Cochlear Dysfunction in Type 2 Diabetes: A Complication Independent of Neuropathy and Acute Hyperglycemia

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The effects of type 2 diabetes on evoked otoacoustic emissions (e-OAEs) elicited by clicks in subjects with normal hearing and the involvement of the central (CNS) and peripheral nervous system and acute hyperglycemia were investigated. In study 1, 110 type 2 diabetic patients and 106 control subjects matched for age and gender were investigated by e-OAEs. Central and peripheral neuropathy were evaluated respectively by auditory brainstem responses (ABRs) and according to San Antonio Consensus Conference criteria. In study 2, 10 healthy and 10 type 2 diabetic men matched for age, all with normal e-OAEs, underwent a 5-hour hyperglycemic clamp study. e-OAE tests were performed before and during the hyperglycemic clamp. In study 1, e-OAEs were impaired in 51.8% (57 of 110) of the diabetic subjects, in comparison to 4.7% (five of 106) of the control group ($P < .0001$). Diabetics with impaired e-OAEs (e-OAEs−), in comparison to those with normal e-OAEs (e-OAEs+), were older (51.0 ± 5.8 *v* 45.1 ± 6.0 years, $P < .001$), had diabetes longer (11.5 ± 4.4 *v* 7.0 ± 3.9 years, $P < .001$), achieved poorer metabolic control as judged by hemoglobin A_{1c} ([HbA_{1c}] 6.9% ± 0.4% *v* 6.5% ± 0.3%, $P < .001$), and had more peripheral neuropathy (46% *v* 23%, $P < .02$). No difference was observed between e-OAEs− and e-OAEs+ subjects for retinopathy or nephropathy. Nevertheless, when the duration of diabetes was corrected by multiple regression analysis, the correlation between sensorineural damage and peripheral neuropathy lost significance ($P = .12$). Diabetic groups (e-OAEs+ and e-OAEs−) showed greater latency in waves I, III, and V and greater interwave latency for waves I to V than the control group, but there was no significant difference in ABRs between e-OAEs+ and e-OAEs− subjects. In study 2, there were no significant changes in e-OAE intensities compared with basal values during the entire hyperglycemic clamp in either type 2 diabetic or control subjects. No difference was observed between the two groups at each time of the clamp. Thus, type 2 diabetic subjects show a higher rate of compromised e-OAEs than healthy individuals. The e-OAE dysfunction does not associate with either an injury to the auditory nervous pathway or diabetic microvasculopathy. The apparent interference of peripheral neuropathy in e-OAEs loses significance when corrected for the duration of diabetes.

THE ASSOCIATION OF DIABETES and hearing loss has been postulated since the middle of the last century.[1] Several investigators[2,3] have described a progressive bilateral sensorineural deafness with gradual onset predominantly affecting the higher frequencies. Nevertheless, in male hearing loss, low frequencies are also affected.[4] There is a likelihood that these alterations are related to microangiopathy as confirmed by histopathological studies on the inner ear in diabetics. Capillary wall thickening in the stria vascularis[5,6] and narrowing of the lumen, as well as loss of ganglion cells and demyelination of nerve sheath VIII, have been observed.[7] Moreover, the vascular involvement of the basement membrane is associated with reduced hair cells. In fact, a significant outer hair cell (OHC) loss has been found in the cochlea of the SHR/N-cp rat, a model for type 2 diabetes mellitus, with no statistical difference between the obese and lean phenotypes.[8] These findings suggest a relationship between type 2 diabetes mellitus and inner-ear damage, and indicate that OHC loss is related either to hyperglycemia or to a genetic predisposition for glucose intolerance.[9] The relationship between the duration of diabetes and hearing loss has not always been demonstrated. Deafness appears to be twice as common in patients with severe proliferative retinopathy.[10] The inner ear can receive and emit sound by the following active mechanisms of the OHCs: contraction by an actin-myosin system, changes in cellular turgidity due to the osmotic effect of polysaccharides, and dynamic stretch between OHCs and Deiters' cells. A noninvasive clinical tool to investigate cochlear function has been available for 20 years.[11] Evoked otoacoustic emissions (e-OAEs) reflect cochlear dynamics related to OHC function.[12,13] OHCs modulate hearing, and any changes could thus lead to sensorineural deafness and, moreover, might be the anatomic basis for functional damage objectively measured by e-OAEs.

