The Journal of Laryngology and Otology
March 1993, Vol. **107**, pp. 179–182

Hearing loss in diabetics

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Abstract
The relationship between diabetes and sensorineural hearing loss has been disputed. This study compares 44 insulin-dependent diabetics with 38 age and sex matched controls. All had pure tone and speech audiometry performed, with any diabetic showing sensorineural deafness undergoing stapedial reflex decay tests. In 14 diabetics stapedial reflex tests showed no tone decay in any patient, but seven showed evidence of recruitment. Analysis of variance showed the diabetics to be significantly deafer than the control population. The hearing loss affected high frequencies in both sexes, but also low frequencies in the male. Speech discrimination scores showed no difference. Further analysis by sex showed the males to account for most of the differences. Analysis of the audiograms showed mostly a high tone loss. Finally duration of diabetes, insulin dosage and family history of diabetes were not found to have a significant effect on threshold.

Key words: Hearing loss, sensorineural; Diabetes mellitus, insulin-dependent

Introduction

The relationship between diabetes mellitus and sensorineural hearing loss has been a subject of debate since Jordao (1857) reported a case of hearing impairment with incipient diabetic coma. Some authors have confirmed hearing loss (Camisaca, 1950; Borsuk *et al.*, 1956; Jorgensen and Buch, 1961; Dietzel, 1964; Zelenka and Kozak, 1965; Devald *et al.*, 1966; Rozen *et al.*, 1972; Roach, 1973; Koslov, 1975; Taylor and Irwin, 1978; Wackym and Linthicum, 1986; Kurien *et al.*, 1989), quoting widely different incidences, while others have refuted it (Kindler, 1955; Marshak and Anderson, 1960; Axellson and Fargerberg, 1968; Axellson *et al.*, 1978; Gibbon and Davis, 1981; Strauss *et al*, 1982; Sieger *et al.*, 1983). The typical hearing loss described, is a progressive, bilateral, sensorineural deafness of gradual onset affecting predominantly the higher frequencies (Axellson and Fargerberg, 1968). This hearing loss is similar to that found in presbyacusis but is greater than would be expected. If sensorineural hearing loss *is* associated with diabetes, other points to be considered are:

(1) is the hearing loss cochlear or retrocochlear?
(2) is there any association with other aspects of the disease, such as duration of diabetes, treatment or family history?

The purpose of this study was to examine the relationship between diabetes and sensorineural hearing loss and, if any was found, to see if it was cochlear or retrocochlear, or related to other aspects of the disease. It was decided to limit the study to insulin-dependent diabetics as it was felt that if diabetes caused hearing loss, these patients would be the most likely to show it.

Patients and methods

Forty-four insulin-dependent diabetics were randomly selected from a diabetic clinic. They were compared with 38 controls drawn from staff and visitors to the hospital. Subjects with a past history of ear disease, exposure to excessive noise, ototoxic drugs, head or ear trauma or a family history of deafness, were excluded from each group. Excessive noise exposure was defined as heavy industrial noise, firearms, and, as there were many farmers in the community, exposure to farm machinery and chain saws. No age restrictions were placed on the diabetics as the controls were matched for age and sex. Subjects were matched as far as possible on a one to one basis, a control being specifically obtained for each diabetic as close in age as possible and of the same sex. This was more difficult in older subjects, where more leeway was permitted. Also as the national study of hearing (Davis, 1983) has shown that social class has an effect on hearing, the two groups were balanced as far as possible in this respect.

All subjects underwent an ENT examination and then had pure tone and speech audiograms performed in a sound proof room, using a Madsen® OB822 audiometer. Pure tone thresholds were obtained from 125 to 4000 Hz at octave intervals, both by air and bone conduction. Narrow band masking was used where indicated. Any diabetic who showed sensorineural hearing loss had stapedial reflex decay tests performed with a Grayson Staedler®

This work was carried out at Tyrone County Hospital, Omagh.
Accepted for publication: 15 October 1992.

middle ear analyser (model 1723), after the method of Owens (1964).

Results

Thirty-two male and 12 female diabetics (average age 46.9 years), were compared with 27 male and 11 female controls with an average age of 40.4 years. No subject in either group showed any evidence of conductive hearing loss. In fourteen diabetics, audiometric results were obtained which showed pure tone thresholds of between 15 dB and 35 dB across the middle frequencies. Stapedius reflex decay tests were done at 500 Hz and 1 kHz. No patient showed reflex decay at these frequencies. By contrast, seven patients showed evidence of recruitment, one patient did not show any stapedius reflexes and six were within normal limits.

