Early Identification of Hearing Impairment in Patients With Type 1 Diabetes Mellitus

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Objective: The aim of this study was to evaluate the cochlear micromechanics and central auditory function in patients with type 1 diabetes mellitus and to identify the site of possible dysfunction.

Methods: Cochlear activity was evaluated by recording distortion product otoacoustic emissions (DPOAEs). DPOAEs were performed using an ILO 92 Otodynamics Analyser. Functional changes in the retrocochlear auditory pathway were evaluated by auditory brainstem responses (ABRs). DPOAEs and ABRs were measured in 42 normally hearing patients with type 1 diabetes mellitus aged 21 to 42 years, and 33 age- and sex-matched nondiabetic control subjects.

Results: Both of the groups (diabetic and control) had normal and undifferentiated results in tonal and impedance audiometry. ABR peripheral transmission time (wave I) and central transmission time (interpeak latencies I–V) were significantly delayed in the diabetic compared with normal subjects, and the mean amplitudes of various DPOAEs were significantly reduced in the diabetic patients compared with the control subjects.

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Auditory brainstem responses
In general, ABR morphology was normal in all the groups. Latencies for waves I, III, and V and interpeak latencies I to V were significantly longer in the diabetic patients than in the control group. Table 2A shows the mean ABR results in the control and diabetic subjects.

Relationship between auditory brainstem responses and diabetic microangiopathy
Our results showed no significant differences between the mean ABR latencies in the two groups of diabetic patients, those with microangiopathy and those without microangiopathy (Table 2B). In addition, no correlations were observed between microvascular complications, peripheral transmission time (wave I), and brainstem conduction parameters in diabetic patients.

Distortion product otoacoustic emissions
The mean DPOAE amplitudes were significantly reduced in the diabetic group compared with the control subjects. Reduced mean DPOAE amplitudes in diabetic patients were found in the region of middle and high frequencies, from f2 = 1000 to f2 = 6000 Hz (p < 0.05). Mean results are presented in Table 3.

Relationship between distortion product otoacoustic emissions and diabetic microangiopathy
Although the mean DPOAE amplitudes in patients with microangiopathy were found to be more reduced than the mean DPOAE in patients without microangiopathy, in general no significant differences between the two diabetic groups were found (Table 4). In addition, no correlations were observed between diabetic microangiopathy and DPOAE amplitude reduction.

DISCUSSION
The relationship between diabetes mellitus and hearing impairment has been studied for more than a century, and is still a matter of controversy. Conventional audiometric tests are not sensitive enough to detect the initial phases of sensory loss, nor are they capable of determining the site and pattern of this hearing loss. In our study, however, the combined use of different procedures for

RESULTS
Tonal and impedance audiometry
Both groups (diabetic and control) had normal and undifferentiated results in tonal and impedance audiometry.

Distortion product otoacoustic emissions
We recorded distortion product otoacoustic emissions (DPOAEs) in a group of diabetic patients. The mean DPOAE amplitudes in diabetic patients were significantly reduced compared with the control subjects. Reduced mean DPOAE amplitudes in diabetic patients were found in the region of middle and high frequencies, from f2 = 1000 to f2 = 6000 Hz (p < 0.05). Mean results are presented in Table 3.

Relationship between distortion product otoacoustic emissions and diabetic microangiopathy
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TABLE 2. Auditory brainstem latencies of control and diabetic subjects*

<table>
<thead>
<tr>
<th></th>
<th>I</th>
<th>III</th>
<th>V</th>
<th>I-V</th>
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<tbody>
<tr>
<td>A. Diabetic and control subjects</td>
<td></td>
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<tr>
<td>Diabetic</td>
<td>1.77 ± 0.12</td>
<td>3.93 ± 0.2</td>
<td>5.9 ± 0.23</td>
<td>4.09 ± 0.2</td>
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<tr>
<td>Control</td>
<td>1.66 ± 0.08</td>
<td>3.77 ± 0.09</td>
<td>5.62 ± 0.11</td>
<td>3.96 ± 0.13</td>
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<tr>
<td>p</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
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<tr>
<td>B. Diabetic groups without (group A) and with (group B) microangiopathy</td>
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<tr>
<td>Group B</td>
<td>1.79 ± 0.12</td>
<td>3.94 ± 0.22</td>
<td>5.92 ± 0.23</td>
<td>4.1 ± 0.2</td>
</tr>
<tr>
<td>Group A</td>
<td>1.74 ± 0.11</td>
<td>3.91 ± 0.16</td>
<td>5.87 ± 0.22</td>
<td>4.07 ± 0.2</td>
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<tr>
<td>p</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
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*Data given as means ± standard deviations.

monitoring cochlear function (DPOAEs) and central portions of the auditory pathway (ABRs) in diabetic patients showed alterations in cochlear micromechanics and the retrocochlear auditory pathway. These findings (i.e., cochlear dysfunction in patients with type 1 diabetes mellitus) are in agreement with previous studies by Alborn et al. (8), DiLeo et al. (9) and Simoncelli et al. (10). They can be explained by some of the published theories on the etiology of inner ear damage in diabetes. Histopathologic observations of the inner ear in diabetic patients have demonstrated a number of changes: thickening of the capillary walls in the stria vascularis (3,11), endolymphatic and perilymphatic hemorrhages (12), degenerative changes in the organ of Corti (13), and reduction of the outer hair cells (11,14).

All of these factors could alter the cochlear mechanics and consequently otoacoustic emissions (OAEs). Our results indicate the existence of alterations in the cochlear

<table>
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<th>Group</th>
<th>Frequency [Hz]</th>
<th>Stimulus level (L1 = L2)</th>
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</thead>
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<tr>
<td>Diabetic</td>
<td>1000</td>
<td>2000</td>
</tr>
<tr>
<td>p</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
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<tr>
<td>Control</td>
<td>10.6 ± 5.9</td>
<td>12.8 ± 6.7</td>
</tr>
<tr>
<td>p</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
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TABLE 4. Mean distortion product otoacoustic emission amplitudes and standard deviations in diabetic subjects with (group B) and without (group A) microangiopathy
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CONCLUSION

The combined use of different procedures for monitoring the central and peripheral portions of the auditory pathway in diabetic patients showed the existence of alterations in cochlear microanatomies and the retrocochlear auditory pathway. Compared with alterations in the central auditory pathway, the impairment of cochlear receptor function is relatively subtle in diabetes. The cochlea therefore seems less prone to damage than brain tissue. This may be because at the capillary level, brain tissue is more susceptible to anoxia resulting from diabetic angiopathy than the cochlea (1).

Hearing impairment in diabetic patients is in general mild and subclinical and can be detected early by accurate and objective audiometric methods. DPOAEs and ABRs seem to be able to detect the earliest cochlear and retrocochlear dysfunction in patients with type 1 diabetes mellitus.

REFERENCES


