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Involvement of the auditory organ in type 1 diabetes mellitus

Zaburzenia funkcji narządu słuchu w cukrzycy typu 1

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Abstract

Introduction: The aim of this study was to evaluate auditory organ function in relatively young type 1 diabetic patients, with short duration of the disease and without overt hearing loss. The impact of age, diabetes duration and metabolic control on hearing function was also analysed.

Material and methods: Thirty-one patients with type 1 diabetes, aged below 45 years (mean 29.1 ± 7.1 years), with diabetes duration of less than 120 months (mean 54.7 ± 32.5 months), and no evident hearing impairment, were compared to 26 age-matched (30.3 ± 7.8 years, $p = 0.567$) healthy volunteers. In all subjects, pure-tone audiometry, transient evoked otoacoustic emissions (TEOAE), and auditory brainstem responses (ABR) were performed, after an ENT examination.

Results: In diabetic patients, compared to healthy subjects, the mean hearing threshold in the pure-tone audiometry was significantly higher at high frequencies, the mean amplitude of TEOAE was lower (7.75 ± 4.43 dB *v.* 10.00 ± 4.01 dB, $p < 0.001$), and latency times of wave V and interval I–V in ABR were longer (5.78 ± 0.25 ms *v.* 5.69 ± 0.18 ms, $p = 0.025$ and 4.03 ± 0.20 ms *v.* 3.95 ± 0.17 ms, $p = 0.017$ respectively). In the diabetic group, the hearing threshold showed positive linear correlation with age, whereas TEOAE was inversely correlated with this parameter. In ABR latency, times of wave V and interval I–V were negatively correlated with diabetes duration.

Conclusions: This study confirms the involvement of the auditory organ in type 1 diabetes mellitus. To determine the prognostic and predictive values of this finding, and methods of possible prevention of hearing loss, further prospective observations are required.
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Key words: *type 1 diabetes, hearing impairment, pure-tone audiometry, otoacoustic emissions, auditory brainstem responses*

Streszczenie

Wstęp: Celem badania była ocena funkcji narządu słuchu u relatywnie młodych osób z cukrzycą typu 1, o krótkim czasie trwania choroby i bez jawnych klinicznie ubytków słuchu. Analizie poddano także wpływ wieku, czasu trwania cukrzycy i jej kontroli metabolicznej na funkcję słuchu.

Materiał i metody: Trzydzieści jeden osób z cukrzycą typu 1, w wieku poniżej 45 lat (średnio 29,1 ± 7,1 lat), z czasem trwania cukrzycy poniżej 120 miesięcy (średnio 54,7 ± 32,5 miesięcy), i bez jawnego ubytku słuchu, porównano z dwudziestoma sześcioma zdrowymi ochotnikami dopasowanymi pod względem wieku (30,3 ± 7,8 lat, $p = 0{,}567$). U wszystkich osób, po przeprowadzeniu badania laryngologicznego, wykonano badanie audiometrii tonalnej progowej, emisji otoakustycznej wywołanej trzaskiem oraz słuchowych potencjałów wywołanych pnia mózgu.

Wyniki: U osób z cukrzycą średni próg słuchu w audiometrii tonalnej progowej był znamiennie wyższy w wysokich częstotliwościach, średnia amplituda emisji otoakustycznej była niższa (7,75 ± 4,43 dB *v.* 10,00 ± 4,01 dB, $p < 0{,}001$), także czasy latencji fali V i interwału I–V w badaniu słuchowych potencjałów wywołanych były dłuższe (odpowiednio 5,78 ± 0,25 ms *v.* 5,69 ± 0,18 ms, $p = 0{,}025$ i 4,03 ± 0,20 ms *v.* 3,95 ± 0,17 ms, $p = 0{,}017$) w porównaniu z osobami zdrowymi. U osób z cukrzycą próg słuchu wykazywał dodatnią, zaś amplituda emisji otoakustycznej ujemną korelację liniową z wiekiem. W badaniu słuchowych potencjałów wywołanych czasy latencji fali V i interwału I–V ujemnie korelowały z czasem trwania cukrzycy.

