The Seoul Journal of Medicine
Vol. 33, No. 1: 57-64, March 1992

Significance of Brainstem Auditory Evoked Potentials as an Initial Evaluation for Dizziness†

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=Abstract=**The authors performed brainstem auditory evoked potential (BAEP) studies on 207 patients with dizziness symptoms, and evaluated the significance of BAEPs in differentiating among various causes of dizziness.**

The results showed abnormal BAEPs, which were suggestive of brainstem dysfunction, in 20 of 106 of the probable vertebrobasilar transient ischemic attack (VB TIA) group (18.8%), and in 12 of 101 of the vague dizziness (VD) group (11.8%). Additionally, there were abnormal BAEPs, which were suggestive of the end-organ dysfunction, in 4 of 101 of the VD group (3.9%). When we analysed 32 abnormal BAEPs suggestive of brainstem dysfunction, the most frequent BAEP abnormality was the prolongation of I-III interpeak latency (IPL) (53.1%). Prolonged I-V IPL was the second most common abnormality (28.2%), with III-V IPL prolongation occurring less commonly (18.7%). The follow-up studies of abnormal BAEPs showed that the initial abnormal BAEPs reverted to normal in three of six patients, but in the remaining three the abnormality persisted during follow-up period of one to four years.

Therefore, it is concluded that BAEP tests would be useful in differentiating the dizziness as one of those symptoms of brainstem dysfunction from non-brainstem syndrome which mimics it.

Key Words: *Brainstem auditory evoked potential (BAER), Dizziness, Vertebrobasilar transient ischemic attack*

INTRODUCTION

Brainstem auditory evoked potentials (BAEPs) have been used for the evaluation of many neurologic diseases affecting the brainstem as they provide a sensitive tool for the assessment of brainstem functions (Jewett and Williston 1971). It has been established that BAEPs are abnormal when there are anatomic or physiologic dysfunctions along the auditory pathway or in nearby structures (Oh *et al.* 1978; Chiappa 1987).

The clinical usefulness of BAEPs has been reported in strokes and in other vascular ischemic attacks of the brainstem (Maurer *et al.* 1979 ; Brown *et al.* 1981 ; Raggazoni *et al.* 1982). There have been several reports that abnormal BAEP results were well correlated with structural lesions in the brainstem by neuroimaging studies (Faucht and Oh 1985; Kim *et al.* 1987). Recently Factor and Dentinger (1987) reported that BAEPs were all abnormal

Received January 1992, and in final form March 1992.

† This study was supported by a grant from Seoul National University Hospital (1991).

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in patients with vertebrobasilar transient ischemic attack (TIA) one to sixteen days after the attacks. However Lee *et al.* (1987) have reported that abnormal BAEPs were found in fourteen of thirty six cases with vertebrobasilar TIA (38.8%). The authors thought that this discrepancy would be caused by the time interval after the attack as well as by the diagnostic criteria of vertebrobasilar TIA (Factor and Dentinger 1987).

In this study, the authors performed the BAEP tests on patients, whose dizziness was one of their chief complaints, and evaluated the significance of BAEPs for differentiating the dizziness as one of those symptoms of brainstem dysfunction from other causes of dizziness.

MATERIALS AND METHODS

207 patients with dizziness were studied at the Neurophysiological Laboratory, Seoul National University Hospital from July 1986 to September 1990. The one group consisted of 106 patients who were considered to have probable vertebrobasilar TIAs because they did not fulfil the diagnostic criteria of definite vertebrobasilar TIAs, which were defined as episodes that were sudden in onset, lasted 24 hours or less, and consisted of two or more signs and symptoms referrable to the brainstem (Toole 1984). They said that the patients in the probable VB TIA group did have sudden onsets of dizziness and one of the symptoms, such as blurred vision, slurred speech or abnormal sensation in one part of the body.

The other group consisted of 101 patients whose chief complaint was only of vague dizziness. These symptoms were relatively transient, repeated, and moderately aggravated with positional changes. The authors classified those patients in the vague dizziness (VD) group, because these nonspecific symptoms might be caused by the dysfunctions of the brainstem, vestibular neuronal systems, or other nonvestibular dysfunctions in the body.

