Comparative analysis of neurophysiological studies in the diagnosis of bulbar syndrome in patients with Chiari malformation Type 1

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SUMMARY

The incidence of bulbar syndrome in craniovertebral junction anomalies is between 12% and 35%. Although the use of evoked potentials intraoperatively and preoperatively has advanced in recent years, their use in predicting the development of neurological deficits remains a challenge. This research explores the predictive significance of evoked potentials in the diagnosis of bulbar syndrome in Chiari 1 anomaly. Data from 39 patients and 30 controls were reviewed**.** Standard multimodal neurophysiological investigations including Brainstem auditory evoked potentials (BAEPs), somatosensory evoked potentials (SSEPs), Nerve Conduction Studies (NCS), and Electromyogram (EMG) were performed. All studies were conducted on the 4-channel complex "Synapsis" (Neurotech, Russia) with computer data processing.

The threshold Nerve conduction velocity (NCV) value was 21.5 m/s. The sensitivity and specificity were 75.5% and 71.2% respectively. The area under the ROC curve (AUC) was 0.96±0.36 (95% CI: 0.89-1.00) and p-value 0.004. The M-response amplitude threshold dividing the study group into high and low-risk groups was 1.01 microV. The sensitivity and specificity were 78.0 and 71.2%, respectively. The BAEPs interpeak intervals III-V and I-V were significantly prolonged ($P< 0.05$). SSEPs showed a decreased amplitude and reduced NCV ($P < 0.01$). Glossopharyngeal nerve electroneuromyography is the most informative test. A decrease in M-response amplitude of bulbar muscles and NCV on efferent fibers is highly predictive of the development of bulbar disorders in patients with Chiari 1, even in subclinical cases.

Key words: Chiari malformation Type 1 – Bulbar syndrome – Brainstem auditory evoked potential (BAEP) – Somatosensory evoked potentials (SSEP) – Electroneuromyography (ENMG)

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INTRODUCTION

The rapid development of modern methods of neuroimaging has led to an increase in the number of patients with diagnosed central nervous system abnormalities (Voronov, 2010; Ismailova, 2020; Levy et al., 1983; Munshi et al., 2000). The modern "gold standard" for diagnosing Chiari malformation is an MRI study. It accurately identifies pathologies of the cranioventral junction, in particular, Chiari malformations, the degree of cerebellar tonsillar ectopia, and the presence of basilar impression (Gushcha et al., 2010; Aronson et al., 1991; Milhorat and Bolognese, 2003). At least a third of Chiari 1 malformations are associated with cervical syringomyelia (OlimjanovnaIsmailova and Kariev, 2020). One of the most dangerous neurological complications of craniovertebral junction abnormalities is bulbar syndrome (Mojaev and Sterlikova, 2009; Sevostiyanov, 2011). The incidence of bulbar syndrome in craniovertebral junction anomalies is between 12 and 35% according to various authors (Aronson et al., 1991; Guo et al., 2007).

Clinical manifestations of the bulbar syndrome in Chiari 1 malformation have been studied by many authors (Sevostiyanov, 2011; Aronson et al., 1991; Møller et al., 1995). Neurophysiological studies have gained popularity in recent years. Their use in intraoperative monitoring during various spinal and cranial surgeries including Chiari 1 surgeries is well studied in the literature (Anderson et al., 2003; Holliday et al., 1985; Moncho et al., 2017; OlimjanovnaIsmailova and Kariev, 2020). Despite their extensive use, whether evoked potentials can be used to predict the development of bulbar disorders in Chiari 1 anomaly is still unclear. Mechanical compression of the brainstem structures by the herniated cerebellar tonsils and abnormalities of CSF dynamics with the formation of a syrinx in the upper cervical region and lower medulla oblongata have been shown to cause local damage to the various tracts and nuclei in the medulla oblongata (OlimjanovnaIsmailova and Kariev, 2020). This causes gross clinical neurological deficits usually picked up on clinical examination. However, in the subclinical cases, evoked potentials have shown promise in making an early diagnosis (Moncho et al., 2017). This research explores the predictive significance of evoked potentials in the diagnosis of bulbar syndrome in Chiari 1 anomaly.