The pure tone threshold and speech discrimination values were formally analysed by full analysis of variance (ANOVA) with left and right ear thresholds as dependent variables and group (×2), sex (×2), and frequency, including speech discrimination (×8) as the independent variables. Frequency was used as a repeated measure. As increasing age affects auditory thresholds, age was included as a covariate to minimize variance due to this effect. Sources of main effects and interactions were examined by one-way analysis of variance including Scheffe procedures.

Table 1 shows the ANOVA for all the data with respect to the independent variables. As can be seen, age does have a significant effect on threshold values, confirming its need for inclusion as a covariate. Significant main effects were seen for group, frequency, and sex. There was also a highly significant group by sex, and group by frequency interaction. A three-way analysis of group by sex and by frequency was not significant.

These main effects were further examined to detect exactly where the differences were arising. One-way analysis of the group by frequency interaction was performed and the results including the hearing level mean values are shown in Table II and illustrated graphically in Figure 1. This shows firstly that there is a significant difference at all frequencies in the left ear varying from $p<0.05$ for the low and middle frequencies to $p<0.001$ for the high frequencies. In the right ear there was no difference between the groups at 125, 250 and 500 Hz, but a significant difference was seen at frequencies above that maximal at the high frequencies ($p<0.001$). No difference was noted in speech discrimination for either group ($p>0.05$).

Thresholds were then compared for each ear with a group by sex variable to see if sex could account for some of the differences noted above. For this procedure all results were broken down into four groups for comparison:

(1) diabetic males.
(2) diabetic females.
(3) control males and
(4) control females.

One-way analysis using a Scheffe procedure for right and left thresholds, independent of frequency, showed a significant ($p<0.05$) left ear difference when diabetic males and diabetic females, were compared with males in the control group. A similar result was obtained when diabetic males were compared with females in the control group. The right ear showed a significant difference ($p<0.05$) between diabetic males and diabetic females compared with males in the control group. Further analysis was carried out at each frequency to see exactly where these differences lay. At 125 Hz diabetic males were significantly ($p<0.05$) deafer in both ears than their controls. They were also noted to be deafer than females in the control group ($p<0.05$). At 250 Hz diabetic males were deafer than their controls ($p<0.05$) in the left ear only. No differences in either ear were observed for any group at the frequencies 500 Hz, 1 kHz and 2 kHz. At 4 kHz, diabetic males differed significantly from their controls ($p<0.05$), while at 8 kHz both diabetic males and diabetic females were significantly ($p<0.05$) deafer than males in the control group.

The audiograms of the diabetics were also analysed to see if any particular pattern predominated. Eleven patients

TABLE I

GROUP BY SEX AND BY FREQUENCY ANALYSIS OF VARIANCE FOR LEFT AND RIGHT EARS

	F value	Degrees of freedom	p value
Left ear			
Age (covariate)	241.6	1	<0.001
Group	27.0	1	<0.001
Sex	4.25	1	<0.05
Frequency	508.7	7	<0.001
Group by sex	7.74	1	<0.01
Group by frequency	6.81	7	<0.001
Sex by frequency	0.60	7	N/S
Group by sex by frequency	0.48	7	N/S
Right ear			
Age (covariate)	242.0	1	<0.001
Group	20.59	1	<0.001
Sex	2.69	1	<0.05
Frequency	674.58	7	<0.001
Group by sex	9.31	1	<0.01
Group by frequency	7.68	7	<0.001
Sex by frequency	0.402	7	N/S
Group by sex by frequency	0.916	7	N/S

N/S = not significant.