Wnioski: Praca ta potwierdza wpływ cukrzycy typu 1 na funkcję narządu słuchu. Dla oceny wartości prognostycznej uzyskanych wyników, jak też potencjalnych sposobów zapobiegania uszkodzeniu słuchu w cukrzycy konieczne są dalsze prospektywne badania tego zjawiska. **(Endokrynol Pol 2011; 62 (2): 138–144)**

Słowa kluczowe: *cukrzyca typu 1, upośledzenie słuchu, audiometria tonalna progowa, emisje otoakustyczne, potencjały wywołane pnia mózgu*

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Introduction

Type 1 diabetes is a chronic metabolic disorder characterised by hyperglycaemia resulting from autoimmune destruction of the beta-cells of the pancreas. An elevated blood glucose level can lead to dysfunction, damage and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels [1]. One of the lesser known consequences of diabetes is auditory organ dysfunction.

Although there is mounting evidence for a relationship between type 1 diabetes and hearing impairment [2–15], the awareness of auditory organ involvement in the course of diabetes is still not widespread among healthcare providers involved in diabetes care. Altered hearing function in diabetic patients has been found in both pure-tone audiometry [2–8] and otoacoustic emissions [9–12], as well as in auditory brainstem responses [7, 9–11, 13–15].

Pure-tone audiometry is used to determine a hearing threshold, i.e. the softest sound audible to the person being studied. This measure reflects auditory organ function as a whole, both its peripheral and central parts [16]. An otoacoustic emission (OAE) test is used to determine the status of the cochlear micromechanics, especially the function of the outer hair cells (OHC) [17]. Auditory brainstem response audiometry is a neurological test used to determine function of the retrocochlear part of the auditory pathway, up to brainstem level [18].

Since previous studies had examined highly heterogeneous populations, predominantly patients with long lasting diabetes and often with microvascular diabetic complications, we decided to evaluate whether auditory organ dysfunction exists in relatively young type 1 diabetic subjects, with a short duration of the disease, and with no clinically evident hearing impairment. The secondary objective of our study was to evaluate the impact of age, duration of diabetes, and metabolic control on hearing function in type 1 diabetes. Associations of retinopathy and urinary albumin excretion with auditory function were also analysed.

To evaluate the whole auditory pathway, with both its cochlear and retrocochlear parts, the three audiological tests described above were performed.

Material and methods

Participants

Inclusion criteria were: to be aged below 45 (to avoid the impact of presbyacusis), and to have had diabetes for less than 10 years (for the diabetic group) to exclude patients with advanced diabetic complications. Exclusion criteria were: clinically overt hearing loss and/or prolonged exposure to noise and/or a history of ototoxic medications. The study group consisted of 31 type 1 diabetic patients (eight female and 23 male), with a mean age of 29.1 ± 7.1 years (range 18–43), who were being treated in the diabetic outpatient clinic at the Beta-Med Medical Centre, Rzeszow, Poland. The control group consisted of 26 healthy age-matched (30.3 ± 7.8, range 19–43, $p = 0.567$) volunteers. They included hospital staff, students and drug company representatives (19 female and seven male).

In all but one of the diabetic subjects, an ophthalmoscopic eye fundus evaluation was performed by an ophthalmologist.

The metabolic control of diabetes was determined by HbA_{1C} measurement, and the presence of microalbuminuria was determined by the albumin concentration and albumin/creatinine ratio assessment from a morning sample of urine. Both measurements were performed using a DCA 2000® + analyser (Bayer Corporation, Elkhart, IN, USA) using the monoclonal antibody method.

Among the diabetic group, three patients had early background retinopathy, one had urinary albumin excretion in the microalbuminuric range, but none had clinically overt diabetic neuropathy.