Only two studies have investigated the relationship between diabetes and e-OAEs.[14,15] The relationship with type 2 diabetes has not been explored. Moreover, sensory damage as an aspect of central and peripheral neuropathy in type 2 diabetes has not been investigated. Evoked potentials, ie, auditory brainstem responses (ABRs), are electrophysiological methods of investigation to provide quantitative information about central nervous system (CNS) changes.[16-18]

The aim of this study was to investigate (1) the effects of type 2 diabetes on e-OAEs elicited by clicks in subjects with normal hearing and the involvement of the CNS and peripheral nervous system in e-OAEs (study 1), and (2) the role of acute hyperglycemia in e-OAEs in both type 2 diabetic and control subjects (study 2).

SUBJECTS AND METHODS

Study 1

One hundred ten patients with type 2 diabetes according to National Diabetes Data Group criteria[19] (56 men and 54 women aged 48.4 ± 5.7 years, mean ± SD) and 106 control subjects matched for age and gender (53 men and 53 women aged 47.9 ± 6.9 years) were enrolled. The diabetic subjects were in good metabolic control, as judged by hemoglobin A_{1c} (HbA_{1c}) levels, with diet and oral hypoglycemic agents.

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All subjects were investigated with e-OAEs and ABRs. Their clinical characteristics are shown in Table 1.

Study 2

Ten healthy men and 10 type 2 diabetic men matched for age (Table 2) underwent a 5-hour hyperglycemic clamp study according to the method of De Fronzo et al.[20] The inclusion criterium for all subjects was the presence of normal e-OAEs. Plasma glucose was acutely increased by a 15% glucose primed-continuous infusion starting at 800 mL/h and decreasing to 500 mL/h at 5 minutes. By minute 8, the infusion rate was changed according to glycemia to attain a level of 12 mmol/L, generally achieved in less than 15 minutes. After achieving this glucose level, glycemia was measured at 5-minute intervals and the glucose infusion was adjusted accordingly. e-OAE tests were performed before and during the hyperglycemic clamp (5, 10, 15, 20, 30, 60, 120, 180, 240, and 300 minutes). The coefficient of variation for glycemia did not exceed 6% during the interval from 15 to 300 minutes.

All subjects were examined as outpatients on an audiological ward. Each had a blood sample drawn from an antecubital vein for plasma glucose determination just before starting the e-OAE evaluation and in study 2, before and during the clamp. All participants had a previous audiological history, microotoscopy, standard pure-tone audiogram, and tympanometry to disclose acute and chronic inflammatory processes in the middle ear or the outcome of such pathologies capable of altering the study of e-OAEs. Only subjects with tympanometry type A according to Liden's classification[21] were enrolled. Moreover, within the two groups in study 1 (diabetic and control), both normal-hearing subjects and those with slight hypoacusis (<30 dB hearing threshold level [HTL]) for more acute tones (6,000 to 8,000 Hz) were included. This condition does not jeopardize the study, as e-OAEs are manifest if tones between 500 and 4,000 Hz are well preserved.[22] In study 2, both normal hearing function and the presence of cochlear echos were required. The investigations were performed in accordance with the principles of the Declaration of Helsinki.

e-OAE Recordings

e-OAE tests were performed using a computer-based Otodynamic Analyzer (ILO 88; Amplaid, Milan, Italy). The non–linear-difference method was used, and the stimulus was an unfiltered 80-μ click; its level in the external canal was 80 dB sound pressure level (SPL). An average of 260 clicks were obtained. The absence or presence of e-OAEs was analyzed. The following criteria were used for evaluation: (1) study 1: presence or absence of cochlear echos. Other parameters (reproducibility, amplitude, and frequency spectrum) in the healthy group are so highly interindividually variable[23] that their range is too wide to be used as a normal indicator; (2) study 2: modifications of e-OAE layouts performed for the same subject and the eventual disappearance of the layout and its changes in amplitude were determined.

ABR Recordings

Patients were examined in a reclining position. The recordings were made with an Amplaid MK 15 evoked-response system. The ABR technique was performed standardly. The acoustic stimulus consisted of clicks delivered monoaurally through cushioned earphones. Alternate polarity clicks were generated by digital stimulators at a rate of 21/s. In patients and controls in the first study, complete ABR audiometry was performed to evaluate the absolute latency of waves I, III, and V and the interwave latency for I to V, I to III, and III to V measured from peak to peak.[24] In at least two trials, responses to 2,000 clicks were recorded at 110 dB in each ear, and no traces were superimposed to check for peak replicability. The potentials were measured between an earlobe electrode and a vertex surface electrode (ground to nose).

