# Temporary threshold drift in myxoedema

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HEARING loss in patients suffering from goitre was described by Bircher in 1883, and, in 1888, the Myxoedema Committee of the Clinical Society of London found 'auditory dysfunction' in 38 out of 69 patients suffering from myxoedema. In 1926 Lawrence published an audiogram of a patient with 'thyroid failure', showing a flat bilateral hearing loss which improved following thyroid therapy.

Lawrence did not specify the nature of the deafness, and for many years it was generally assumed to be conductive in type. Hilger (1956) published audiograms of 4 patients, 3 of whom had sensorineural losses, and the fourth an additional conductive component. This, and subsequent work by Marquet (1956), Ritter and Lawrence (1960), and De Vos (1963) suggests that the dominant otological lesion in acquired hypothyroidism is a flat sensorineural loss, with perhaps a small additional conductive component. The findings of Marquet (1956) and of De Vos (1963) showing an absence of recruitment, together with De Vos's finding of no abnormal fatigue, lead one to question the site of the sensorineural lesion. It is of note that to assess 'auditory fatigue', De Vos used Békésy audiometry, a technique that has been found (Stephens and Hinchcliffe, 1968) to be less sensitive than the Carhart (1957) manual audiometric technique. In their study, Stephens and Hinchcliffe found a significant correlation ($p<0\cdot001$) between a diagnosis of myxoedema, and fatigue or temporary threshold drift (T.T.D.) measured at 8,000 Hz by the Carhart technique. Taking a total shift exceeding 20 db. as an indication of T.T.D. by the Békésy method, (Hinchcliffe, 1959), a less significant correlation with myxoedema was found ($p<0\cdot01$), and if observable decay was taken as the criterion, the correlation was not significant.

However, in their study, Stephens and Hinchcliffe found that Carhart T.T.D. at 8,000 Hz was also significantly correlated with both the age of the subject and the hearing level. The aim of the present study was to compare the T.T.D. at 4,000 Hz and 8,000 Hz of 11 myxoedematous patients with a group of normal subjects whose average age, and 4,000 Hz bone conduction hearing level was the same as that of the patients with whom they were matched for sex.

## Procedure

*Subjects*

(a) Male myxoedema patients—Four patients who were included in the previous study by Stephens and Hinchcliffe.



https://doi.org/10.1017/S0022215100071929 Published online by Cambridge University Press

(b) Female myxoedema patients (n = 7). Seven patients, six from that study, together with one further subject tested at Charing Cross Hospital after the conclusion of the main study.

(c) Male control group (n = 5) selected from the large study to match the male myxoedematous patients for age and 4,000 Hz bone conduction threshold level. They were selected as having no overt auditory pathology.

(d) Female control group 1 (n = 14), this included one subject from the large study, suffering from surgically confirmed otosclerosis, matching a female myxoedematous patient with that condition. The other 13 subjects were chosen from the civilian subject panel of the M.R.C. Applied Psychology Unit, Cambridge.

There, 16 subjects were selected, merely on the basis of their age. Three of these were rejected on the basis of gross auditory pathology, following a screening examination.

(e) Female control group 2 (n = 10), this was as above except that four subjects who gave a history of basilar artery insufficiency, or idiopathic vertigo suggestive of possible underlying VIIIth nerve pathology were excluded.

## History and examination

An otological and a general medical history, concentrating on symptoms of thyroid dysfunction was obtained from each subject. A mirror examination of the ears, nose and throat of each subject was performed, together with Weber, Rinne and Bing tuning fork tests.

*Audiometric tests*

Routine auditory threshold determinations were performed on Groups (a), (b) and (c), in a sound treated booth using an Amplivox model 82 audiometer. Air conduction thresholds were determined for each subject and bone conduction thresholds for those with a negative Bing or occlusion test.

In Groups (d) and (e) similar determinations were made using a Peters model SPD5 commercial audiometer in a sound treated room.

Carhart T.T.D. determinations were performed at 250, 1,000, 4,000 and 8,000 Hz in the first three groups of subjects, and at 4,000 and 8,000 Hz in the subjects tested at Cambridge.

