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Main Article

Neuro-otological findings in tinnitus patients with normal hearing

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Abstract

Introduction: Tinnitus is usually associated with hearing loss, and patients with tinnitus and normal hearing are unusual. Neuro-otological findings have not previously been described in tinnitus patients with normal hearing.

Aim: To analyse neuro-otological examination results from a group of tinnitus patients with normal hearing.

Materials and methods: Seventeen normal-hearing tinnitus patients seen over a 10-year period were retrospectively evaluated. Their results were compared with those of a control group of 17 normal subjects without tinnitus.

Results: The main neuro-otological finding in the tinnitus patients was caloric test abnormality: a unilateral canal paresis was present in 15 of the 17 patients. Caloric tests were normal in 15 of the 17 control subjects.

Conclusion: We may infer from these results that tinnitus could be the only clinical manifestation of a cochlear – and presumably cochleo-vestibular – lesion, and that unilateral canal paresis may be the only abnormal finding on neuro-otological examination.

Key words: Tinnitus; Vestibular Function Tests; Hearing

Introduction

Tinnitus is the general term for a sound sensation experienced in the head or ears which cannot be attributed to an external sound source.[1] It is a common disorder which is very prevalent in the general population and causes considerable morbidity, and which may interfere with sleep, concentration, emotional balance and patients' social life.[2] Although tinnitus may occur in children, it is most common among the elderly, with an increase in prevalence as age increases.[3]

Tinnitus is usually associated with hearing loss,[4] and the severity of tinnitus is often found to increase with the degree of hearing loss.[5] It has been reported that 85 to 96 per cent of patients with tinnitus have some degree of hearing loss,[6] and that only 8 to 10 per cent have normal audiometry results.[7] The limited literature on patients with tinnitus but normal audiometry is restricted to the study of otoacoustic emissions,[8] auditory brainstem responses,[9] auditory processing,[10] high frequency audiometry[11] and zinc deficiency.[12] Although tinnitus characteristics have been described in patients with normal hearing,[13] neuro-otological findings have not been reported in such patients.

We report a neuro-otological clinical study in a group of 17 tinnitus patients with normal hearing.

Materials and methods

Of 457 patients examined over a period of 10 years (1998–2007), we retrospectively evaluated a group of 17 patients with normal audiometric findings (3.7 per cent) whose only symptom was tinnitus.

We excluded patients presenting with a history of hearing loss, a sensation of fullness in the ears or vestibular symptoms (such as vertigo, dizziness or gait ataxia). The same exclusion criteria were applied to control subjects.

All patients underwent a complete neuro-otological assessment, which included tests of stance and gait, cerebellar examination, audiological evaluation, caloric tests (Fitzgerald and Hallpike technique),[14] electronystagmography, and evaluation of spontaneous, positional and optokinetic nystagmus.

A control group of 17 normal subjects without tinnitus was also studied as a baseline. All these subjects had normal hearing and no auditory or vestibular symptoms. These subjects underwent caloric testing, audiometry (using an Interacoustic Clinical Audiometer

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AC 30; Interacoustics, Denmark), Romberg testing and Bárány's pointing test.

Results

The 17 patients in the tinnitus group comprised 11 (65 per cent) men and six (35 per cent) women, with a male–female ratio of approximately 3:2. Patients' age distribution ranged from 24 to 56 years, with a mean age of 44.29 ± 9.58 years.

In the tinnitus group, localisation of tinnitus was unilateral in 15 patients (10 in the right ear and five in the left ear) and bilateral in the two remaining cases. The tinnitus tone was high-pitched in eight patients, low-pitched in seven and of mixed pitch in the remaining two. Only one patient had tinnitus so severe it interfered with sleep. The clinical course of the tinnitus was classified as either fluctuating (i.e. tinnitus intensity significantly varied during the day; 11 patients), permanent (i.e. no changes in intensity; three) or progressive (i.e. intensity increased with time; three).

All patients showed normal and symmetrical pure tone thresholds in all frequencies (up to 25 dB HL in the frequency range 0.25 to 8 kHz). Gait was normal in all patients, and there was no evidence of neocerebellar signs, spontaneous nystagmus, positional nystagmus or abnormal optokinetic nystagmus. Although all patients were negative for the Romberg test, all exhibited a sensitised Romberg test response (i.e. when the patient was pushed sideways in the Romberg test position, they tended to lose balance to one side only). In all 15 patients with unilateral tinnitus, the Romberg test was sensitised towards the same side as the tinnitus. In the remaining two cases with bilateral tinnitus, the Romberg test was sensitised to the right, and right canal paresis was found. Bárány's pointing test was abnormal in five of the 17 patients (29.4 per cent). Of these five patients, the four with complaints of unilateral tinnitus had a deviation towards the same side as the tinnitus, and the remaining patient, with bilateral tinnitus, had an ipsilateral deviation to the side of the canal paresis. Caloric tests were abnormal in the 15 patients (15/17; 88.2 per cent) with unilateral canal paresis; the remaining two patients had normal caloric tests results. Tinnitus was unilateral in 13 of the 15 patients with unilateral canal paresis. In these 13 patients, 12 had tinnitus ipsilateral to the canal paresis; in the remaining patient, tinnitus was contralateral. In the two patients with bilateral tinnitus, the side of the canal paresis corresponded well with the side of the sensitised Romberg test.