Table 1. Clinical Characteristics of Type 2 Diabetic Patients and Control Subjects in Study 1

Group	No. of Subjects (male/female)	Age (yr)	Duration of Diabetes (yr)	HbA_{1c} (%)
Diabetics	110 (56/54)	48.4 ± 5.7	8.1 ± 4.1	6.7 ± 0.3
Controls	106 (53/53)	47.9 ± 6.9		4.4 ± 0.3

Table 2. Clinical Characteristics of Type 2 Diabetic Patients and Control Subjects in Study 2

Group	No. of Subjects (male/female)	Age (yr)	Duration of Diabetes (yr)	HbA_{1c} (%)
Diabetics	10 (10/0)	51.6 ± 4.4	8.4 ± 3.5	6.5 ± 0.3
Controls	10 (10/0)	50.1 ± 5.7		4.4 ± 0.3

Diabetic Complications

All subjects were screened for microvasculopathy and neuropathy. Direct fundoscopy and three 24-hour samples of sterile urine, collected over 2 months to assess the albumin excretion rate, were used to evaluate diabetic retinopathy and nephropathy. Studies of sensory and motor nerve conduction were performed according to criteria from the San Antonio Consensus Conference.[25] Autonomic neural function was assessed by the following cardiac reflex tests: (1) heart rate response to deep breathing at six breaths per minute with calculation of the ratio of the longest RR interval during expiration and the shortest RR interval during inspiration, (2) Valsalva maneuver and calculation of the ratio of the longest RR interval after and the shortest RR interval during the maneuver, (3) heart rate response to standing (30:15 ratio), and (4) blood pressure response to standing. The vibratory-perception threshold was evaluated by biothesiometry (Bio-Thesiometer; Bio Medical Instrument, Newbury, OH). Diabetic patients underwent a neurological clinical examination and completed a questionnaire for neurological symptoms. The criteria for diabetic neuropathy were from the San Antonio Consensus Conference, according to age-related reference values.[26]

None of the participants were taking any medications other than oral hypoglycemic agents (diabetic patients). All subjects were normotensive according to World Health Organization criteria (blood pressure < 140/90 mm Hg).

The urinary albumin concentration was measured in the timed urine samples by radioimmunoassay (interassay coefficient of variation, 2.5%). The lower limit of microalbuminuria was set at 20 μg/min in at least two of three consecutive 24-hour samples, the lowest detection limit being 1.5 μg/min. HbA_{1c} (normal range, 4.3% to 6.2%) was determined chromatographically using a Bio-Rad Hemoglobin A_{1c} Column Test (Bio-Rad Laboratories, Hercules, CA). Plasma glucose was assessed by a glucose oxidase method (Beckman Glucose Analyzer II; Beckman Instruments, Fullerton, CA).

Statistical Analysis

Statistical analysis was performed using one-way ANOVA. When differences were significant, Student's *t* test for unpaired data was used. Multiple logistic regression analysis was performed with e-OAE responses as the dependent variable and peripheral neuropathy, duration of diabetes, and HbA_{1c} as independent variables. Differences between proportions were analyzed by the chi-square (χ^2) test. All calculations were performed by a statistical package (Statgraphics 6; Manugistics, Rockville, MD). A *P* value less than .05 was considered statistically significant. The data are reported as the mean ± SD.

Table 3. Clinical Characteristics of Type 2 Diabetic Patients With e-OAEs+ and e-OAEs−

Group	Age (yr)	Duration of Diabetes (yr)	HbA_{1c} (%)	Plasma Glucose (mg/dL)*	Peripheral Neuropathy (%)	Retinopathy (%)	Nephropathy (%)
e-OAEs+ (n = 53)	45.1 ± 6.0	7.0 ± 3.9	6.5 ± 0.3	134.2 ± 29.5	23	36	17
e-OAEs− (n = 57)	51.0 ± 5.8	11.5 ± 4.4	6.9 ± 0.4	145.1 ± 30.9	46	37	19
	$P < .001$	$P < .001$	$P < .01$	P = NS	$P < .02$	P = NS	P = NS

Abbreviation: NS, nonsignificant.
*At the time of e-OAEs recording.