Békésy testing to assess T.T.D. was performed using the audiometer built by Burns and Morris (1956). This machine was used on all groups.

In any subject with abnormal hearing, various tests to assess recruitment were performed, i.e. Fowler's binaural loudness balance (1936), Reger's monaural loudness balance (1935), amplitude reduction in Békésy audiometry, and Watson's (1944) uncomfortable loudness level.



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*Tests of thyroid function*

The clinical diagnosis of myxoedema was confirmed by a number of tests. These were Protein bound Iodine, Triiodothyronine Resin uptake (Godden and Garnett, 1964), neck uptake of Iodine 131, plasma cholesterol, and anti-thyroid antibody studies.

## Results

Three of the myxoedematous patients had confirmed Hashimoto's disease (Hashimoto, 1912), one had had a thyroidectomy 18 years previously for thyrotoxicosis, and the cause of the thyroid dysfunction of the remaining seven was unknown.

The hearing of two of these patients was complicated by longstanding middle-ear pathology. One patient had surgically proven bilateral otosclerosis, and the other, unilateral chronic suppurative otitis media dating from scarlet fever in infancy.

The overall audiometric finding in these patients was a flat sensorineural hearing loss of the order of 30 to 40 db. H.L.(B.S.). In three cases this loss was complicated by a small conductive loss giving an air-bone gap of 5–15 db., negative occlusion tests, positive Rinne, and some degree of retraction of the tympanic membrane. In the absence of other aetiological factors, this conductive component appeared to have resulted from the auditory tube obstruction produced by myxoedematous deposits. There was no evidence of recruitment in any of the myxoedematous cases. The results of the Békésy assessed T.T.D. were described in the previous study and found to be a poor measure of the hearing loss in myxoedema. The Carhart T.T.D. is described below.

Table I shows the degree of matching of the various groups. The poorest match was with the males, and this may partially explain the low level of significance of the differences of the T.T.D. at 8,000 Hz found between the normal and control groups (Table III). A non-parametric test (Mann-Whitney) (Siegel, 1956) was performed on this data and showed a difference significant to $p<0{\cdot}05$ at 8,000 Hz, but not significant at 4,000 Hz.

TABLE I.

COMPARISON OF MYXOEDEMA AND CONTROL GROUPS.

| | | n | Age (yrs.) | | 4,000 Hz b.c. threshold | |
|---|---|---|---|---|---|---|
| | | | Mean | S.D. | Mean | S.D. |
| Male Subjects | Myx. | 4 | 62·7 | 11·6 | 43·1 | 13·9 |
| | Controls | 5 | 62·4 | 6·0 | 41·7 | 21·3 |
| Female Subjects | Myx. | 7 | 64·1 | 4·1 | 31·4 | 17·8 |
| | Controls (1) | 14 | 64·9 | 5·2 | 33·4 | 21·3 |
| | (2) | 10 | 64·9 | 3·8 | 33·2 | 19·1 |



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Tables II and III show that for the female subjects, the differences in T.T.D. between the myxoedematous group and the two control groups were significant at both 4,000 and 8,000 Hz. The higher level of significance found when the four subjects with histories of brainstem deficiencies were excluded, confirms previous findings that these conditions are commonly accompanied by abnormal T.T.D.

TABLE II.

COMPARISON OF T.T.D. AT 4,000 Hz.

| | | n (ears) | T.T.D. (db.) | | t—value | Significance |
|---|---|---|---|---|---|---|
| | | | Mean | S.D. | | |
| Males | Myx. | 8 | 20·6 | 19·0 | 0·44 | N.S. |
| | Controls | 10 | 16·5 | 17·6 | | |
| Females | Controls (1) | 28 | 6·4 | 12·1 | 2·77 | $p<0·02$ |
| | Myx. | 14 | 21·4 | 21·5 | | |
| | Controls (2) | 20 | 1·0 | 1·6 | 4·05 | $p<0·01$ |

TABLE III.

