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Investigation of Auditory Brainstem Function in Elderly Diabetic Patients with Presbycusis

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Abstract: We performed brainstem auditory evoked potential (BAEP) examinations in 100 patients older than 60 years and having type I diabetes mellitus and presbycusis. The aim of our investigation was to compare the BAEP results of this group with those of healthy controls with presbycusis and to look for possible correlations between alteration of the auditory brainstem function and the aging of elderly diabetic patients. Absolute and interpeak latencies of all waves were prolonged significantly in the study group of diabetic patients. The amplitudes of all waves I through V were diminished in the study group as compared to those in the control group, with statistical significance present for all waves. Analysis of the latencies (waves I, II, III, and V), interpeak latencies (I–V), and amplitudes (I, II, III, and V) of BAEP revealed a significant difference between those of diabetics and those of healthy elderly controls with presbycusis. These data support a hypothesis that there is a brainstem neuropathy in diabetes mellitus that can be assessed with auditory brainstem response testing even in the group of elderly patients with sensorineural hearing loss.

Key Words: brainstem auditory evoked potentials; diabetes mellitus; presbycusis

Over the past decade, interest has been growing in evaluation of human auditory function in diabetics, although an early hearing impairment in diabetics is still a matter of controversy [1] because hearing loss is considered a nontypical symptom of this disease. There is a need for tests able to detect central auditory dysfunctions, and brainstem auditory evoked potentials (BAEPs) were recently recognized as offering the possibility of providing some information about subclinical demyelination and similar focal changes in the central nervous system (CNS).

The recording of electrically evoked potentials, especially auditory brainstem responses (ABRs), over the scalp has been proposed to evaluate the functional integrity of pathways in the CNS and, thus, for the diagnostic assessment of several neurological demyelinating or compressive diseases [2]. Very few data exist on the involvement of the CNS in diabetics [1]. Therefore, we performed ABR testing in all examined patients, as ABRs are generated mainly in the structures along the auditory lemniscal pathway [2].

The aim of this study was to evaluate the hearing functions and BAEPs in a group of elderly diabetics with presbycusis and to compare data with a group of healthy controls with presbycusis. We looked for possible correlations between alterations of the auditory brainstem function and aging of elderly diabetics with sensorineural hearing loss. All the subjects had presbycusis and sensorineural hearing loss not greater than 60 dB to avoid possible complications in BAEP data evaluation. The mean pure-tone threshold in the study and in the control group matched.

PATIENTS AND METHODS

We studied 100 patients older than 60 years (49 women, 51 men) and having insulin-dependent diabetes mellitus and presbycusis. Ages ranged from 60 to 98 years (mean age, 72.4 ± 7.7 years). The mean duration of the patients' diabetes was 11.6 ± 9.0 years (range, 1–33 years). The control group included 64 age-matched healthy patients with presbycusis.

We divided the group of diabetic patients and patients in the control group into three age-related subgroups:

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60–69 years, 70–79 years, and older than 80 years. For audiological evaluation, we carried out micro-otoscopy and standard pure-tone audiometry, tympanometry, stapedial reflex, and speech audiometry and obtained BAEP investigations from all subjects. We excluded from the study those subjects with a history of ototoxicity, noise exposure, or previous ear diseases.

We recorded ABR by the Amplaid MK 15 clinical system. With subjects resting with their eyes closed, we placed scalp electrodes at the vertex, forehead, and mastoid ipsilaterally to the stimulated ear. The impedance was kept below 5,000 ohms. At least two trials were performed on each side to obtain reproducible recordings. Stimuli were represented by alternate polarity clicks of 0.1-msec duration at 110 dB sound pressure level, and analysis time was 10 msec. We calculated the latency and amplitude values of waves I, II, III, and V and the interpeak latencies (IPL I–III; IPL III–V; IPL I–V). Wave I latency values were considered for peripheral transmission time and wave I–V interpeak intervals for central transmission time. Results are presented as means plus or minus the standard deviation. We performed statistical evaluation of data with the χ^2 test, an analysis of variance, and a least significant difference post-hoc test and the Pearson correlation coefficient.

RESULTS

The analysis of the gender differences shows no significant difference in latency and amplitude values between female and male subjects, both in normal and in diabetic subjects. All absolute latencies and interpeak latencies were significantly higher in diabetic patients than in the control group, although the difference in latency values was generally reduced with aging of the subjects. There was no significant difference in interpeak intervals I–III, III–V, and I–V within both groups of subjects (diabetics and healthy subjects) older than 80 years.

