-N30	7.62±1.14	18.8±2.3**
N30-P37	8.05±1.32	16.7±0.8**
N22-P37	15.7±1.65	24.4±1.05*

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

The presented data show that the latencies of the N22 and N30 components were significantly increased in the group of patients relative to the control parameters. The latency of the P37 component was relatively unchanged compared to the norm. There was a marked reduction in the amplitudes of the N22 and

N30 components in comparison with the control group. Depression of the amplitudes of the N22 and N30 components was asymmetric in 65% of cases. Deviations of the P37 amplitude from the normal values were insignificant. The most variable were the parameters of the peak intervals N22-N30, N30-P37, N22-P37. The increase in the latency of the MPI was observed in all patients of this group, significantly compared with the control group. The N22-N30 values were increased to a greater extent when the syringomyelitis cyst was located in the thoracic and cervicothoracic spinal cord. The presence of isolated syringmyelia in the cervical spine was characterized by a significant, relatively healthy person, expansion of the N30-P37, N22-P37 MDI with asymmetry on the sides.

Thus, changes in the indices of somatosensory evoked potentials in patients with clinical manifestations of syringomyelitis syndrome with Chiari malformation 1 indicated a violation of segmental afferentation at the level of the cervical and lumbar regions, indicated functional insufficiency of the proximal spinal roots and posterior regions of the spinal structures at these levels. The delay in the central conduction time during somatosensory evoked potentials for stimulation n.medianus et n.tibialis confirmed the presence of both segmental and conduction disorders with involvement of the pontomedullary level.

We carried out electroneuromyography examination of patients with syringomyelia for Chiari 1 anomaly. We registered motor responses obtained during stimulation from the oculomotor, facial and glossopharyngeal nerves, as well as the median and tibial nerves. The research results are presented in Table 16.

Table 16: Electroneuromyography indices for the oculomotor, facial, glossopharyngeal, median and tibial nerves in healthy controls (n = 30) and patients with syringomyelitis syndrome Chiari malformation 1 (n = 78)

Controlgroup (n = 30)	SPI m/s	Amax, μV	Additionalpathologicalwaves
Oculomotornerve	29.4±2.2	1080±105.5	-
Facialnerve	39.5±1.8	1235±126.3	-
Glossopharyngealnerve	42.6±2.0	1860±164.0	-
Mediannerve	61.0±1.7	6254±267.0	-
Tibialnerve	49.6±2.1	7125±745.5	-
Syringomyelitissyndrome (n = 78)			
Oculomotornerve	29.6±2.1	1075±103.8	
Facialnerve	39.2±1.7	1200±118.5	
Glossopharyngealnerve	41.5±1.9*	1730±160.8*	+
Mediannerve	27.4±2.8**	2286±184.5*	+++
Tibialnerve	32.1±1.9**	3850±435.2*	

*Significant differences in identical indicators between the control group and the group of patients (Student t-test) * - P< 0.05, ** - P < 0.01*

In the study of the cranial nerves, a significant decrease in the speed of the efferent impulse along the glossopharyngeal nerve was noted in comparison with the control group. SPI indices for the oculomotor and facial nerves remained unchanged relative to normal values. The amplitudes of muscle responses during stimulation of the indicated cranial nerves were formed and preserved in comparison with the control. In 42% of cases, an isolated decrease in STI along the efferent fibers of the glossopharyngeal nerve was observed when syringomyelia was localized at the level of the upper cervical segments C1-C2 and indicated reactive irritative processes. The decrease in the speed of the impulse conduction along the motor fibers of the median nerve was significant, more often asymmetric in comparison with the control group, more than two times. In the group of patients with syringomyelitis syndrome, significant depression of the amplitude of the n.medianus M-response was recorded relative to the control group. The phenomenon of the appearance of pathological waves of fibrillation, noted at rest and during stimulation of the median nerve in 30 (38%) patients with cervical syringomyelia, requires attention. The STI values for the motor fibers of the tibial nerve in patients of this group were significantly reduced in comparison with normal values. However, the decrease in SPIEff in the lower extremities was less pronounced than in the upper extremities in 52 (66%) patients of this group. The maximum amplitude of the M-response in tibial muscle groups significantly decreased in syringomyelitis syndrome compared with healthy individuals. In the study of n.tibialis, additional pathological potentials characteristic of segmental disorders were not recorded.

Thus, the presence of mixed segmental disorders at the level of the cervical spine, in severe cases involving the anterior spinal structures, is characteristic of the syringomyelitis syndrome in patients with Chiari 1 anomaly during electroneuromyography studies. Conductive disturbances predominated in the

lower extremities, were often symmetrical in nature and were caused by functional disturbances both at the level of the cervicothoracic spinal cord and by a slowdown in pontomedullary conduction.

Neurophysiological data have diagnostic value in determining treatment tactics. Moreover, in the preoperative period, the most significant were the dynamic changes of the latent parameters, including acoustic brainstem evoked potentials and somatosensory evoked potentials of indicators, which indicated a violation of functional conductivity at the level of the pons, medulla oblongata or spinal structures. We evaluated the changes in the indicators of evoked potentials by the degree of conduction disturbance:

- Mild irritation and slowing down of efferent and afferent conduction (deviation up to 20% from the norm)
- Moderate - violation of efferent and afferent conduction (deviation 20-50% from the norm)
- Pronounced - partial or complete block of conductivity (deviation more than 50% from the norm).

Based on the data obtained, evoked potentials, then further treatment tactics were built in patients with Chiari malformation of type 1.

IV. Conclusion

1. In cerebellar syndrome in patients with Chiari malformation of type 1, the most significant diagnostic criteria are an increase in the latencies of the PIII and PV components, as well as the PIII-PV MPI according to the data including acoustic stem evoked potentials, indicating a slowing of conduction at the pontomesencephalic level.
2. For bulbar syndrome, the defining neurophysiological indicators are a decrease in SPI along the glossopharyngeal nerve and pathological waves of fibrillation along the hypoglossal nerve, indicating damage to the structures of the medulla oblongata with involvement of the cranial nerve nuclei.
3. Pyramidal syndrome is characterized by impaired efferent conduction along the median and tibial nerves, more often of a symmetric nature, according to electroneuromyography, and an increase in MPI PIII-PV with a study including acoustic brainstem evoked potentials, indicating a lesion of the intersection of the motor pathways at the level of the craniovertebral junction.
4. Syringomyelitis syndrome with Chiari malformation of type 1 has pronounced changes in the latent parameters of N9-N20 components; N22-P37 with somatosensory evoked potentials, which is caused by impaired afferentation at the pontomedullary level.

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