The BAEP tests were performed two to four weeks after the dizziness attacks. Those 6 patients whose initial BAEP was abnormal were followed up during the period of treatment. Neuroimage studies such as brain MRI were also done on patients who showed sequential abnormal BAEPs and who did not improve so much with medical treatment.

BAEP methods

BAEP tests were performed using by the standard methods of the Neurophysiological Laboratory, Seoul National University Hospital which have been described elsewhere (Lee *et al.* 1987 : Kim *et al.* 1987 : Ha *et al.* 1988 : Park *et al.* 1988 : Lee *et al.* 1989 : Kim *et al.* 1989). Briefly, the BAEPs were tested using a Nicolet Pathfinder II machine. The BAEPs were recorded simultaneously from ipsilateral and contralateral mastoid processes (Mi or Mc) referred to the central vertex (Cz). Ground electrodes were placed on the forehead (FPz).

On each side, the click stimuli were presented at a rate of 11.1 per second for the duration of 100 usec. The click intensity was 60 dB above the patient's hearing threshold and the contralateral ear was masked with white noise at 40 dB less than the intensity of the presenting click stimuli. The potentials of more than 100uv were rejected automatically.

The responses were amplified to a gain of 10^4 and filtered with a band pass of 150 to 3,000 Hz. More than 2,000 trials were done each time, and two obtained responses were superimposed to demonstrate the reproducibility.

The BAEP results were interpreted by measuring the absolute latencies of wave I, II, III, IV, V, interpeck latencies (IPL) of I-III, III-V & I-V and amplitude ratio of peaks V:I. In addition, wave morphology and asymmetries were considered if the above measurements were normal. The results were considered abnormal when the measures exceeded the value above the mean plus 2.0 standard deviations.

RESULTS

36 of 207 patients revealed abnormal

findings at the initial BAEP tests. When we divided those 207 cases into the probable vertebrobasilar TIA (probable VB TIA) group and the vague dizziness (VD) group, abnormality was found in 20 of 106 in the former 8%), and 16 of 101 in the latter (15.8%). The BAEP findings were suggestive of brainstem dysfunctions in 20 cases in the probable TIA group (18.8%) and 12 in the VD group (11.8%). In 4 cases in the VD group, the BAEP abnormalities were compatible with end organ dysfunctions. In this study, the overall rate of abnormal BAEPs was 17.4%, but the abnormality which was suggestive of brainstem dysfunction was only 15.4%.

In the probable VB TIA group the abnormal features were bilateral in 12 and unilateral in 8 out of 20 abnormal patients (Table 1). The six showed abnormal I-III or I-V interpeak latencies without any prolonged absolute latencies of wave I, II, III, IV, V. The remaning fourteen revealed prolonged I-III, III-V or I-V interpeak latencies with the prolonged latencies of wave III or V. The abnormal BAEPs were indicative of lower brainstem dysfunctions in 11, upper in 5, and diffuse in 4. In two of the latter four, the abnormalities were prolonged I-V interpeak latencies without I-III or I-V abnormality.

Among the 12 patients suggestive of brainstem dysfunction in the VD group, the BAEP abnormalities were bilateral in 5 and unilateral in 7. The ten showed abnormal I-III or I-V interpeak latency without any prolonged absolute latencies of wave I, II, III, IV, V. However in the remaining two the I-V interpeak latencies were prolonged with prolonged absolute latencies of wave III & V. The abnormal BAEPs were indicative of lower brainstem dysfunction in 6, upper in 1, and diffuse in 5. The other 4 subjects showed abnormal BAEPs which were suggestive of end organ dysfunction (Table 2).