Audiological tests

In all subjects, before the audiological tests, a detailed ear examination by a single otorhinolaryngologist was performed to exclude abnormalities in the external and middle ear. Then pure-tone audiometry, transient evoked otoacoustic emissions and auditory brainstem responses were assessed. All evaluations were performed in the Department of Otorhinolaryngology at the Provincial Specialist Hospital in Rzeszow, Poland.

Pure-tone audiometry was performed in a soundproof booth, using a Madsen OB822 audiometer (GN Otometrics, Taastrup, Denmark) with Telephonics TDH 39 earphones. The air conduction was measured at the frequency range 125–12,000 Hz, and the bone conduction was measured at frequencies 250–6,000 Hz. The initial stimulus was 10 dB HL (hearing level). Then the level was increased in 5-dB HL steps. Mild hearing impairment was recognised at hearing thresholds above 20 dB in at least one frequency, and moderate hearing impairment was recognised at hearing thresholds above 40 dB.

Transient evoked otoacoustic emissions were obtained using a Scout Sport 580-OAE SP6 Analyser (Bio-logic Systems Corp., Mundelein, IL, USA) with a 'non-linear' click stimulus of 80 μs duration, a repetition rate of 50 Hz, and an intensity of ~80 dB. The results were presented in dB as an average for band range 1.2–3.5 kHz, and also for particular frequencies of TEOAE spectrum: 1, 1.5, 2, 3 and 4 kHz. A mean TEOAE amplitude below

6 dB at band range 1.2–3.5 kHz was considered as a lack of otoacoustic emission.

Auditory brainstem responses were evaluated using a Centor-C analyser (Racia-Alvar, Paris, France) with click stimulus of 100 μs duration, a repetition rate of 19.1 Hz, an intensity of 70 dB, and a contralateral ear masking of -30 dB. Electrodes were placed on the forehead (positive), the ipsilateral mastoid (negative), and chin (ground). The latency time of waves I, III and V, and the intervals between them, were measured.

Statistical analysis

Statistical analysis was performed using SigmaStat for Windows Version 3.5 (Systat Software Inc., San Jose, CA, USA). Data from the diabetic and control groups was compared using an unpaired Student's *t*-test, after performing a Kolmogorov-Smirnov normality test and a constant variance test. In the case of normality and/or constant variance test failure, the Mann-Whitney rank sum test was performed. The linear correlations between hearing function and age, diabetes duration, HbA_{1C}, and UAE in diabetic subjects were analysed using a Pearson product moment correlation test. In cases of normality and/or constant variance test failure, a Spearman rank order correlation test was performed. To assess the strength and independency of associations, a multiple linear regression test was used. A p value < 0.05 was considered statistically significant.

Results

Pure-tone audiometry

Data from the air-conduction hearing thresholds of both ears at each frequency was used in the analysis. In the study group, 25 patients had normal hearing, five had mild hearing loss, and one had moderate hearing loss. In the control group, 23 subjects had normal hearing, and three had mild hearing loss. In the diabetic group, the mean hearing thresholds at frequencies 3,000–12,000 Hz were significantly higher compared to the control group (Table I, Fig. 1).

In the diabetic group, in univariate analysis, the hearing threshold showed a highly significant positive linear correlation with age at frequencies 2,000–12,000 Hz. In multivariate analysis, after adjustment for diabetes duration, HbA_{1C} level and UAE, the correlation between age and hearing threshold remained significant at frequencies 3,000–12,000 Hz (Table II).

Transient evoked otoacoustic emissions

The otoacoustic emissions in the diabetic group were absent bilaterally in six, and unilaterally in two, patients.