RESULTS

Study 1 (cochlear function and auditory pathway)

e-OAEs were impaired in 51.8% (57 of 110) of the diabetic subjects, as compared with 4.7% (five of 106) of the control group ($\chi^2 = 56.24$, $P < .0001$). Diabetics with impaired e-OAEs (e-OAEs−), in comparison to diabetics with normal e-OAEs (e-OAEs+), were older and had a longer duration of diabetes, poorer metabolic control, and greater peripheral diabetic neuropathy (Table 3). Nevertheless, when corrected for by multiple regression analysis, the correlation between sensorineural damage and peripheral neuropathy lost significance (Table 4). The mean values for plasma glucose were higher in e-OAEs− versus e-OAEs+ subjects, but the difference did not achieve statistical significance. No difference was observed between e-OAEs− and e-OAEs+ subjects for retinopathy or nephropathy.

The diabetic groups (e-OAEs+ and e-OAEs−) showed a longer latency for waves I, III, and V and interwave latency for I to V versus the control group, but there was no significant difference in ABRs between e-OAEs+ and e-OAEs− subjects (Table 5).

Study 2 (hyperglycemic clamp)

Neither type 2 diabetic nor control subjects had a significant change in e-OAE intensity with respect to basal values during the entire hyperglycemic clamp. No difference was observed between the two groups during each time of the clamp (Fig 1).

DISCUSSION

Two main observations justify the study of cochlear function in diabetes: (1) selective damage of OHCs has been observed in diabetic animal models,[8-10] and (2) diabetes is frequently associated with a sensorineural alteration of the inner ear that leads to hypoacusis. A recent study on cochlear function in type 1 diabetes[15] showed greater impairment of e-OAEs in diabetics with peripheral neuropathy. In the presence of retinopathy, a greater impairment of cochlear function was also found. There was no correlation between e-OAEs and HbA_{1c} or between e-OAEs and the duration of diabetes.

Our investigation is the first to demonstrate that in a population of type 2 diabetics the prevalence of cochlear compromise is higher than in matched controls. The damage seems correlated with the duration of diabetes, degree of metabolic control, and peripheral neuropathy. The sensorineural alteration appears to be associated with peripheral neuropathy, but does not seem to be linked to the compromise of the central nervous pathway. This supports the hypothesis that the CNS

Table 4. Results of Multiple Logistic Regression Analysis With Diabetes Duration, HbA_{1c}, and Peripheral Neuropathy as Independent Variables and Amplitude of e-OAEs as the Dependent Variable (age-corrected values)

Variable	Odds Ratio	95% Confidence Interval	P
Diabetes duration	1.187	1.102-1.272	<.05
Peripheral neuropathy	2.224	1.709-2.739	.12
HbA_{1c}	1.632	1.326-2.938	<.01

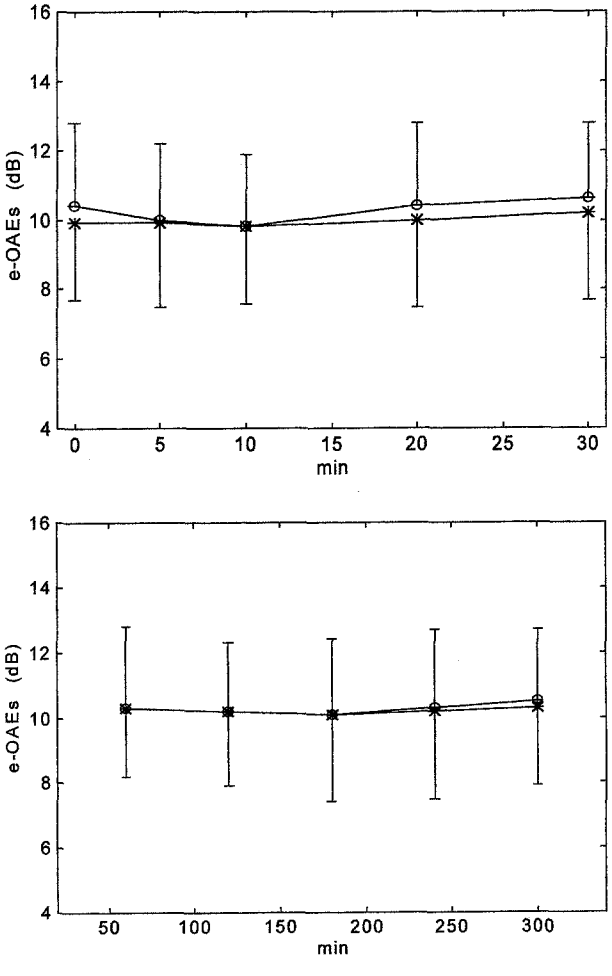

Fig 1. e-OAE amplitudes during hyperglycemic clamp in diabetic patients (x) and control subjects (o).