Table 1. Data of abnormal BAEP findings in 20 cases from the probable VB TIA group

Case No	Sex/Age	Right			Left		
		I-III	III-V	I-V	I-III	III-V	I-V
1	M/59	2.40	2.08	4.48*	2.44*	1.96	4.90*
2	M/72	2.30	2.16	4.46*	2.24	2.16	4.40*
3	F/49	2.28	2.50*	4.78*	2.34	2.56*	4.90*
4	M/69	2.40	1.76	4.16	2.44*	1.84	4.28
5	F/35	2.76*	1.90	4.66*	2.12	1.88	4.00
6	M/41	2.62*	1.92	4.54*	2.46*	2.06	4.52*
7	M/66	2.38	1.98	4.36	2.22	2.26*	4.48*
8	M/48	2.42	2.18	4.60*	2.30	2.52*	4.82*
9	F/48	2.48*	2.48*	4.96*	1.98	1.76	3.74
10	M/57	2.30	1.78	4.08	2.76*	1.92	4.68*
11	M/40	2.30	1.98	4.28	2.56*	2.00	4.56*
12	M/60	2.36	2.02	4.38	2.46*	2.06	4.52*
13	M/35	1.86*	2.12	3.98*	2.66*	1.94	4.60*
14	M/60	2.36	2.10	4.46*	2.20	2.10	4.30
15	M/46	2.42	1.96	4.38	2.66*	1.80	4.46*
16	M/34	2.16	2.28*	4.40*	2.10	2.12	4.22
17	M/57	2.48*	1.82	4.30	2.36	1.86	4.22
18	M/49	2.24	1.96	4.20	2.64*	1.88	4.52*
19	F/64	1.94	2.32*	4.26	2.22	2.14	4.36
20	M/55	2.32	1.88	4.20	3.18*	1.72	4.90*

*: 〉 mean value + 2.0SD

Table 2. Data of abnormal BAEP findings in 16 cases from the VD group

Case No	Sex/Age	Right			Left		
		I-III	III-V	I-V	I-III	III-V	I-V
1	M/67	2.26	2.24	4.50*	2.00	2.30*	4.34
2	F/55	2.46*	2.20	4.68*	2.40	2.02	4.42*
3	F/65	2.30	2.12	4.42*	2.24	2.22	4.46*
4	M/67	2.28	1.92	4.20	2.40	2.08	4.48*
5	M/33	2.16	2.12	4.28	2.42	2.18	4.60*
6	M/57	2.12	2.14	4.26	2.50*	2.20	4.70*
7	M/55	2.32	1.84	4.16	2.48*	1.80	4.28
8	F/45	2.46*	1.62	4.08	2.38	1.90	4.28
9	M/54**	2.38	1.80	4.18	2.22	1.68	3.90
10	M/57	2.20	2.20	4.40*	2.24	2.02	4.26
11	M/55	2.42	2.02	4.44*	2.42	1.92	4.34
12	M/79**	2.40	1.84	4.24	2.32	1.78	4.10
13	M/53**	2.14	2.00	4.14	2.34	1.76	4.10
14	M/48**	2.22	2.08	4.30	2.08	1.92	4.00
15	F/76	2.50*	1.82	4.32	2.50*	1.52	4.02
16	M/40	2.72*	1.88	4.60*	2.86*	1.88	4.74*

*: 〉 mean value + 2.0SD

**: end-organ dysfunctions

In cases 12,13 and 14 of the VD group, the absolute latencies of wave I,III,V were prolonged with normal I-III, III-V, I-V interpeak latencies. In case 9, the morphology of BAEP potentials was very poor and considered to be typical of peripheral auditory dysfunctions.

In summary, 32 patients of 207 showed abnormal BAEPs which were suggestive of brainstem dysfunction. The BAEP abnormalities were bilateral in 17 (53.1%) and unilateral in 15 (46.9%). When we determined the level of brainstem dysfunction in terms of abnormal BAEPs, there were lower brainstem dysfunctions in 17 (53.1%), upper in 6 (18.7%), and diffuse in 9 (28.2%). However there was no clear preponderance of brainstem lesions between the probable VB TIA and the VD group.