Table I. *Hearing thresholds in pure-tone audiometry*
Tabela I. *Próg słuchu w audiometrii tonalnej progowej*

Frequency (Hz)	Hearing threshold (dB) (mean ± SD)		p value
	Diabetes	Control	
125	10.16 ± 1.27	10.00 ± 0.00	NS*
250	10.16 ± 1.27	10.00 ± 0.00	NS
500	10.24 ± 1.41	10.00 ± 0.00	NS
1,000	10.65 ± 3.07	10.00 ± 0.00	NS
2,000	10.57 ± 2.38	10.00 ± 0.00	NS (0.065)
3,000	12.18 ± 6.38	10.00 ± 0.00	0.003
4,000	13.47 ± 8.90	10.10 ± 0.69	0.002
6,000	13.95 ± 8.26	10.87 ± 3.39	0.004
8,000	13.47 ± 8.76	10.77 ± 2.87	0.013
12,000	15.32 ± 10.59	11.63 ± 4.61	0.018

*NS = non significant

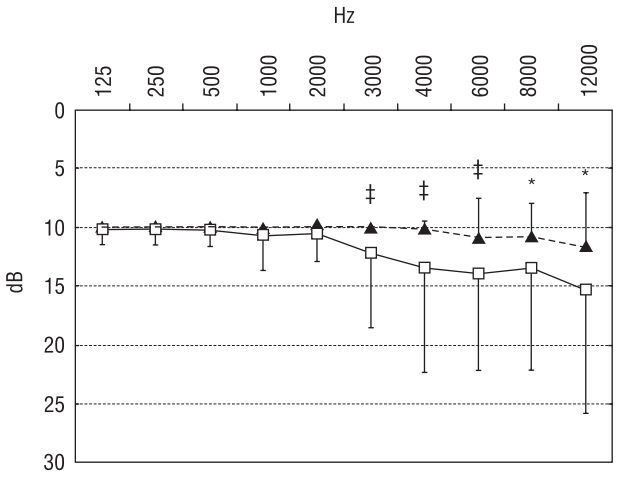

Figure 1. *Hearing threshold in pure-tone audiometry (mean ± SD); diabetes – solid line and open squares; control – interrupted line and solid triangles; *p < 0.05; ‡p < 0.005*

Rycina 1. *Próg słuchu w audiometrii tonalnej progowej (średnia ± ± SD); cukrzyca — linia ciągła i puste kwadraty, kontrola — linia przerywana i pełne trójkąty; *p < 0,05, ‡p < 0,005*

In the control group, they were absent bilaterally in two and unilaterally in one subject. The mean amplitude of TEOAE at band range 1.2–3.5 kHz was significantly lower in the study group compared to the control group. Also at 1.5, 2 and 4 kHz the TEOAE response was significantly lower in diabetic patients (Table III, Fig. 2).

In the diabetic patients, in univariate analysis, the mean amplitude of TEOAE showed a negative linear correlation with age (correlation coefficient R = –0.353,

Table II. *Linear correlation between age and hearing threshold at particular frequencies*

Tabela II. *Korelacja liniowa pomiędzy wiekiem a progiem słuchu w poszczególnych częstotliwościach*

Frequency (Hz)	Correlation coefficient R	p value	Adjusted p value*
125	0.050	NS†	NS
250	0.050	NS	NS
500	0.181	NS	NS
1,000	–0.105	NS	NS
2,000	0.259	0.043	NS (0.076)
3,000	0.442	< 0.001	< 0.001
4,000	0.564	< 0.001	< 0.001
6,000	0.545	< 0.001	< 0.001
8,000	0.587	< 0.001	< 0.001
12,000	0.475	< 0.001	0.002

*Adjusted for diabetes duration, HbA_{1C} level and urinary albumin excretion; †NS — non significant

p = 0.005). In multivariate analysis, the impact of age on TEOAE amplitude appeared to be independent of diabetes duration, metabolic control or UAE.