MATERIAL AND METHODS

This was a retrospective study carried out at the Republican Specialized Scientific and Practical Medical Center of Neurosurgery. We analyzed the neurophysiological data of 39 patients with clinical manifestations of the bulbar syndrome in Chiari 1 malformation, who were treated at our institution from 2015 to 2018. The patients' ages ranged from 18 to 65 years, with 11 males and 28 females. These data were compared with data from 30 healthy individuals seen during this period herein called the control group. The standard for determining the degree of cerebellar tonsillar ectopia in Chiari malformation was the Chamberlain line, which runs from the hard palate to opisthion (a point located in the center of the posterior edge of the foramen Magna) (Gushcha et al., 2010; Levy et al., 1983; Milhorat and Bolognese, 2003). Cerebellar tonsillar ectopia more than 5mm below the Chamberlain line was considered significant. In this research, we used the Chamberlain line to determine the presence of the anatomical anomalies of the craniovertebral junction and the degree of cerebellar tonsillar ectopia (Fig. 1).

All patients were investigated according to the multimodal neurophysiological monitoring protocol, including Brainstem auditory evoked potentials (BAEPs), somatosensory evoked potentials (SSEPs), neuro-conduction studies (NCS), and electromyography (EMG) (Isu et al., 1993). All studies were conducted on the 4-channel complex "Synapsis" (Neurotech, Russia) with computer data processing.

Subdermal needle electrodes were used for BAEP recording and were inserted at the vertex to left ear mastoid (Cz/A1); vertex to right ear mastoid (Cz/A2); and vertex to cervical C2 (Cz/Cv2). Stimulation was performed through headphones with 0.1 ms audio clicks with a 20Hz feed frequency and 70dB sound. Filters were set at High Pass/Low Frequency Filter ≥100Hz (-3dB) and Low Pass/High Frequency Filter ≤ 3kHz (-3dB).

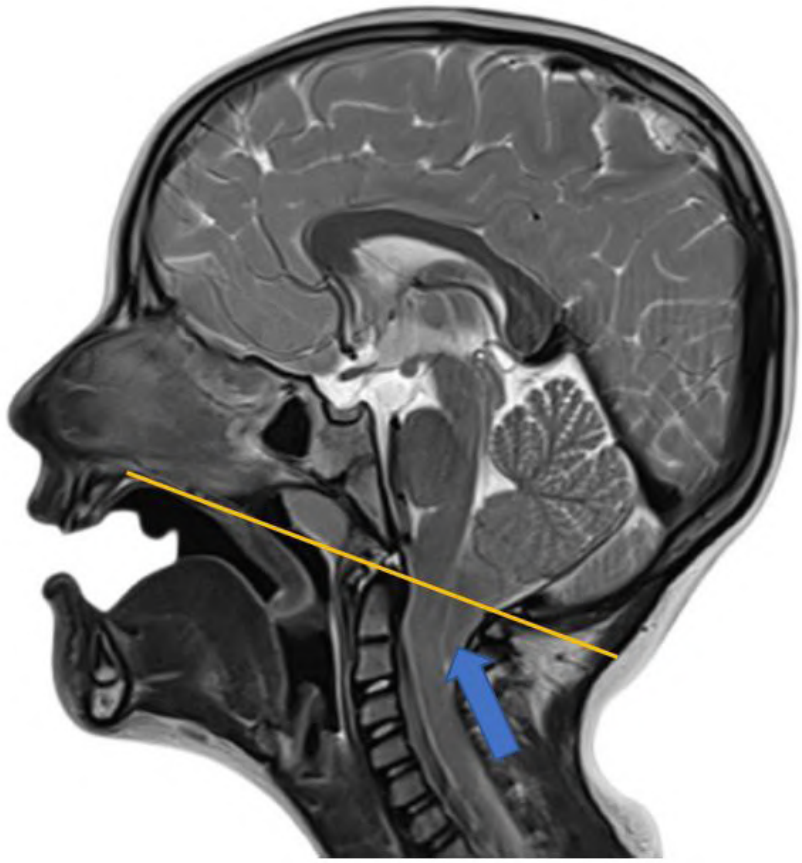

Fig. 1.- MRI showing displacement of the cerebellar tonsils (blue arrow) below Chamberlain's line (orange line) bilaterally.