The authors did follow BAEP tests in six patients whose initial BAEPs were abnormal. As shown in table 3, the abnormal BAEPs were suggestive of lower brainstem dysfunction in cases 3,4 and 5 as the major abnormalities

Talbe 3. Summary of six patients with follow-up BAEP studies

Sex/ Age	Sxs & Signs	Time of BAEPs	Right			Left		
			I-III	III-V	I-V	I-III	III-V	I-V
1. M/59	Dizziness.	Initial	2.40	2.08	4.48*	2.44*	1.96	4.40*
	Facial	6mos later	2.36	2.18	4.54*	2.36	1.98	4.34
	numbness	12mos later	2.36	2.16	4.52*	2.52*	1.76	4.28
2. M/55	Vertigo	Initial	2.32	1.88	4.20	3.18*	1.72	4.90*
	Facial	1.5yr later	2.20	1.82	4.02	2.60*	2.20	4.80*
	numbness	3.5yr later	2.28	1.78	4.06	2.70*	1.96	4.66*
3. M/57	Dizziness,	Initial	2.12	2.14	4.26	2.50*	2.20	4.70*
	Tinnitus	1yr later	2.10	2.14	4.24	2.38	2.18	4.56*
		2yr later	2.16	2.10	4.26	2.26	2.04	4.30
4. M/55	Dizziness.	Initial	2.32	1.84	4.16	2.48*	1.80	4.28
	Nystagmus	3mos later	2.34	1.80	4.14	2.36	1.78	4.14
5. M/45	Vague	Initial	2.46*	1.62	4.08	2.38	1.90	4.28
	Dizziness	9mos later	2.18	1.80	3.98	2.22	1.88	4.10
6. M/40	Dizziness.	Initial	2.72*	1.88	4.60*	2.86*	1.88	4.74*
	Tinnitus	1.5yr later	2.40	2.04	4.44*	2.56*	1.94	4.50*
		4.5yr later	2.46*	1.86	4.32	2.74*	1.82	4.56*

were prolonged I-III interpeak latencies. In case 3, the follow-up BAEPs one year later showed only the prolongation of I-V interpeak latencies, and the third BAEP became normalized (Fig. 1). However in cases 1, 2 and 6, there were no definite interval changes of abnormal BAEPs in spite of clinical improvements. The BAEP abnormalities were indicative of functional distur-