Nine of the diabetic patients (29.0%) achieved HbA_{1C} < 7%. In this group, the mean TEOAE amplitude was higher in comparison with the remaining 22 patients with lesser metabolic control (9.96 ± 4.58 dB *v.* 6.85 ± ± 4.09 dB, p = 0.011). However, no linear correlation was found between HbA_{1C} level and TEOAE amplitude.

Auditory brainstem responses

In the diabetic group, the latency time of wave V and interval I–V duration was significantly longer compared to the control group (5.78 ± 0.25 ms *v.* 5.69 ± 0.18 ms, p = 0.025 and 4.03 ± 0.20 ms *v.* 3.95 ± 0.17 ms, p = 0.017 respectively). The interval I–III was also prolonged in the study group (2.17 ± 0.15 ms *v.* 2.12 ± 0.18 ms), but did not reach statistical significance (p = 0.059) (Table IV, Fig. 3).

In the diabetic patients, in univariate analysis, we found a negative linear correlation between diabetes duration and latency time of wave V (R = –0.256, p = 0.045) as well as interval I–V (R = –0.382, p = 0.004) (Fig. 4). In multivariate analysis, the impact of diabetes duration on wave V and interval I–V latency was independent of age, HbA_{1C} level and UAE.

When diabetic patients were split into two subgroups according to diabetes duration (more or less than five years), the 17 patients with a shorter history of diabetes demonstrated longer latency time of wave V (5.84 ± 0.24 *v.* 5.70 ± 0.24 ms, p = 0.023), and also longer intervals I–III (2.21 ± 0.14 ms *v.* 2.12 ± 0.15 ms, p = 0.026) and I–V (4.08 ± 0.18 ms *v.* 3.97 ± 0.21 ms, p = 0.030), compared to the 14 patients with a longer diabetes history.

Table III. *Transient evoked otoacoustic emissions (TEOAE) in diabetic and control groups*

Tabela III. *Emisja otoakustyczna wywołana trzaskiem (TEOAE) u osób z cukrzycą i w grupie kontrolnej*

Band (kHz)	TEOAE amplitude (dB) (mean ± SD)		p value
	Diabetes	Control	
1.2 – 3.5	7.75 ± 4.43	10.00 ± 4.01	< 0.001
1	3.67 ± 4.02	4.81 ± 4.74	NS*
1.5	7.12 ± 5.49	10.57 ± 5.77	0.002
2	7.21 ± 4.75	10.73 ± 4.81	< 0.001
3	7.18 ± 5.16	8.16 ± 4.05	NS
4	4.75 ± 3.79	6.45 ± 3.08	0.017

*NS — non significant

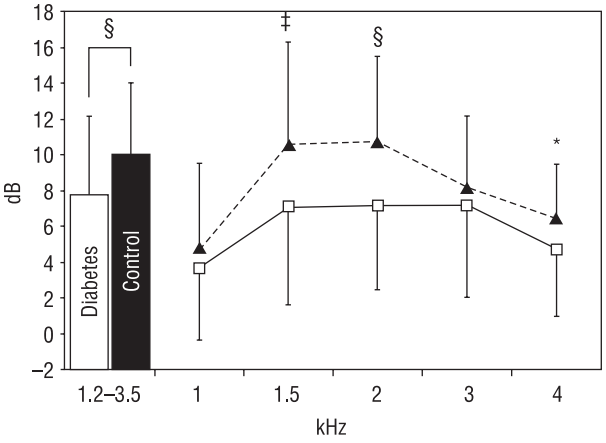

Figure 2. *Mean amplitude of TEOAE at band range 1.2–3.5 kHz (bars) and at particular frequencies of TEOAE spectrum (presented as TE-gram) (mean ± SD); diabetes – solid line and open squares; control – interrupted line and solid triangles; *p < 0.05; ‡p < 0.005; §p < 0.001*

Rycina 2. *Uśredniona amplituda emisji otoakustycznej w zakresie 1.2–3.5 kHz (słupki) oraz w poszczególnych zakresach widma TEOAE (przedstawiona jako TE-gram) (średnia ± SD); cukrzyca — linia ciągła i puste kwadraty, kontrola — linia przerywana i pełne trójkąty; *p < 0,05, ‡p < 0,005, §p < 0,001*

Retinopathy

Early background retinopathy was found in three diabetic patients. Despite this very small number, the patients with retinopathy appeared to have significantly higher hearing threshold in pure tone audiometry at 1, 2 and 4 kHz. No relationship between retinopathy and otoacoustic emissions or ABR was demonstrated.

