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Hearing in acromegaly

by

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Abstract

Fifty-six individuals with acromegaly referred for pituitary surgery were studied to determine any relationship between acromegaly and sensorineural or conductive hearing loss. Compared to a matched population control sample, no significant difference between the acromegalics and controls has been found, either for air conduction or for bone conduction. The differences between these findings and those published previously are discussed, and reasons proposed to explain the discrepancies.

Introduction

Acromegaly is a chronic disease caused by excessive secretion of growth hormone by the pituitary, and is characterized by overgrowth of bone including the skull. It is a rare disease, affecting some 0.004 per cent of the population (Alexander *et al.*, 1980), but has received attention from otologists because of its potential effects on auditory structures. In acromegaly nearly all tissues are affected by excessive growth, and there exists the possibility that it could cause hearing loss by disturbed growth of the temporal bone or ossicles.

Menzel (1966) reported one case of a 72-year-old patient with acromegaly who had a bilateral sloping sensorineural hearing loss and postulated that this deficit followed hypertrophy of the temporal bone causing narrowing of the internal auditory canals. In one of the few papers available in the world literature, Maj *et al.* (1965) mentioned briefly the high incidence of a conductive or mixed hearing loss in a group of 34 acromegalics.

The most comprehensive study to date was reported by Richards (1968), and consisted of 15 patients. Five of the thirty ears had a conductive hearing loss considered to be due to otosclerosis because of surgical histology in two cases, and the absence of any other recognizable middle ear pathology in the other three. The group as a whole was compared with a control population from the United States of America (Glorig *et al.*, 1957), and it was claimed that they showed significantly increased hearing loss over the controls, though no statistics were quoted nor were the control levels shown. A further unexpected finding was an inverse relationship between the circulating growth hormone level and the degree of sensorineural hearing loss.

In a study of the findings in the temporal bone during middle ear surgery on three acromegalics, Graham and Brackmann (1978) found normal relationships of the middle and inner ear structures despite the marked hypertrophy of the mastoid cortical bone and posterior bony canal wall.

Thus the literature, while small and somewhat contradictory, would suggest a possible relationship between acromegaly and hearing loss, and a properly controlled study might help to clarify this potential relationship.

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Subjects

Fifty-six individuals, 22 males and 34 females, were studied. These patients were referred to the Institute of Neurological Sciences, Glasgow (a neurosurgical centre serving a population of some 2.5 million) for pituitary surgery. All fulfilled the following criteria:

(i) the usual clinical features of acromegaly
(ii) a fasting serum growth hormone level greater than 10 m*U*/l.

These patients were operated on between 1973 and 1981, and some of their relevant characteristics are shown in Table I. The individuals studied had a median age of 51.2 years (range 25–71 years), the median reported duration of disease was 5 years (range 1–17 years), and the pre-operative median growth hormone level was 65.0 m*U*/l (range 10–240 m*U*/l).

Method

The 56 subjects were assessed before and after surgery and underwent a clinical interview and audiometric testing. The clinical data included age, sex, social class, a noise exposure history, past and present drug ingestion, previous treatment for acromegaly, reported duration of acromegaly, presence of any diseases associated with acromegaly or auditory function, and an otoscopic examination.

The assessment of pure tone thresholds for both acromegalics and controls was conducted in a commercial sound-reduced room with ambient noise levels in which thresholds of 0 dB HL could be measured in a normal hearing person. Equipment was calibrated to ISO 389 and thresholds measured using a standard method (British Society of Audiology, 1981) at the frequencies 500, 1000, 2000, 4000 and 8000 Hz for air conduction, and 500, 1000, 2000 and 4000 Hz for bone conduction. Masking was applied to the contralateral ear where required using a shadow method (Coles and Priede, 1970). A control group matched for age, sex, social class and noise exposure, was selected for both the pre-operative and post-operative assessment from the random population sample in Glasgow obtained in the MRC Institute of Hearing Research's National Study of Hearing (MRC, 1981).

The data were coded and analysed on a PDP 11/60 computer using a standard statistical package (Nie and Hull, 1980). A preliminary analysis of the data showed that the conditions for the use of parametric statistics were satisfied, and accordingly Student's t-test for paired samples was applied to the air and bone conduction thresholds, broken down by frequency and ear, for the acromegalic and control groups.

Results

Table II shows the mean pre-operative air conduction and bone conduction thresholds for the right and left ears, over different frequencies for the two groups. There is no statistically significant difference ($p>0.05$) between the acromegalics and controls either for air or bone conduction at any frequency in either the left or right ears. A series of

TABLE I
CHARACTERISTICS OF THE PATIENTS ADMITTED TO THE STUDY

	Age yrs	Reported duration of disease yrs	Pre-operative growth hormone level m*U*/l	Post-operative growth hormone level m*U*/l
Mean	50.5	6.2	93.0	20.2
Median	51.2	5.0	65.0	5.3
Standard Deviation	11.7	4.9	133.9	48.6
Minimum	25	1	10.0	1.0
Maximum	71	17	840.0	240.0

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TABLE II
MEAN PRE-OPERATIVE THRESHOLDS DB HL

		Frequency Hz				
		500	1000	2000	4000	8000
Right ear						
Acromegalics	AC:	14.1	15.5	18.6	22.7	25.0
	BC:	16.0	14.5	19.0	23.1	—
Controls	AC:	16.9	16.8	16.4	22.7	30.0
	BC:	16.5	17.0	17.0	22.3	—
Left ear						
Acromegalics	AC:	15.9	13.4	17.7	29.5	28.9
	BC:	14.0	12.1	17.0	27.0	—
Controls	AC:	13.3	15.8	16.5	26.4	28.8
	BC:	15.5	15.5	16.6	25.7	—

similar analyses on the upper quartiles and 90th percentiles rather than the sample means again showed no difference between the acromegalic group and the matched controls. Similarly there is no significant difference between the air-bone gaps in the acromegalics and those of the controls, showing no evidence of conductive hearing loss associated with acromegaly.

