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EVOKED POTENTIALS IN GUILLAIN – BARRÉ SYNDROME*

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SUMMARY: Topçu M, Ergin M, Nurlu G, Renda Y, Kanra G, Seçmeer G. (Neurology and Infectious Diseases Units, Department of Pediatrics, and the Department of Neurology, Hacettepe University Faculty of Medicine, Ankara, Turkey). Evoked potentials in Guillain-Barré syndrome. Turk J Pediatr 35: 79-85, 1993.

A prospective evaluation was made in a total of 19 patients with Guillain-Barré syndrome (GBS) by neurologic examination, visual evoked potentials (VEP), brainstem auditory evoked potentials (BAEP) and somatosensory evoked potentials (SEP) during the acute phase of the disease, three weeks later, and then again three months after the onset of symptoms. The results of electrophysiologic studies of the patients were compared with those of 19 healthy children. There were no statistically significant differences found in the mean VEP and BAEP values between the patient and control groups. However, the VEP and BAEP values were abnormal during the first and second stages of the disease in only a few patients. There was a statistically significant prolongation of the response in tibial and median SEP during the first and second stages of the disease when compared with the third stage and the control group. The Erb-Cervical 7 (C7) interwave latency was elevated in the median SEP. It was concluded that the prolongation of the response in SEP was diagnostically important in the acute phase of GBS. There was no correlation found between SEP and clinical progression. *Key words: evoked potentials, Guillain-Barré syndrome.*

Electrophysiologic studies have made significant contribution to the differential diagnosis and evaluation of Guillain-Barré syndrome (GBS). The findings obtained from electroneuromyographic and F-wave studies have demonstrated proximal conduction delays in some patients in the early phase of the disease[1,2]. The SEP technique, which can determine whether there is a block in sensory conduction, is almost as useful as the F-wave[3]. Consequently, it has been suggested that SEP and F-wave studies be used as complementary techniques[4,5]. The evoked potentials of patients with GBS have also been studied with the aim of ascertaining whether there is demyelination of the second and eighth nerves[3,4,6-8]. Although abnormalities in BAEP have been reported[8], some authors are skeptical about correlating abnormalities in BAEP with demyelination of the eighth nerve[3].

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The purpose of this study is to investigate the evoked potentials of the patients with GBS through the course of the disease and to correlate these values with the clinical findings for determining prognosis.

Material and Methods

Twenty-one patients with GBS were admitted to Hacettepe University Children's Hospital between November 1988-March 1990 for prospective evaluation. Evoked potential responses could not be obtained in two patients, who died shortly after a fulminating course of the disease. Those patients were excluded from the statistical evaluation. A study was made of the remaining 19 patients, sixteen of whom were re-evaluated three weeks after the acute phase of the illness and 11, three months after the acute phase by neurologic examination, VEP, BAEP and SEP. One patient was diagnosed as having Fisher syndrome. Eleven age and height-matched healthy children were chosen as the controls. Muscle weakness was assessed according to the grading criteria used by the GBS Study Group[9]. The motor disability grading criteria devised by this group is as follows;

Grade 0 Healthy.

Grade 1 Minor signs and symptoms.

Grade 2 Able to walk five meters without requiring a walker or equivalent support.

Grade 3 Able to walk five meters with a walker or support.

Grade 4 Bed or chairbound (unable to walk five meters with a walker or support).

Grade 5 Requires assisted ventilation (for at least part of the day).

Grade 6 Deceased.

VEP was performed by placing the electrodes according to 10-20 in the international EEG system (active at Oz, reference at Fz), and pattern stimulation was repeated 128 times. The mean on the waves with a frequency of 1 to 100 Hz was obtained. While evaluating VEP results, the P100 value was calculated as latency and the N1-P1 extreme points were calculated as the amplitude. Before BAEP was performed, the electrodes were placed according to 10-20 in the international EEG system (active at mastoid, reference at Cz) and the stimulation was repeated 1024 times. The amplitude of the sound in the stimulated ear was 75 decibels, and 45 decibels of masking sound was applied to the contralateral ear. The principal evaluation was based on I-V interpeak latency and first wave latency.