Table IV. *Auditory brainstem responses in the diabetic and control groups*

Tabela IV. *Słuchowe potencjały wywołane pnia mózgu (ABR) u osób z cukrzycą i w grupie kontrolnej*

Parameter	Time (ms) (mean ± SD)		p value
	Diabetes	Control	
Latency time			
Wave I	1.73 ± 0.12	1.73 ± 0.11	NS*
Wave III	3.91 ± 0.19	3.86 ± 0.16	NS
Wave V	5.78 ± 0.25	5.69 ± 0.18	0.025
Interval duration			
I–III	2.17 ± 0.15	2.12 ± 0.18	NS (0.059)
III–V	1.85 ± 0.16	1.83 ± 0.17	NS
I–V	4.03 ± 0.20	3.95 ± 0.17	0.017

*NS — non significant

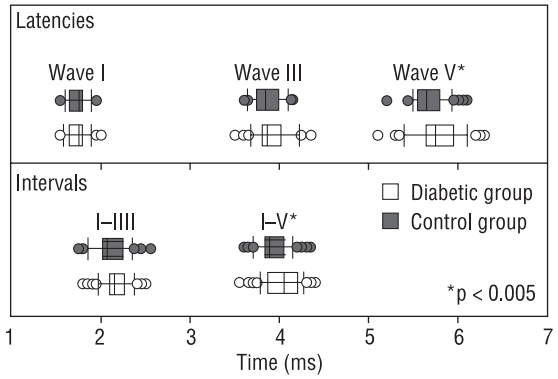

Figure 3. *Latencies and interval times in ABR (line: median; box: 25th and 75th percentile; error bars: 10th and 90th percentile; dots: outliers) (the interval III – V is omitted); *p < 0.05*

Rycina 3. *Czasy latencji poszczególnych fal i interwałów pomiędzy nimi (linia: mediana, ramka: 25 i 75 centyl, słupki błędów: 10 i 90 centyl, kropki: wartości odstające) (interwał III — V pominięto), *p < 0,05*

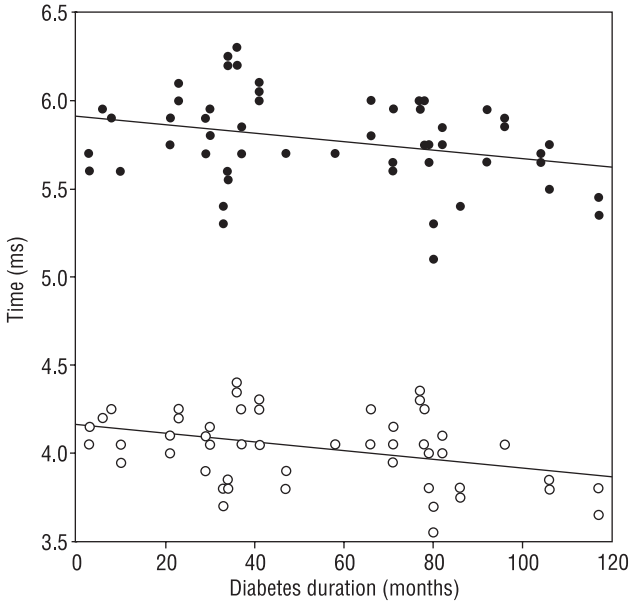

Figure 4. *Negative linear correlation between diabetes duration and latency time of wave V (solid circles, R = –0.256, p = 0.045) and interval I–V (open circles, R = –0.382, p = 0.004) (scatter plot and regression line)*