TABLE II

ONE-WAY ANALYSIS OF VARIANCE OF GROUP (DIABETIC OR CONTROL) BY FREQUENCY (INCLUDING SPEECH DISCRIMINATION)

Frequency (Hertz)	Mean hearing Level (dB) diabetic	control	p value
Left ear			
125	7.76	13.18	<0.05
250	6.32	12.39	<0.01
500	5.79	10.57	<0.05
1000	4.61	10.68	<0.05
2000	7.37	15.0	<0.01
4000	13.42	30.23	<0.001
8000	14.87	33.82	<0.001
Speech discrimination	96.5	94.3	N/S
Right ear			
125	8.95	12.05	N/S
250	8.29	11.82	N/S
500	6.18	10.11	N/S
1000	5.66	9.43	<0.05
2000	7.76	13.75	<0.05
4000	13.42	26.93	<0.001
8000	14.61	33.03	<0.001
Speech discrimination	96.5	95.4	N/S

N/S = not significant.

https://doi.org/10.1017/S0022215100122571 Published online by Cambridge University Press

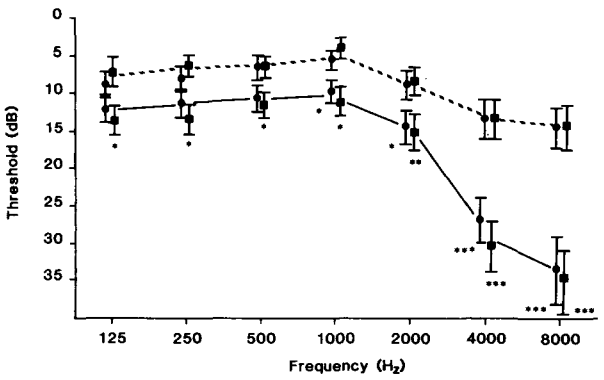

FIG. 1

Group audiograms comparing diabetics with controls. Right ear ○; Left ear ■; Diabetics ———; Controls - - - - -; $*p<0.05$; $**p<0.01$; $***p<0.001$.

had no discernible loss; eighteen showed a high tone loss, with a further nine showing a high tone notch and one other a unilateral high tone loss. Four cases showed a plateau loss with a superimposed high tone deficit. Finally one case had both high and low tone losses symmetrically.

Family history, duration of diabetes and dosage of insulin had no effect on pure tone threshold values.

Discussion

In a review of the recent literature, there have been six papers in which statistical analyses have been applied to the results. Of these three have supported the relationship between diabetes and sensorineural deafness (Friedmann *et al.*, 1975; Taylor and Irwin, 1978; Kurien *et al.*, 1989) and three have challenged it (Axellson and Fargerberg, 1968; Gibbon and Davis, 1981; Sieger *et al.*, 1983). Our results show diabetics to be significantly deafer than a control population overall. This hearing loss was most pronounced at the higher frequencies in both ears. A similar though less marked trend was also noted across the middle and lower frequencies, but was statistically significant in the left ear only. The data suggested that male diabetics were deafer than female diabetics: a high and low frequency difference was noted in male diabetics, but only a high frequency difference was noted in diabetic females when compared with the control group. Diabetic females were deafer than males in the control group but not compared with female controls. This latter finding was suggested by group mean analysis, with females in the control group having better average thresholds in the low frequencies compared with control males. This could be a function of the control females having abnormally raised threshold values, or men in the control group being deafer for the low frequencies at any specific age than females in this group. Therefore, it would seem that diabetic males are deafer than male controls at high and low frequencies. Diabetic females, however, in this study were not significantly deafer than their controls at any frequency, but were deafer than male controls at 8 kHz. Thus, most of the significant differences noted in this study seem to be carried by the males. These results differ from those obtained by Taylor and Irwin (1978), who found female diabetics to be significantly deafer than male diabetics but support the findings of Camisaca (1950) and Dietzel (1964). Axellson and Fargerberg (1968) found no sex difference. Davis (1983), in the National Study of Hearing, also noted a sex difference in the population studied. He found a difference in the mid-frequencies of 2 dB between males and females. This difference reduced to 0.3 db when account was taken of age, social group and noise exposure. He felt most of this difference was due to differing degrees of noise exposure in the two sexes. As well as this mid-frequency difference, he noted that males were deafer at high frequencies, but that females were deafer at low frequencies. Thus the finding of male diabetics being deafer than female diabetics at low frequencies in this study, is contrary to the finding in the general population, where women tended to have worse low frequency thresholds.