When recording the SSEPs of the upper limbs, the recording electrodes CP3-Fz or CP3-CP4 for right arm stimulation; CP4-Fz or CP4-CP3 for left-arm stimulation were used. The tests were carried out by electrical stimulation of the median nerve at the wrist level with a current of 15-20 mA and a frequency of 2 Hz.

Lower limb SSEPs were recorded by stimulation of the posterior tibial nerve at the ankle. The traditional derivation of recording is CPz-Fz was used.

The EMG testing was performed on the glossopharyngeal nerve by inserting the recording electrodes in the associated muscles. Where necessary, we analyzed the function of the nerves based on the presence of gross neurological deficits.

Statistical analysis of the data was done using the IBM SPSS Statistics Package version 26. Differences in the distribution of quantitative values were assessed using the student parametric tests. To assess the predictive significance, Receiver operating characteristic (ROC) analysis was used followed by calculation of specificity and sensitivity and area under the ROC curve (AUC).

RESULTS

The BAEPs of all patients with bulbar impairments were analyzed. The data analyzing the latency and amplitude parameters of the study and control groups are presented in Table 1. The latency of the components PIII and PV were moderately prolonged bilaterally compared to the control group. There was a reduction in the amplitudes of peaks III and V. Asymmetric changes in BAEPs were recorded in 76% of the patients examined and a complete absence of PIII and PV components was observed in 24% of cases.

We analyzed SSEP data in 39 Chiari 1 patients with clinical features of the bulbar syndrome. Registration of SSEPs was done by stimulation of the median and tibial nerves in both the study

and control groups. The results of the SSEPs are presented in Table 2. The difference between the control and study group was statistically significant (student t-test *P< 0.05, **P < 0.01). As can be seen from the above data, in the patients with the bulbar syndrome, there was a significant increase in the latency period N13 to 18.4 ms. In the study group, there was a significant decrease in the amplitude of components N13 and N20, mostly bilateral with asymmetry in 61% of the patients.

The peak interval N13-N20 was significantly increased in most patients to 8.0 ms, but the intervals N9- N13 and N9-N20 were only slightly increased relative to normal values. Further, we studied SSEPs, obtained following stimulation of the tibial nerve in Chiari 1 patients with the bulbar syndrome. The data is presented in Table 3.

We identified a significant isolated prolonged N30 latency period to 42.8ms in Chiari 1 patients with the bulbar syndrome, while the latency period of components N22 and P37 were relatively preserved. In these patients, there was a decrease in the amplitude of component N30 to 0.28microV while the amplitude of components N22 and P37 were preserved relative to the control group. A significant increase in the peak interval N30-P37 to 17.8 ms was registered in Chiari 1 patients with the bulbar syndrome with pronounced asymmetry (61%) compared to the control group. However, the peak intervals N22-N30 and N22-P37 showed minor insignificant deviations from the normal.

The Electroneuromyography (ENMG) data was analyzed. The results of MEPs obtained following stimulation of the oculomotor, facial, glossopharyngeal, median, and tibial nerves

Table 1. BAEPs waveforms: Latency period, Peak amplitude, and Peak intervals in the control group (n=30) and Chiari 1 patients with bulbar syndrome (n=39).

Latency period, ms					
	PI.	**PII.**	**PIII.**	**PIV.**	**PV.**
Control Group (n=30)					
Right	1.79± 0.16	2.95 ± 0.18	3.94 ± 0.24	5.06 ± 0.22	5.97 ±0.25
Left	1.72 ± 0.17	2.98 ± 0.19	3.92 ± 0.22	5.13 ± 0.20	6.02± 0.25
Bulbar Syndrome (n=39)					
Right	1.80 ± 0.18	2.98 ± 0.17	4.35 ± 0.25	5.30± 0.21	7.05± 0.22**
Left	1.76± 0.16	3.01 ± 0.20	4.70 ± 0.21**	5.65± 0.19	8.01 ±0.24**

Amplitude, microV			
	PI.	**PIII.**	**PV.**
Control Group (n=30)			
Right	0.286± 0.05	0.262± 0.04	0.368 ±0.06
Left	0.282± 0.04	0.265± 0.06	0.338± 0.08
Bulbar Syndrome (n=39)			
Right	0.348± 0.03	0.050± 0.01**	0.050 ±0.02**
Left	0.340± 0.04	0.180± 0.02*	0.220± 0.04*