Rycina 4. *Ujemna korelacja liniowa pomiędzy czasem latencji fali V (pełne kółka, R = –0,256, p = 0,045) oraz interwału I–V (puste kółka, R = –0,382, p = 0,004) (wykres punktowy i linia regresji)*

Discussion

Our study confirmed the existence of auditory organ dysfunction in relatively young type 1 diabetic patients, with a short duration of the disease and without clinically overt hearing impairment. Significant differences between diabetic and control groups were found in all audiological tests.

The hearing threshold in the pure-tone audiometry was in our study significantly higher at high and, partially, middle frequencies in type 1 diabetic subjects in comparison with healthy controls. Similar findings have been revealed in other studies [2–8].

The amplitude of otoacoustic emissions in our observation was significantly lower in the diabetic patients compared to the control group, which confirmed previous observations [9–12]. In one of those studies, DPOAE (distortion product otoacoustic emissions), which are more frequency-sensitive, rather than TEOAE, were used [10]. Nevertheless, in all cases, OAE were lower in the diabetic patients.

Auditory brainstem responses in our study showed significant differences between diabetic and control groups. The latency time of wave V and interval I–V in diabetic patients were significantly longer compared to healthy subjects. Other studies have also demonstrated prolonged wave V latency [9, 11, 13–15] and prolonged interval I–V duration [10, 13–15] in diabetic patients. Some of them have also found longer latency time of wave I and wave III [9, 11, 15], as well as prolonged intervals I–III [7, 15] and III–V [14].

The impact of age on hearing function is well documented. The hearing threshold increases, predominantly at middle and high frequencies [19], whereas amplitude of OAE decreases, with age [20]. Thus, unsurprisingly, our study found a strong positive correlation between age and hearing threshold, as well as a negative correlation between age and TEOAE in the diabetic group. A similar impact of age on hearing threshold was revealed in the study by Pudar et al. [8].

In the same study, differences regarding gender in pure-tone audiometry (i.e. a higher threshold in men) and in ABR (i.e. a shorter latency of wave III and V, as

well as longer I-III interval in women) were found. However, it was not observed in the control group [8]. Since most previous studies comparing males and females with diabetes have found no sex differences in audiological tests [6, 21, 22], we didn't try to strictly match the two groups regarding gender.

In our study, diabetes duration appeared to have no impact on hearing threshold in diabetic patients. This is contrary to other observations [2, 3, 5–8], which have found a higher hearing threshold in longer lasting diabetes. The amplitude of OAE, neither in our study, nor in others [9, 11] has shown an association with the duration of diabetes. One paper noted a correlation between diabetes duration and prolongation of interval I–III [8], and another found prolongation of latency time of wave V and intervals I–V and III–V [14]. However, this has not been observed in other studies [9, 11, 13, 15]. Interestingly, in our study, wave V latency and interval I–V time were inversely correlated with diabetes duration.

Metabolic control is a well-known risk factor for diabetic complications [23]. In our study, patients with near-normal glycaemic control (HbA_{1C} < 7%) demonstrated higher TEOAE amplitude than subjects with lesser glycaemic control. However, no linear correlation between HbA_{1C} level and TEOAE amplitude was found. In other studies, an association between metabolic control and TEOAE amplitude was not revealed [9, 11]. We didn't find a relationship between metabolic control and hearing threshold, or ABR results. Most other studies also failed to find such a correlation [9, 11, 13, 14]. However, two studies showed a correlation between poor metabolic control and higher hearing threshold [6, 7], as well as with prolonged interval I-III in ABR [7]. An interesting finding by Virtaniemi et al. was that ABR disturbances did not reverse, even when an improvement in metabolic control was achieved [24].