Table 5. ABR Recordings in Type 2 Diabetic Patients With e-OAEs+ or e-OAEs− and Control Subjects

	Wave			Interpeak		
Group	I	III	V	I-III	III-V	I-V
Diabetics						
e-OAEs+	1.65 ± 0.11*	3.88 ± 0.12*	5.80 ± 0.23*	2.22 ± 0.09	1.83 ± 0.14	4.11 ± 0.20*
e-OAEs−	1.68 ± 0.14*	3.90 ± 0.12*	5.83 ± 0.19*	2.22 ± 0.11	1.88 ± 0.20	4.13 ± 0.23*
Controls	1.60 ± 0.13	3.79 ± 0.15	5.57 ± 0.21	2.11 ± 0.10	1.81 ± 0.15	3.90 ± 0.20

*$P < .05$ *v* controls.

damage is less than the peripheral impairment, and thus a dichotomy between central and peripheral neuropathy exists, likely on the basis of different axon lengths and coating. The compromise of e-OAEs is related to functional cochlear damage. In the initial stage, this is not associated with manifest hypoacusis, often described in diabetes.[2-4,27-29] These observations call for a wider discussion on auditory function in diabetes. The association between cochlear damage and peripheral diabetic neuropathy can be explained as an epiphenomenon that has to be correlated with the duration of disease. In fact, if the influence of the diabetes duration is corrected for by multiple regression analysis, the association remains statistically significant with HbA_{1c} but not with peripheral neuropathy. Damage to the stria vascularis has been observed in animal models of diabetes,[5-6] and a recent investigation on type 1 diabetes found a correlation between retinopathy and a reduction of e-OAE amplitudes.[15] The lack of a significant difference in the prevalence of retinopathy in our type 2 diabetic e-OAEs+ and e-OAE− subjects may have several explanations. It could be that in type 2 diabetic subjects with long-standing disease and a high prevalence of retinopathy, any initial differences in microvascular damage between e-OAEs− and e-OAEs+ subjects lose significance. Moreover, the higher prevalence of e-OAE alterations in type 2 diabetes than cochlear dysfunction in type 1 diabetes[15] can contribute to this lost significance. It is likely that the cochlear changes in various components of Corti's organ described in diabetic animals[5-9] may play an even greater role than microvasculopathy in man. The physiological role of the osmotic mechanism in the genesis of e-OAEs, as well as the higher mean glycemia in e-OAEs− versus e-OAEs+ subjects at the moment of e-OAE recordings, observed in the first study led us to study the effect of acute hyperglycemia on e-OAEs. The absence of an influence of acute hyperglycemia clamped for several hours, on e-OAEs in both type 2 diabetic and healthy subjects and the presence of a significant correlation between glycemic control and cochlear function in type 2 diabetic subjects suggest that acute plasma hyperosmolarity does not cause sensorineural damage, which may be due to chronic hyperglycemia.

Nevertheless, independently of the e-OAE response, there was a delay in the absolute latency of waves I, III, and V and an increase in the interval latency for waves I to V in all diabetics. These findings, suggesting a delay of the central conduction time, agree with other studies[30] but cannot exclude cochlear receptor involvement in the dysfunction of the peripheral auditory pathway recently hypothesized in type 1 diabetes.[15]

In conclusion, type 2 diabetics show a higher prevalence of e-OAE compromise than healthy subjects. The cochlear dysfunction does not associate either with auditory nervous pathway injury or with diabetic microvasculopathy, and does not seem to be an expression of the classic chronic complications of diabetes. Therefore, the compromised OHCs might be a sensorineural deficit following chronic exposure to a hyperglycemic milieau that modifies the sophisticated biomechanics on which the genesis of e-OAEs is based.

It might be of interest to design a longitudinal study of diabetic subjects with normal hearing with pathological otoacoustic emissions to evaluate whether they are at risk to develop clinically evident hypoacusis. If so, e-OAEs might be an early marker of sensorineural deafness in diabetes.

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