Peak intervals			
	PI-PIII.	**PIII-PV.**	**PI-PV.**
Control Group (n=30)			
Right	2.19± 0.16	2.06± 0.18	4.38± 0.22
Left	2.24± 0.18	2.08± 0.22	4.46± 0.24
Bulbar Syndrome (n=39)			
Right	2.36± 0.15	3.96± 0.15**	6.05±0.20*
Left	2.48± 0.17	3.65± 0.20**	6.35±0.21**

are presented in Table 4. The results showed a significant decrease in efferent NCV of the glossopharyngeal nerve in 60% of Chiari 1 patients with bulbar syndrome. In addition to changes in conduction velocity indicators, there was a marked decrease in the amplitude of the M-response of the glossopharyngeal nerve relative to the control group.

The Amplitudes of M-response of the facial and oculomotor nerves showed an insignificant reduction in the study group. Fibrillations were seen in 30% of patients during stimulation of the glossopharyngeal nerve, indicating the involvement of the nuclei. We evaluated the possibility of predicting the development of bulbar syndrome using efferent NCVs of the

Table 2. Median nerve SSEPs: Latent period, Peak amplitude, and Peak intervals in the control group (n=30) and Chiari 1 patients with bulbar syndrome (n=39).

Latency, ms		
	Control Group (n=30)	**Bulbar Syndrome (n=39)**
N9 Peripheral	9.6±0.7	10.1±0.8
N13 Cervical	13.2±0.8	18.4±1.2*
N20 Cortex	18.8±1.0	18.7±1.5
Amplitude microV		
	Control Group (n=30)	**Bulbar Syndrome (n=39)**
N9 Peripheral	5.4±2.5	5.1±2.0
N13 Cervical	2.9±1.3	1.1±0.5**
N20 Cortex	2.8±1.6	1.2±0.4**
Peak intervals, ms		
	Control Group (n=30)	**Bulbar Syndrome (n=39)**
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*

Table 3. Tibial nerve SSEPs: Latency period, Peak amplitude, and Peak intervals in the control group (n=30) and Chiari 1 patients with bulbar syndrome (n=39).

Latency, ms		
	Control Group (n=30)	**Bulbar Syndrome (n=39)**
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, microV		
	Control Group (n=30)	**Bulbar Syndrome (n=39)**
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
Peak intervals, ms		
	Control Group (n=30)	**Bulbar Syndrome (n=39)**
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

Table 4. ENMG indicators for oculomotor, facial, Glossopharyngeal, and tibial nerves in the control group (n=30) and Chiari 1 patients with bulbar syndrome (n=39).

	Efferent NCV, m/s	Amplitude max, microV	Pathological waves
Control Group (n=30)			
Oculomotor Nerve	29.4±2.2	1080±105.5	-
Facial nerve	39.5±1.8	1235±126.3	-
Glossopharyngeal nerve	42.6±2.0	1860±164.0	-
Median nerve	61.0±1.7	6254±267.0	-
Tibial nerve	49.6±2.1	7125±745.5	-
Bulbar Syndrome (n=39)			
Oculomotor	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**	+++
Median nerve	54.5±1.8*	5011±256.5	
Tibial nerve	42.7±1.7	6450±628.5	

glossopharyngeal nerve. Using the ROC analysis method, the graph as shown below was obtained (Fig. 2).

The area under the resulting ROC curve (AUC) was 0.96±0.36 (95% CI: 0.89-1.00), p-value 0.004. The relationship between the amplitude of the M-response of the glossopharyngeal nerve and the presence of bulbar syndrome in patients with Chiari malformation was analyzed. The threshold value of the M- response amplitude separating patients into high and low-risk groups for bulbar syndrome was analyzed using ROC analysis and the results are shown below (Fig. 3).