Analysis of the peripheral (wave I latency) and central transmission time (wave I–V interpeak intervals) showed that they are significantly higher with aging in diabetics. Central transmission time (waves I–V) was most significantly delayed in the first group (ages 60–69 years) of diabetic subjects. Peripheral transmission time (wave I) was most significantly delayed in the group of the oldest diabetics (>80 years). The control group showed no significant difference in central transmission due to aging of the subjects.

Mean amplitude values were generally reduced in diabetic patients. The wave I amplitude was significantly reduced in all age groups of the diabetics. The amplitude of the V wave was reduced only in the group of the oldest diabetics (>80 years). Mean amplitude values were generally not significantly reduced in the control group.

Mean latency and amplitude values of ABR components from diabetic and control subjects are reported in Tables 1 and 2.

Table 1. Clinical, Metabolic, and ABR Data of 100 Insulin-Dependent Diabetic Subjects

	Age Groups						Statistic	
	60–69 (N = 57)	*p*	70–79 (N = 24)	*p*	≥80 (N = 19)	*p**	F	*p*
PTA	34 ± 3	<.001	40 ± 2	<.001	47 ± 6	<.001	70.4	<.001
SSA	92 ± 6	<.001	86 ± 4	<.001	75 ± 6	<.001	59.1	<.001
GUK	8.86 ± 3.22	.005	11.14 ± 3.61	.855	10.9 ± 2.48	.015	5.7	.005
Duration (yr)	7 ± 4	<.001	13 ± 4	<.001	19 ± 10	.004	32.1	<.001
HbA1C	8.1 ± 1.9	<.001	10.1 ± 1.8	.556	9.7 ± 1.6	<.001	12.1	<.001
AI	70 ± 46	.002	39 ± 28	<.001	30 ± 19	.454	10.1	<.001
AII	26 ± 30	.012	11 ± 10	.833	9 ± 9	.010	5.3	.006
AIII	138 ± 94	.061	99 ± 74	.375	75 ± 54	.005	4.7	.011
AIV	28 ± 20	.083	20 ± 15	.103	11 ± 15	<.001	6.6	.002
AV	416 ± 201	.005	281 ± 199	.028	148 ± 144	<.001	15.0	<.001
LI	1.76 ± 0.23	.036	1.88 ± 0.23	.992	1.88 ± 0.22	.049	3.3	.039
LII	2.85 ± 0.44	.088	3.05 ± 0.60	.518	3.15 ± 0.47	.020	3.4	.036
LIII	4.06 ± 0.29	.074	4.2 ± 0.30	.434	4.27 ± 0.37	.011	4.0	.020
LIV	4.97 ± 0.46	—	5.15 ± 0.43	—	5.24 ± 0.32	—	2.6	.076
LV	6.05 ± 0.26	.047	6.2 ± 0.36	.017	6.44 ± 0.4	<.001	11.4	<.001
LI–III	2.8 ± 0.27	—	2.29 ± 0.26	—	2.38 ± 0.37	—	0.9	.375
LIII–V (ms)	1.99 ± 0.29	—	2.02 ± 0.34	—	2.16 ± 0.32	—	2.1	.120
LI–V	4.28 ± 0.25	.574	4.32 ± 0.27	.011	4.55 ± 0.38	<.001	6.4	.002

A = amplitude of the ABR wave (in microvolts); GUK = blood glucose level (in millimoles per liter); HbA1C = glycosylated hemoglobin (percentage); L = latency of the ABR wave (in milliseconds); PTA = pure-tone audiometry (in decibels); SSA = standard speech audiometry (percentage).
* In comparison with 60–69 age group.