The values in Table III show a similar breakdown for the mean post-operative thresholds. Again no significant difference between any pair may be observed. Also if the data in Table II and Table III are compared, no significant improvement in either air or bone conduction thresholds may be detected, despite the success of the surgery as evidenced by the pre- and post-operative growth hormone levels in Table I. Indeed the differences between Tables II and III show that the acromegalics suffer a progressive hearing loss with time similar to the control population.

We may define presumptive otosclerosis as an air-bone gap of more than 15 dB averaged over the frequencies 500 and 1000 Hz, when the ear is otoscopically normal. Three of the 112 acromegalics ears satisfied this definition while in the control ears the criteria were satisfied by 1 ear. There is no significant difference in the incidence of presumptive otosclerosis between these two populations.

Correlation coefficients were computed between the pre-operative growth hormone levels and the air and bone conduction thresholds both at individual frequencies and for the average of 500, 1000, 2000 and 4000 Hz (Table IV). None of these coefficients reached statistical significance.

It is possible that any relationship between

TABLE III
MEAN POST-OPERATIVE THRESHOLDS DB HL

		Frequency Hz				
		500	1000	2000	4000	8000
Right ear						
Acromegalics	AC:	18.4	19.1	16.5	27.7	34.8
	BC:	17.0	16.6	17.6	24.8	—
Controls	AC:	17.8	17.2	20.6	30.9	37.0
	BC:	16.9	16.5	19.7	28.5	—
Left ear						
Acromegalics	AC:	18.3	19.5	22.7	29.0	35.0
	BC:	19.3	16.7	21.4	29.4	—
Controls	AC:	16.6	16.9	21.2	28.1	35.2
	BC:	17.1	16.3	19.4	27.2	—

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TABLE IV
CORRELATION COEFFICIENTS BETWEEN GROWTH HORMONE LEVEL AND HEARING LEVELS AND AGE

	Growth Hormone Level	
	(i) Simple correlation coefficients:	
Mean AC 0.5–4 kHz	r = −.22	(p = 0.11)
Mean BC 0.5–4 kHz	r = −.19	(p = 0.14)
Age	r = −.31	(p = 0.04)
	(ii) Partial correlation coefficients with age partialled out:	
Mean AC 0.5–4 kHz	r = 0.02	(p = 0.83)
Mean BC 0.5–4 kHz	r = 0.04	(p = 0.78)

acromegaly and hearing may be influenced by factors such as patient age, disease severity (as measured by the growth hormone level), disease duration, previous medical treatment, previous radiotherapy and the presence of diabetes. Each of these potential effects was tested in an analysis of co-variance with hearing levels appropriately controlled for age, sex, noise and social class. In no instance was any significant effect, or any interaction between effects detected.

Discussion

The above results showing no effect of acromegaly on hearing are directly opposed to those quoted by Richards in 1968, and the differences deserve examination. Richards used as a control group data from the U.S.A. published in 1957, and suggested that his acromegalic group were significantly different. The control data as published by Glorig *et al.* were not gathered by a recognized random sample method and equipment calibrated to the ASA Standard (1951) was used, while we assume (though it is not stated) that Richards used equipment to ISO 389, the standard current in Great Britain at that time. In fact these two standards differ by some 11 dB, the ASA standard resulting in thresholds that are apparently more sensitive than the ISO standard. If Richards' control data are converted to ISO 389 (Katz, 1978), the apparent significant increase in hearing levels in his acromegalic group no longer exists. Thus it would seem likely that Richards' audiometric conclusions are an artefact of calibration differences between his sample and the controls. It is also possible that the controls were not suitably random and matched.

Another finding from Richards' data was an inverse relationship (when subjects with diabetes or a conductive hearing loss were excluded) between growth hormone level and degree of sensorineural hearing loss: this did not achieve statistical significance at the $p<0.05$ level in our sample. There is, however, a significant inverse correlation between age and growth hormone level, and when age is partialled out (part (ii) of Table IV) any apparent relationship between hearing level and growth hormone level disappears.

The reason for the apparent difference in incidence of otosclerosis or presumed otosclerosis between our acromegalic group and that of Richards' remains unresolved. The null result in our larger group suggests, however, that otosclerosis should not be listed as a clinically significant feature of acromegaly until the question is settled by an even larger series. Given the low prevalence of acromegaly, this would appear to require multicentre pooling of data, if a sufficient number of centres with adequate and similar recording procedures can be grouped.

Conclusion

The results from 56 individuals suffering from acromegaly have shown no significant relationship between the disease and its associated factors and either conductive or sensorineural hearing loss. These results support the findings in temporal bones, and while they contradict previous work, the present study with relatively large numbers and an adequate control group, has a degree of rigour lacking in previous reports.

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