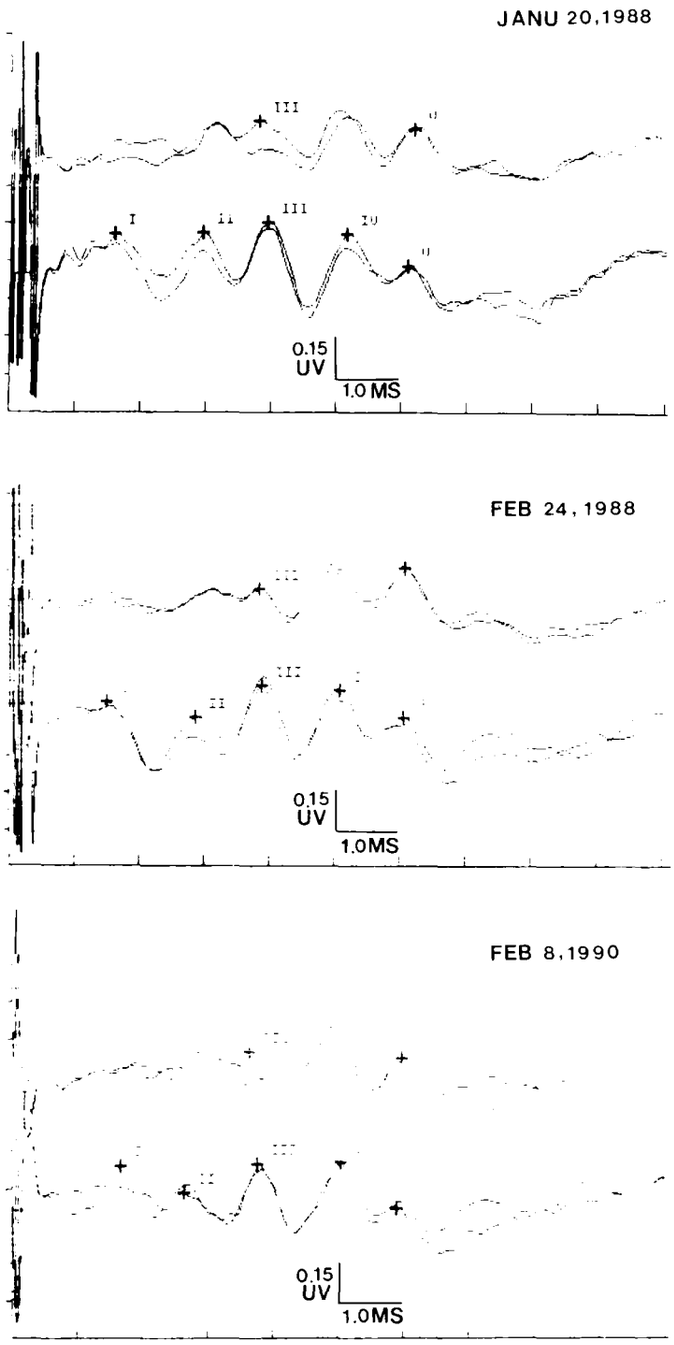

Fig. 1. The follow-up studies of left BAEPs in patient 6 of the VD group. Initial BAEPs revealed prolonged I-III and I-V interpeak latencies and reverted to normal one year later.