The association between hearing impairment and other diabetic complications has been described in several papers [11, 12, 14, 25]. Our study revealed a higher hearing threshold at middle frequencies in patients with retinopathy. Due to the fact that only one patient had microalbuminuria, and none had clinically overt neuropathy, we did not analyse the relationship between these complications and audiological test results.

Some discrepancies between the results of our study and the studies mentioned above can be explained by the fact that those studies used a different methodology, as well as different inclusion/exclusion criteria, and also studied highly heterogeneous populations.

Auditory organ involvement is present not only in patients with type 1 diabetes, but also in type 2 diabetic subjects. Among a population drawn from the National Health and Nutrition Examination Survey (NHANES), hearing impairment was more prevalent in adult diabetic subjects than in those without diabetes. This association was independent of known risk factors for hearing loss, such as noise exposure, ototoxic medication use, or smoking [26, 27]. Among veterans with type 2 diabetes, elevated hearing threshold and prolonged latencies of wave V and interval I-V were observed, especially in younger patients (below 50 years), treated with insulin, and presenting other diabetic complications [28, 29]. NHANES also revealed that over the last 30 years, the prevalence of hearing impairment has decreased significantly in the non-diabetic adult population of the USA. However, this has not been replicated in subjects with diabetes, where prevalence has remained at the same level [30]. On the basis of the paper by Bainbridge et al. [26], Hirose drew the conclusion that audiometry should be considered as a routine evaluation in an annual test battery for diabetic patients [31].

The mechanisms involved in the development of hearing dysfunction in diabetic patients are not so well recognised as in the retina or in the kidney, primarily due to the fact that they cannot be assessed by intravital examination. Few histopathological studies have been carried out in humans. One such study found microangiopathic changes in the vessels of stria vascularis, the endolymphatic sac and the basilar membrane of the cochlea [32]. Another study found thickening of the capillary walls in the stria vascularis and in the basilar membrane [33]. In addition, a greater loss of the outer hair cells (OHC), predominantly in the lower basal turn of the cochlea, and atrophy of the stria vascularis in diabetic subjects were also demonstrated. Since the basal turn of the cochlea (where the microangiopathic changes were predominantly seen) is responsible for receiving high frequencies [34], these findings could explain the higher hearing threshold in diabetic patients at these frequencies. The lower amplitude of OAE observed in diabetic subjects can also be, at least in part, explained by a greater OHC loss in this group.

The delay of auditory brainstem evoked potentials observed in our study could be a manifestation of acoustic nerve and central auditory pathway neuropathy, as it is seen in the peripheral nerves [35]. The relationship between ABR disturbances and cardiac autonomic neuropathy as well as peripheral neuropathy has been described by Várkonyi et al. [36, 37]. The negative correlation between diabetes duration and latency time of wave V and interval I–V revealed in our work can be potentially explained by nervous tissue metabolism, where glucose is an essential nutrient. Chronic hyperglycaemia in long lasting diabetes can induce adaptational changes towards normalisation of intracellular metabolism, and thus the tendency to normalisation of conduction velocity, something speculated as regards the retina by Klemp et al. in their paper [38].

Conclusions

Although the study was performed on a small number of participants, our results demonstrated that a relationship between type 1 diabetes mellitus and auditory organ dysfunction exists. Both the cochlear and the retrocochlear part of the auditory pathway, up to brainstem level, are involved. Although the abnormalities were subtle, they were statistically significant, and they were detected before development of other diabetic microvascular complications. It is worth noting that in our study, elevated hearing threshold was also present at speech frequency. Since hearing impairment may well have a negative impact on the social function of an affected individual, the use of audiological tests to monitor diabetic patients may be considered as a routine procedure, in the same way as eye fundus examination and microalbuminuria assessment are.

However, the prognostic and predictive value of auditory organ dysfunction, as well as methods of possible prevention of hearing loss, require further prospective observations to be properly determined.

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