One of our patients was of special interest, a 56-year-old man who complained of right-sided tinnitus (described as a 'buzzing sound') for more than three months. Originally present only in the mornings, the tinnitus had become more marked in the last month, being present during the entire day and also occurring at night. The patient denied hearing loss, a sensation of pressure in the ears, dizziness, vertigo or gait disturbance. Audiometry was normal and the Romberg test was negative, but a sensitised Romberg test to the right was found. Caloric tests showed a right canal paresis. This patient's tinnitus disappeared after some months, along with his sensitised Romberg test. However, he returned approximately 18 months later, having experienced a severe vertigo crisis. On this occasion, a markedly sensitised Romberg to the right was again found.

The control group comprised 17 healthy subjects with normal hearing and no auditory or vestibular symptoms, including tinnitus. There were 11 men and six women, with ages ranging from 25 to 62 years and a mean age of 43.88 ± 10.62 years. In marked contrast with the tinnitus group, the Romberg test was negative in all control subjects, as was the sensitised Romberg test. The pointing test was normal in 16 of the 17 subjects. Caloric tests were normal in the majority (15/17; 88.2 per cent), whereas unilateral canal paresis was found in the remaining two subjects (Figure 1).

Discussion

The higher incidence of tinnitus in men has been commented upon by other authors,[3] whose findings correlate very well with our own. Patients with tinnitus and normal audiometry are infrequent, with a reported incidence of 8–10 per cent.[7] A recent study of 744 tinnitus patients found that 7.4 per cent had normal hearing.[13] In the present study, among 457 tinnitus patients examined over a 10-year period, we found 17 (3.7 per cent) with normal hearing.

In the majority of our normal-hearing tinnitus patients (15 of 17), caloric tests indicated a unilateral canal paresis. Of these patients, 13 had unilateral tinnitus and two had bilateral tinnitus. In 12 of the 13 patients with unilateral tinnitus, the canal paresis was ipsilateral to the tinnitus. It is also important to note that the Romberg test was sensitised towards the same side as the tinnitus in all 15 patients with unilateral tinnitus. In four of five patients with an abnormal pointing test tinnitus was unilateral; in all of them tinnitus was ipsilateral to the canal paresis and to the side of the deviation of pointing test.

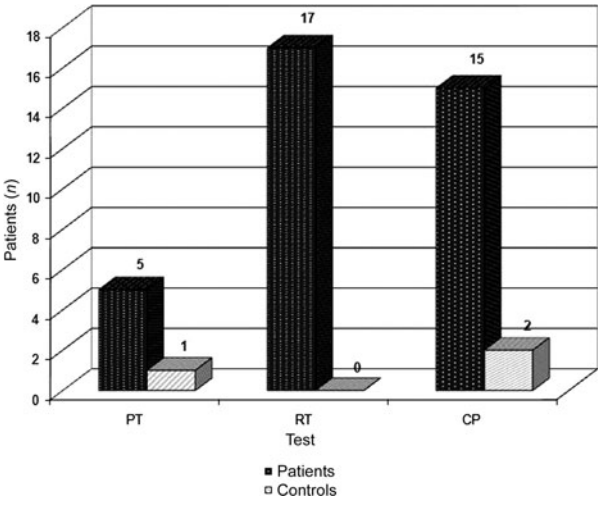

FIG. 1

Patients and control subjects with abnormal pointing test (PT), sensitised Romberg test (RT) and canal paresis (CP) results.

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In the remaining case with bilateral tinnitus, the pointing test deviated to the right and a right canal paresis was found.

In sharp contrast to the findings in our tinnitus patient group, none of our control group subjects had a sensitised Romberg test, 16 of 17 had a normal pointing test, and 15 of 17 had normal caloric tests.

It is interesting to note that patients with substantiated cochlear lesions may exhibit normal pure tone audiograms. Morales-García and Hood[15] reported two patients with normal pure tone audiograms in whom marked tone decay was found to occur. One had a cholesteatoma of the cerebellopontine angle, and the other had unilateral vestibular nerve section. A similar case was reported by Sörensen.[16] Schucknecht and Woellner[17] conducted an experimental study of hearing loss following partial section of the cochlea nerve, using a feline model, and showed that a large number of nerve fibres may be destroyed without affecting the hearing level. It may therefore be inferred that the pronounced tone decay observed in the above-mentioned two cases was the result of injury to the VIIIth nerve fibres.

- **Neuro-otological findings have not previously been reported in patients with tinnitus and normal hearing**
- **This paper presents neuro-otological findings in 17 such patients**
- **In 15 of the 17 patients, a unilateral canal paresis was found on caloric testing**
- **In 12 of 13 patients with unilateral tinnitus, canal paresis was ipsilateral to the tinnitus**
- **Tinnitus may be the only clinical manifestation of a cochlear, and presumably cochleo-vestibular, lesion, and unilateral canal paresis may be the only abnormal finding on neuro-otological examination**

Based on the previously reported fact that patients with cochlear lesions may have normal hearing, we may infer that, in our group of normal-hearing patients, tinnitus may be the only apparent manifestation of a cochlear – and presumably cochleo-vestibular – lesion. Upon neuro-otological examination of these patients, a unilateral canal paresis may be the only additional abnormal finding.

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