Table 2. Clinical, Metabolic, and ABR Data of 64 Control Group Subjects

	Age Groups						Statistic	
	60–69 (N = 35)	*p*	70–79 (N = 18)	*p*	≥80 (N = 11)	*p**	F	*p*
PTA	32 ± 2	<.001	36 ± 2	<.001	42 ± 4	<.001	53.5	<.001
SSA	95 ± 3	.005	91 ± 4	<.001	82 ± 5	<.001	42.9	<.001
AI	148 ± 97	—	159 ± 90	—	115 ± 76	—	0.79	.454
AII	53 ± 43	—	54 ± 64	—	16 ± 16	—	2.70	.075
AIII	186 ± 103	—	157 ± 89	—	136 ± 117	—	1.16	.318
AIV	57 ± 60	—	40 ± 38	—	53 ± 99	—	0.40	.667
AV	478 ± 207	—	375 ± 171	—	351 ± 187	—	2.70	.075
LI	1.70 ± 0.18	.045	1.84 ± 0.20	<.001	2.13 ± 0.34	<.001	14.80	<.001
LII	2.67 ± 0.33	.067	2.87 ± 0.41	.027	3.19 ± 0.40	<.001	8.56	<.001
LIII	3.86 ± 0.26	.519	3.93 ± 0.29	.017	4.25 ± 0.58	.002	5.37	.007
LIV	4.84 ± 0.45	—	5.02 ± 0.47	—	5.07 ± 0.60	—	1.38	.257
LV	5.78 ± 0.29	.012	6.11 ± 0.51	.101	6.39 ± 0.67	<.001	9.25	<.001
LI–III	2.16 ± 0.31	—	2.08 ± 0.31	—	2.07 ± 0.65	—	0.34	.712
LIII–V	1.91 ± 0.28	.083	2.07 ± 0.31	.428	2.16 ± 0.40	.021	3.43	.039
LI–V	4.08 ± 0.29	—	4.16 ± 0.30	—	4.19 ± 0.90	—	0.34	.713

A = amplitude of the ABR wave (in microvolts); L = latency of the ABR wave (in milliseconds); PTA = pure-tone audiometry (in decibels); SSA = standard speech audiometry (percentage).
* In comparison with 60–69 age group.

DISCUSSION

ABR represents the electrical events generated along the auditory pathway. There is general agreement that wave I is produced by acoustic nerve activity, that wave II can reflect activity of the cochlear nucleus with a contribution from the auditory nerve, that wave III can be referred to generators in the superior olivary complex and the lateral lemnisci, and that the wave IV–V-complex is generated in the axons or nuclei of the lateral lemnisci (or both) and probably also from the inferior colliculi [3]. ABR recording can represent an objective, clinically useful, noninvasive procedure to stress the early impairment both of the auditory nerve and of brainstem function [4]. The I–V interval, or central transmission time, is considered the most reliable index of brainstem function [5].

The results of our study show that diabetic patients are characterized by an impairment in latency and amplitude values of all ABR components. The ABR waves are delayed and the amplitude values are generally reduced in diabetic subjects.

Our data showed that the I–V interval, or central transmission time, was mostly delayed in the youngest group of subjects (60–69 years old). With aging, such an impairment affects mostly peripheral structures so that the wave I latency (peripheral transmission time) is significantly higher in diabetic subjects older than 80 years than in younger subjects. This means that the impairment of the CNS pathway is very early in diabetic patients but, with aging, the impairment of the central structures diminishes, and the impairment of the peripheral acoustic pathway (acoustic nerve activity) progresses.

Delay of BAEP waves in diabetic patients has been reported previously. In 1981, Taylor et al. [1] were the first to try to correlate the ABR findings with central diabetic neuropathy. Nakamura et al. [6] presented us with magnetic resonance imaging with multiple lesions in the area of the pons and the thalamus in diabetic patients with pathological ABR. Others [7,8] reported that, like peripheral nerve conduction velocity, ABR was impaired very early in diabetes [2]. Conversely, other authors, such as Verma et al. [9], found no correlation between diabetes mellitus and delayed ABR responses.

In our study, only wave I amplitude was significantly reduced in diabetics in all age groups. Other amplitude values were generally, but not significantly, reduced in diabetic subjects. Our data correlate with the hypothesis that ischemia and lacunar demyelinization and the reduction in the nervous fibers of the acoustic nerve are responsible for the reduction of wave I amplitude [10].

Conversely, abnormal nerve conduction velocity rostral to the pons resulted in reduced wave I amplitude only in the group of oldest diabetics (> 80 years). This can be explained by the fact that as aging progresses, diabetic microangiopathy occurs with brainstem neuropathy and ABR impairment [11–13]. In our study, we observed these changes mostly in the oldest group of diabetic patients.

Our data support the hypothesis of Toth et al. [14] that diabetic neuropathy might be revealed as a cause of certain dysfunctions of the peripheral and central auditory pathways. Our results from the ABR wave latencies and amplitudes show that ABR recording can represent a very simple, noninvasive procedure to detect impairment of both the acoustic nerve and the CNS pathway in

diabetic patients, even when the subjects already suffer from sensorineural hearing loss.

Diabetes mellitus type I is characterized not only by somatic and autonomic nerve dysfunctions but by involvement of the CNS. ABR recording can represent a useful, simple procedure to detect both acoustic nerve and CNS damage even in the group of elderly patients with presbycusis and type I diabetes.

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