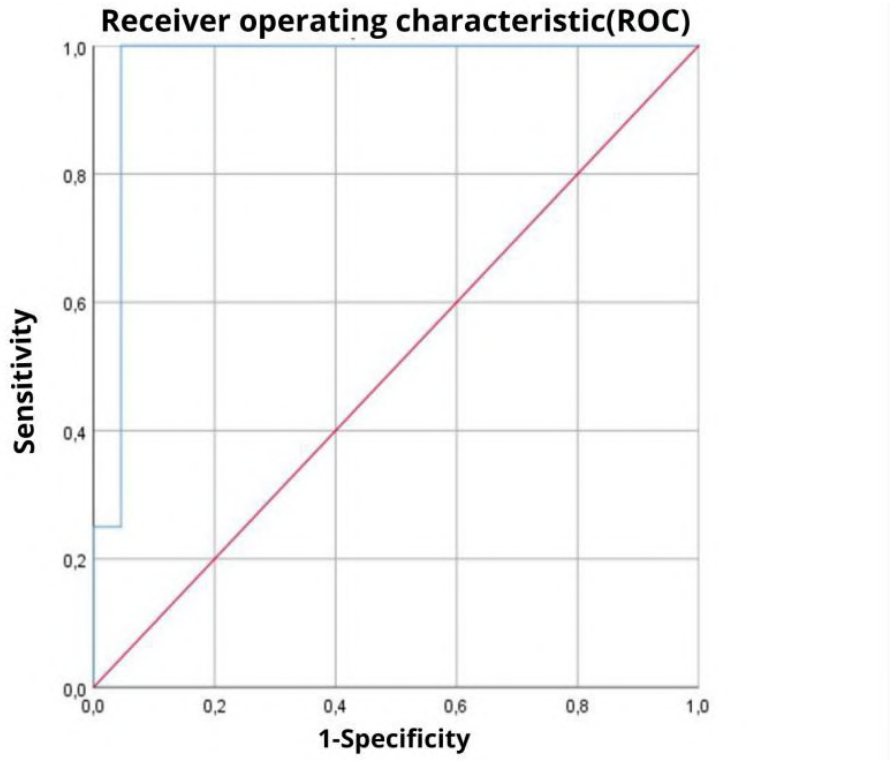

Fig. 2.- ROC curve, assessing the possibility of predicting the development of bulbar syndrome in patients with Chiari type 1, depending on the NCV of the glossopharyngeal nerve.

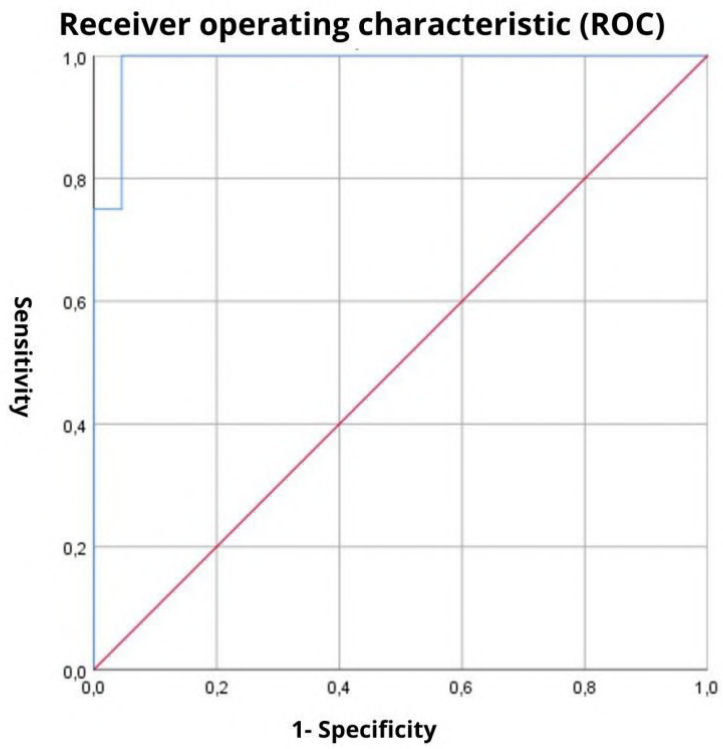

Fig. 3.- ROC curve, outlining the relationship between the development of bulbar syndrome in patients with Chiari 1 and the amplitude of the M-response of the glossopharyngeal nerve.

The resulting ROC curve was characterized by an AUC of 0.98±0.18 (95% CI: 0.95-1.00). There was an inverse relationship between the development of bulbar syndrome and the M-response amplitude p<0,002.