The relationship between the duration of diabetes and hearing loss has also been studied in the past. (Camisaca (1950) and Borsuk *et al.* (1956) found a positive relationship, while Jorgensen and Buch (1961), Dietzel (1964), Axellson and Fargerberg (1968) and Kurien *et al.* (1989) did not. No relationship was found to exist between duration of diabetes and hearing loss in our study. Dietzel (1964) and Taylor and Irwin (1978) also found that the dosage of insulin was not related to the degree of hearing loss and this has been confirmed here, although Kurien *et al.* (1989) noted poorly controlled diabetics to have worse thresholds than those who were well controlled. Diabetics with a family history of deafness did not have significantly worse hearing thresholds than those without such a history. This contrasts with the finding of Taylor and Irwin (1978).

Snashall (1977) and Gibbon and Davis (1981) noted tone decay to be present only at higher frequencies and suggested that this might be due to early presbyacusis. No evidence of stapedius reflex decay (which measures the same parameter) was found in the subgroup studied. The finding of lowered stapedial reflex levels in seven patients, indicating recruitment, is suggestive of a cochlear hearing loss, in this small subgroup, but the numbers are too small to draw any firm conclusions.

Jorgensen (1961) was the first to report on the pathology of 13 diabetic temporal bones. He found Periodic acid-schiff (PAS) positive thickening of the capillary walls of the stria vascularis making the vessels 10 to 20 times thicker than normal. This was confirmed by Costa (1967). Makashima and Tanaka (1971) also noticed this and commented that it led to narrowing of the lumen. They also noted loss of ganglion cells and demyelination of the VIIIth nerve sheath. Naufel and Shuknecht (1972) by contrast studied one case and found no lesion in the inner ear that could not be found in the inner ears of non-diabetics.

Three main theories exist as to the pathogenesis of the hearing loss: neuropathy (Bruhl, 1912; Edgar, 1915; Friedmann *et al.*, 1975), angiopathy (Edgar, 1915) and a combination (Edgar, 1915; Benesi and Somer, 1929). Since 1960 most workers agree that the primary lesion is an angiopathy (Jorgensen, 1961; Jorgensen and Buch, 1961; Zelenka and Kozak, 1965; Makashima and Tanaka, 1971; Rosen and Davis, 1971; Kovar, 1973; Koslov, 1975). The reasons supporting this are that histology has shown microvascular lesions in the inner ear and that diabetics are known have widespread vascular changes. Most workers feel that this interferes with the vascular supply to the cochlea or causes a secondary degeneration of the VIII[a] nerve. Recently Wackym and Linthicum (1986)

https://doi.org/10.1017/S0022215100122571 Published online by Cambridge University Press

compared the temporal bones from known diabetics with controls and calculated that the hearing loss was indeed due to microangiopathic involvement of the basilar membrane vessels and/or the endolymphatic sac. They suggest that the vascular thickening found round the endolymphatic sac may cause accumulation of toxic waste products in the endolymph, which in turn could cause hair cell dysfunction. They found vascular involvement of the basilar membrane to be associated with a significantly lower percentage of hair cells.

Jorgensen and Buch (1961) studied the relationship of hearing loss to other aspects of diabetes such as retinopathy, nephropathy and neuropathy. They found hearing loss twice as common in those with severe proliferative retinopathy. They also found hearing loss to be commoner in patients under 40 with nephropathy compared to those over 40 with it. No correlation was found between peripheral neuropathy and hearing loss. Furthermore Rosen and Davis (1971) found no correlation between severity of microangiopathy and degree of hearing loss in diabetics below 25 years of age, whereas this was present above 25 years. This finding of a correlation between peripheral microangiopathy was noted also by Dietzel (1964) and Zelenka and Kozak (1965). This is the only correlation not to have been challenged in the literature.

Conclusions

Diabetics were found to be significantly deafer than a control population. The hearing loss affected high frequencies in both sexes but in the male affected the low frequencies also. Diabetic men were found to be deafer than diabetic females compared with their respective controls. No difference was found in speech discrimination between either group. A subgroup of diabetics with mild sensorineural hearing loss showed evidence of a cochlear lesion in some cases. Finally, no significant effect on threshold was found in relation to duration of diabetics, insulin dosage or family history of diabetes.

Acknowledgements

We would like to thank Mr R. H. Allen and his staff for carrying out the audiological testing so efficiently and Dr Blair for allowing disruption of his diabetic clinic. Also Mr C. Patterson, Department of Medical Statistics, Queen's University Belfast, for his advice on setting up this project and statistical analysis.

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