SEP was performed by stimulation of the median and the posterior tibial nerves. The electrodes were placed according to 10-20 in the international EEG system (active at Cc, scalp overlying the sensoriparietal cortex 2 cm behind the 10-20

system C3/C4 positions C7, erb and below, reference at Fz for median, active at Cz, reference at Fz for tibial). Vertex P1 peak latency was used in tibial SEP, while the latencies used in median SEP were C7 and Cc peak latencies and interpeak latencies between elbow-erb, erb-C7, and C7-Cc.

Values having a difference of more than 3SD from the controls were considered abnormal.

Results

The general features of the patients are presented in Table I. Prolongations of VEP values were detected in five patients (patients 4, 5, 6, 10, 17) during the acute phase, in three patients (patients 4, 12, 15) during the second examination, and in one patient (patient 4) during the third examination. Patient 4, diagnosed as having

Table I: General Features of Patients with GBS

Patient Number	Age (yrs)	Sex	Height (cm)	Onset of Symptoms (day)	CSF Protein (mg)	Motor Deficit 1.	2.	3.
1	13	F	144	11	240	3	1	1
2	11	M	142	7	240	4	1	0
3	8	F	118	6	144	5	3	0
4*	8	M	120	5	48	2	1	0
5	6	M	108	1	24/168	4	4	2
6	5	M	103	2	72	3	3	2
7	5	M	103	4	36/96	3	2	1
8	4	F	91	5	60/96	4	3	2
9	3	F	95	1	48/90	4	3	1
10	2	F	81	1	169	3	1	0
11	2.5	M	84	10	168	4	3	2
12	2	M	85	6	72	3	3	
13	14	M	162	4	48/108	3	3	
14	13	M	146	2	190	3	1	
15	9	F	121	7	48	4	2	
16	4	M	90	12	504	4	3	
17	4	F	89	6	208/120	4		
18	13	M	140	4	144	4		
19	9	M	122	7	121	3		
20	10	F	130	4	168	6		
21	10	M	135	3	72	6		

* Fisher syndrome.

Fisher syndrome, demonstrated prolongation of conduction time in all three measurements. There was no statistical significance in VEP P100 latency values between the patients and the controls.

An increase in the first wave peak latency was observed in only one patient (patient 10) during the acute phase. Elevations in interpeak latencies were detected in one patient (patient 10) during the acute phase, in one patient (patient 15) during the second examination, and in one patient (patient 10) during the third examination. When we compared the first wave BAEP values with the I-V interwave latency values of the two groups, we found that there were no statistically significant differences between the patients and the controls.

Tibial SEP Vtx peak latencies of five patients during the acute phase (patients 3, 10, 13, 15, 17), and in five patients during the second examination (patients 2, 3, 5, 10, 15) showed prolonged latencies. The differences between the patient and control groups during the first and second examinations were statistically significant ($p < 0.05$), but there was no statistically significant difference in the third measurement (Table II).

Table II: Statistical Comparison of Tibial SEP Vertex Peak Latency Values Between Patient and Control Groups

	Vtx PL (msec)	
Group	X	SE
Patient I	42.26	1.54
Patient II	42.70	1.88*
Patient III	38.24	0.82
Control I (19)	36.54	0.61
Control II (16)	36.34	0.64
Control III (11)	36.13	0.73*

* : $p < 0.05$ and significant; p values were calculated with respect to the difference between the means of the two groups.
SE : Standard error.
X : Mean.
Vtx : Vertex.
PL : Peak latency.

None of the values of median SEP Cc peak latencies in the patients was more than 3 SDs. The differences between the patient and control groups during the first and second examinations were statistically significant ($p < 0.05$) (Table III), but no abnormal values for mean C7 peak latencies were noted in the patients and there was also no significant difference between the patient and control groups

during the acute phase. The values of the patients progressively improved in consecutive examinations (Table III). Although no abnormal values of elbow-erb interpeak latencies were detected in the patients, there were significant differences between the patient and control groups during the first and second examinations (Table III). Mean Erb-C7 interpeak latencies in three patients during the acute phase (patients 3, 15, 19), and in one patient during the second examination (patient 3) revealed prolonged values. The difference between the acute phase and the other phases, and the difference between the patient and control groups in respect to first and second examinations were statistically significant (Table III).

Although there were no abnormal values for mean C7-Cc interpeak latencies in the patients, the values during the acute and other phases of the disease, and the values of the patient and control groups during the first and second examinations were significantly different (Table III).