DISCUSSION

The predictive value of BAEPs has been studied extensively in literature with no consensus (Moncho et al., 2017; Holliday et al., 1985). Henriques et al., in their study of 75 patients, concluded that BAEPs are invaluable in the diagnosis, evaluation, and prevention of further morbidity in Chiari patients (Henriques Filho and Pratesi, 2006). The analysis of BAEPs revealed statistically significant changes consistent with conduction abnormalities. The interpeak intervals III-V and I-V were significantly prolonged to almost double in the group with bulbar symptoms compared to the control group. In 24% of patients, interpeak intervals could not be analyzed due to the complete absence of peak III and V components. These changes in BAEPs objectively indicated gross impairment of conductivity at the level of the lower pons and midbrain with clinical manifestations of bulbar syndrome in patients with Chiari 1. These changes were asymmetrical in some cases with dissociated functional involvement of Ponto-mesencephalic structures.

The analysis of SSEPs changes following stimulation of the median and tibial nerves in Chiari 1 patients with clinical manifestations of bulbar syndrome indicated a pronounced slowing of conductivity at the presynaptic level of the medulla oblongata nuclei with decreased nuclear activation. The pronounced slowed conductivity at the pontomedullary level in patients with bulbar disorders was associated with moderate impairment of thalamocortical conduction. This is similar to the results discussed in the literature. The common SSEP abnormalities in Chiari 1 discussed in the literature include a reduction in cortical amplitude from the posterior tibial nerve, a reduction or absence of cervical median nerve potential, and an increased N13-N20 interval (Moncho et al., 2013).

In the analysis of ENMG data obtained from stimulation of the median and tibial nerves, there was a slight reduction of the efferent nerve conduction velocities in the study group compared to healthy individuals. At the same time, the maximum amplitude of the M-response of the median and tibial nerves was practically unchanged compared to the control group. This phenomenon, in our opinion, is associated with the reactive involvement of conductive efferent pathways in Chiari 1 patients with bulbar syndrome with the development of bilateral pyramidal tract involvement. The ENMG was characterized by a pronounced impairment of conductivity at the level of the nuclei of the medulla, often with the involvement of the decussation of the pyramidal pathways. These data allowed us to assess the condition of Chiari 1 patients with bulbar syndrome, even in the subclinical phase of the disease.

The nerve conduction velocity of the glossopharyngeal nerve in the study was the most predictive for bulbar syndrome. In 60% of the patients, the NCV was significantly reduced. It should be noted that the glossopharyngeal NCV was abnormal in patients with mild and subclinical bulbar disorders as well. BAEP and SSEP studies play an important role in incidentally detected Chiari malformation patients and may help to establish objective evidence of subclinical dysfunctions (Moncho et al., 2017). The threshold value of the NCV was 21.5 m/s. Patients with NCV of 21.5 m/s and below had an increased risk of bulbar syndrome, while the risk was lower in patients with NCV values above 21.5 m/s. The sensitivity and specificity of the model at the chosen threshold value were 75.5% and 71.2% respectively. The observed dependence was significant with a p-value of 0.004. The isolated involvement of the glossopharyngeal nerve in Chiari malformation presenting as neuralgia, central sleep apnea, and syncope has been reported in the literature by various authors (Yglesias et al., 1996; Aguiar et al., 2002; Li et al., 2012; Ruiz-Juretschke et al., 2012; Kanpolat et al., 2001). The predictive value of the glossopharyngeal nerve in Chiari 1 malformation with bulbar syndrome has been poorly explored in literature.

The threshold of the M-response amplitude of the glossopharyngeal nerve dividing the study group into high and low-risk groups was 1.01 microV. In patients with M-response 1.01microV and below, the risk of development of decompensated bulbar syndrome was significantly higher than among patients with higher amplitude. The sensitivity and specificity of the model were 78.0 and 71.2%, respectively.

CONCLUSION

The most informative method of determining the risk of development and prognosis of bulbar syndrome in patients with Chiari 1 malformation is Electroneuromyography (ENMG) of the Glossopharyngeal nerve. The decrease in M-response amplitude of bulbar muscles and the decrease in NCV on efferent fibers are highly predictive of the development of bulbar disorders in patients with Chiari 1, regardless of the presence of clinical symptoms.

Ethics Statement

The studies involving human participants were reviewed and approved by Institutional Review Board at the Republican Specialized Scientific and Practical Medical Center of Neurosurgery. The Ethics Committee waived the requirement of written informed consent for participation as this was a retrospective study.

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