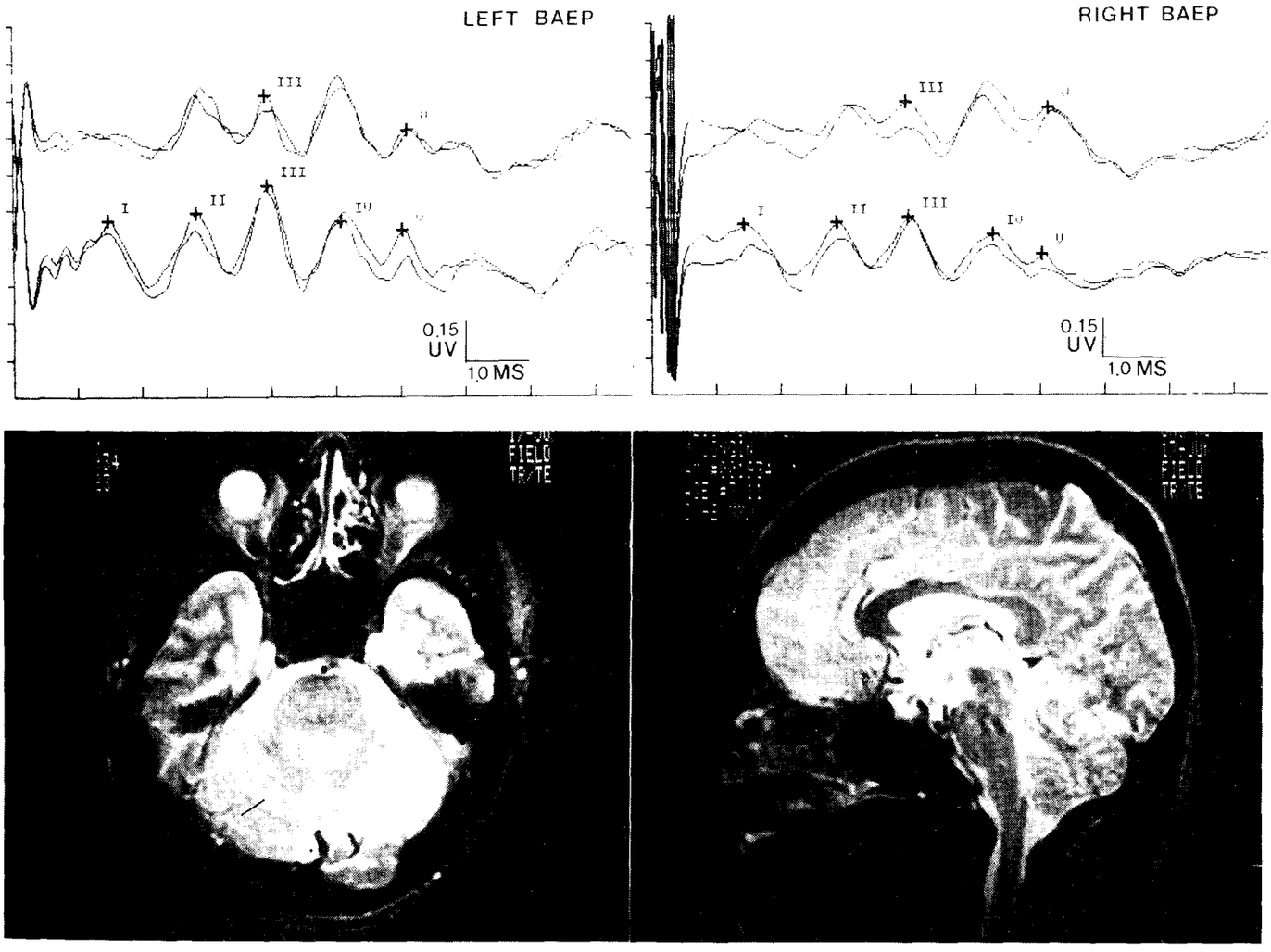

Fig. 2. The abnormal BAEP studies in patient 20 of the probable VB TIA group. The left BAEP definitely showed prolonged I-III and I-V interpeak latencies although the T2 weighted brainstem MRI was within normal limits.

bances of the lower brainstem although there were no definite abnormal features in the brainstem MRI in cases 1 and 2 (Fig. 2).

DISCUSSION

The vague term "dizziness" is one of the commonest of the symptoms which confront the clinical neurologist. The patient applies "dizziness" to a number of different sensory experiences, such as a feeling of rotation, nonrotatory swaying, syncope, faintness, light headedness, unsteadiness, blurring of vision, or even petit mal (Adams and Victor 1989).

There are several mechanisms responsible for the maintenance of posture and for the awareness of the position of the body in relation to its surroundings. Space constancy can be maintained by the neural mechanisms through afferent and efferent pathways. The most important afferent impulses are from the retina of both eyes, from the labyrinth, and from the proprioceptors of the joints & muscles. These sense organs are connected with the cerebellum, the ganglionic centers and the pathways in the brainstem, particularly the oculo motor, red, vestibular nuclei and the medial longitudinal fasciculi. It is known that these are the important coordinators of the sensory data provided for postural adjustment and the maintenance of equilibrium.

It would not be difficult to separate the true

vertigo of the vestibular type from the dizziness of non-vestibular origin by careful history and physical examinations. Also, several criteria have been used to differentiate the vertigo of brainstem origin from the peripheral causes of vestibular system on clinical ground (Baloh and Honrubia 1990).

In our study, 106 subjects was classified into the probable VBTIA group as they showed several symptoms & signs associated with vestibular nuclei and its connections. The other 101 subjects in the VD group complained of dizziness like giddiness or dysequilibrium. Nobody had clinical symptoms and signs typical of vestibular neuronitis, benign positional vertigo, Meniere's syndrome, or tumorous lesions. Thus the majority of the VD group could be characterized as having various degrees of vascular insufficiency through the fine branches of the vertebro-basilar artery, posterior inferior cerebellar artery, or anterior inferior cerebellar artery, and so on. Some subjects in this group would be those with mild brainstem dysfunction, with vestibular dysfunction of peripheral type or with dizziness of nonvestibular origin.

In this study abnormal BAEPs were found in 20 of 106 subjects in the probable VB TIA group (18.8%). In the literature there were conflicting results of the BAEP studies of brainstem lesions of vascular origin, especially on vertebrobasilar TIA. Chiappa (1987) found normal BAEPs in patients with TIA of brainstem structure unless there were permanent deficits in personal experience. Rizzo *et al.* (1983), reported a study of 16 patients and concluded that 36.8% of initial BAEPs were abnormal. Rossi *et al.* (1983) found 32% of 50 subjects to have abnormal BAEPs. Our previous results (Lee *et al.* 1987) showed abnormal BAEPs in 14 of 38 definite VB TIAs (38.8%), which was similar to the results of Rizzo *et al.* (1983) and Rossi *et al.* (1983). However abnormal BAEPs were reported to be 53%, 100% or 61. 8% respectively in others studies (Raggozzoni *et al.* 1987; Factor and Dentiger 1987; Park *et al.* 1990).