COMPARISON OF T.T.D. AT 8,000Hz.

| | | n (ears) | T.T.D. (db.) | | t—value | Significance |
|---|---|---|---|---|---|---|
| | | | Mean | S.D. | | |
| Males | Myx. | 8 | 18·75 | 10·2 | 2·13 | $p<0·1$ |
| | Controls | 10 | 8·50 | 8·7 | | |
| Females | Controls (1) | 27 | 15·4 | 17·2 | 3·43 | $p<0·01$ |
| | Myx. | 13 | 36·5 | 18·2 | | |
| | Controls (2) | 20 | 12·0 | 14·0 | 4·20 | $p<0·01$ |

## Discussion

The data presented above suggest that the T.T.D. at high frequencies found in cases of myxoedema by Stephens and Hinchcliffe represents a real finding and not merely an artefact due to the age and hearing levels of the patients concerned.

The Carhart measure of T.T.D. at 8,000 Hz appears to provide a very sensitive measure of neuronal lesions or degeneration, and suggests that the sensorineural lesion in myxoedema lies proximal to the hair cells. This is also suggested by the fact that no recruitment was found in the studies of Marquet (1956) and of De Vos (1963), a finding confirmed in the present study.

In his studies on experimental mice, rats, and hamsters, in which he induced hypothyroidism by treatment with propyl-thio-uracil, De Vos found degeneration of the spiral ganglion. Ritter's (1967) study on rats treated with Iodine-131 gave some suggestive evidence of lesions in the



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external sulcus and spiral ganglion cells, but his experiment was complicated by the fact that all his experimental animals appeared to develop a suppurative otitis media during the course of the experiment.

One of the few recent descriptions of autopsy findings was given by Price and Netsky (1966). In a patient who had suffered from longstanding myxoedematous ataxia, they found areas of focal degeneration in the cerebellar cortex, and in particular in the vermis, together with round granular glycogen containing bodies. They considered these bodies, which they termed 'myxoedema bodies' to be altered corpora amylacea. Unfortunately they make no reference to temporal bone findings.

In the absence of more refined neuro-otological diagnostic techniques and further evidence from autopsies, the site of the auditory lesion in myxoedema must remain speculative.

**Acknowledgements**

I would like to thank Mr. Lionel Taylor for the use of the audiometric facilities at Charing Cross Hospital, and the Medical Consultant Staff at Charing Cross and Fulham Hospitals, for allowing me access to their patients. I am grateful to my colleagues for their constructive criticism and advice.

REFERENCES

Bircher, H. (1883) *Der Endemische Kropf.* Basel.
Burns, W., and Morris, G. A. (1956) *J. Physiol. (Lond.)*, **131,** 4.
Carhart, R. (1957) *Arch. Otolaryng.* **65,** 32.
Clinical Society of London (1888) *Trans. Clin. Soc. Lond.*, **21,** Suppl.
De Vos, J. A. (1963) *J. Laryng.*, **77,** 390.
Fowler, E. P. (1936) *Arch. Otolaryng.*, **24,** 731.
Godden, J. D., and Garnett, E. S. (1964) *J. Endocr.*, **29,** 167.
Hashimoto, H. (1912) *Langenbecks Arch. klin. Chir.*, **97,** 219.
Hilger, J. A. (1956) *Ann. Otol. (St. Louis)*, **65,** 395.
Hinchcliffe, R. (1959) *J. Laryng.*, **73,** 795.
Lawrence, C. H. (1926) *Laryngoscope (St. Louis)*, **36,** 779.
Marquet, J. (1956) *Acta oto-rhino-laryng. Belg.*, **10,** 423.
Price, T. R., and Netsky, M. G. (1966) *Neurology (Minneap.)*, **16,** 957.
Reger, S. N. (1935) *J. acoust. Soc. Amer.*, **7,** 73.
Ritter, F. N. (1967) *Laryngoscope (St. Louis)*, **77,** 1427.
———, and Lawrence, M. (1960) *Laryngoscope (St. Louis)*, **70,** 393.
Siegel, S. (1956) *Nonparametric statistics for the behavioural sciences.* New York.
Stephens, S. D. G., and Hinchcliffe, R. (1968) *Int. Audiol.*, **7,** 267.
Watson, L. A. (1944) *Laryngoscope (St. Louis)*, **54,** 531.

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https://doi.org/10.1017/S0022215100071929 Published online by Cambridge University Press