Table III: Statistical Comparison of Median SEP Values in Patient and Control Groups

	Cc PL (msec)		C7 PL (msec)		Elbow-Erb IPL (msec)		Erb-C7 (msec)		IPL C7-Cc I (msec)	
	X	SE	X	SE	X	SE	X	SE	X	SE
Patient I	17.05	0.29*	10.95	0.36	3.68	0.13*	3.64	0.24*	6.05	0.1
Patient II	16.30	0.43*	10.35	0.40	3.65	0.14*	3.28	0.24*	5.90	0.1
Patient III	15.64	0.51	9.80	0.45	3.55	0.16	2.96	0.25	5.80	0.2
Control I (19)	15.20	0.39	9.80	0.33*	3.38	0.16	2.60	0.15*	5.41	0,
Control II (16)	15.01	0.45*	9.68	0.38*	3.33	0.17	2.56	0.17*	5.35	0,
Control III (11)	14.89	0.52*	9.75	0.42	3.31	0.19	2.56	0.19	5.16	0,

* : $p < 0.05$ and significant; p values were calculated with respect to the difference between the means of the two groups.
SE : Standard error.
X : Mean.
PL : Peak latency.
IPL : Inter peak latency.

Discussion

Although there are published reports about evoked potentials in GBS, all these studies have been conducted during the acute phase. To our knowledge no investigations have been made during the later phases of the disease.

In our VEP and BAEP studies, we found no statistically significant differences between the patient and control groups in all three stages of the disease. There

was a prolongation of P100 latency in all three measurements taken of the patient with Fisher syndrome. Rudolph et al[10] have detected abnormal BAEP values in Fisher syndrome, and Ropper and Chiappa[3] have found unilateral prolongation of I-V interpeak latency in one of three patients with Fisher syndrome. In our patient with Fisher syndrome, the BAEP and SEP values and central conduction time were within normal limits. The increase in VEP values in this patient may be due to a possible demyelination of the optic nerve. Abnormal BAEP implications were obtained in two of our patients. An increase in interpeak latencies was detected by Ropper and Chiappa[3] in three of 21 patients, and by Schiff et al[7] in five of six patients, two of whom were in the convalescent phase. It was observed that prolongation of latencies detected in our study had no relation to the duration of disease. With these results, it is difficult to say that GBS was limited to the peripheral nerves.

SEP has become a subject increasingly dwelt upon because it provides information about the conduction velocity of both the distal and proximal parts of the peripheral nerve and the central conduction velocity in GBS[3-6].

Median, tibial, peroneal SEP and F wave latencies have been investigated in the same studies[4,5]. SEP and the F wave provide valuable information in the diagnosis of GBS by detecting abnormalities in proximal nerve conduction [4,5]. Brown and Feasby[6] have reported that of 11 patients with GBS in the acute phase, ten had delays in proximal conduction and three had prolongation of cortical potential latencies detected by median SEP.

When we analyzed the SEP results obtained in our study, we found that five patients in the acute phase and seven patients during the third examination revealed abnormal tibial SEP results. The values obtained were over 2 SDs in a significant proportion of patients with nine patients in the acute phase. We believe that tibial SEPs yielded more meaningful results than did median SEPs.

In addition to the abnormal values obtained, the results of tibial and median SEP during the acute phase and three weeks later were significantly longer than in the control group and the values detected three months later in the patient group.

Abnormalities in median SEP C7-Cc interpeak latencies were detected in three patients, and statistically significant values of proximal sensory and cortical latencies compared to the control group and to values in the convalescent phase, which suggest that the disease is not limited to peripheral nerves and proximal parts, and that especially in the acute phase, the central nervous system can also be affected.

Although it was not possible to make a statistical correlation between SEP values and clinical staging because of technical reasons and an insufficient patient, number, it was observed that the prolongations detected by SEP persisted

throughout the acute and subacute phases, and that the latencies returned to normal after three months. It was also noted that muscle strength of the patients improved in three weeks, which was evident also during the last examination. However no correlation could be found between clinical improvement and normalization of SEP values.

We conclude that SEP is an important tool to diagnose GBS in the early phase, and that it should be used with other electrophysiologic methods of diagnosis.

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