Factor and Dentinger (1987) said that the major reasons for the conflicting data were the selection of material and the methods of the BAEP studies. Their definition of VB TIA was more restrictive requiring at least two symptoms & signs, and the BAEP studies were usually performed earlier than seven days after the TIA. In our previous study (Lee *et al.* 1987), the diagnostic criteria of VB TIA were almost the same as those of Factor and Dentinger (1987), but the results showed abnormal BAEPs to be 38. 8%. However, in the present studies the authors thought that the ratio of abnormal BAEP (18. 8%) in the probable VB TIA group was reasonable, because the diagnostic criteria were less restrictive and the BAEPs were performed two to four weeks after the attack.

It was interesting that there were abnormal BAEPs suggestive of brainstem dysfunction in 12 of 101 subjects in the VD group (11.8%). All subjects in this VD group did not have any symptoms suggestive of brainstem lesions, except for vague dizziness. The authors theorized that these subjects with abnormal BAEPs might have subclinical circulatory insufficiency of the brainstem due to a dysautoregulation of brainstem blood flow. There were also abnormal BAEPs which were indicative of end-organ dysfunction of the vestibular system in 4 of 101 subjects in the VD group (3.9%). However, we wondered whether there was any circulatory deficiency through the terminal branches of the anterior inferior cerebellar artery, ie, the labyrinthine artery, or degenerative changes in the vestibular system.

When we analyzed 32 abnormal BAEPs suggestive of brainstem dysfunction, there were 17 bilateral abnormalities (53.1%) and 15 unilateral (46.9%). These data were similar to our previous reports in which 52.7% showed bilateral abnormal BAEPs. The most frequent abnormal BAEPs were the prolongation of I-III interpeak latencies (53.1%) which were suggestive of lower brainstem dysfunction. The BAEP abnormalities suggestive of upper brainstem dysfunction were found in only 6 of 32 subjects (18.7%) and the remaining subjects (28.2%) showed diffuse form of brainstem dysfunctions. Those findings contradicted to those of

Ragazzoni *et al.* (1982) in which the prolongation of I-V interpeck latencies were found in all patients with abnormal BAEPs and the prolonged interpeak latencies III-V were the second most common abnormality, with I-III prolongation occurring less commonly.

The follow up studies of abnormal BAEPs were inconclusive. In three of six patients (cases 3,4 and 5) the initial abnormal BAEPs reverted to normal after certain periods of time. On the contrary, abnormal BAEPs persisted in cases 2 and 6 during the follow-up period of one to four years. Ragazzoni *et al.* (1982) evaluated four subjects with VB TIA and found that none had recovered. In Rossi's report (1983), they found that the abnormal BAEP reverted to normal in 5 out of ten, but concluded that the BAEP abnormalities in the VB TIA tend to persist. However Factor and Dentinger (1987) reported that five of six subjects showed reversal of BAEP changes to normal. So the authors postulated that circulatory insufficiency in the brainstem would be subnormal or borderline for a long period of time after the VB TIA attack.

In conclusion, the authors believe that the BAEP tests would be helpful in differentiating dizziness of brainstem lesion from non-brainstem syndrome. The BAEP could definitely be indicated in patients with definite as well as probable VB TIAs in which the brain MRI would be within normal limits. Among 101 subjects in the VD group, there were abnormal BAEPs in 16 (15.8%), which were suggestive of brainstem dysfunction in 12 and suggestive of end-organ dysfunction in 4. This mean that BAEP tests would be beneficial in diagnosing those patients with vague dizziness, even when the dizziness is a sole and nonspecific manifestation in an elderly person.

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