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ORIGINAL ARTICLE

Changes in audiometric thresholds before, during and after attacks of vertigo associated with Meniere’s syndrome

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Abstract

Conclusion: No significant changes in hearing thresholds were observed during vertigo attacks associated with Meniere’s disease.

Objectives: To determine if the hearing alters during the period of the attacks of vertigo in Meniere’s disease.

Patients and methods: The study group consisted of patients who had a clinical diagnosis of definite Meniere’s syndrome according to the AAOOHNS criteria, a score on the Gibson scale of 7 or over and an enhanced negative summating potential on transtympanic electrocochleography. These patients were supplied with a programmable hearing aid and a portable programmer that allowed them to measure their own hearing in situ. They were asked to measure their audiometric thresholds daily and if possible during the attacks of vertigo.

Results: Six of the patients were able to measure their hearing during attacks of vertigo and their hearing thresholds obtained before, during and after the vertigo attacks were compared. Five of six subjects showed \(B\) 10 dBHL change in the hearing levels at all tested audiometric frequencies before, during and after the attacks of vertigo. One subject had a probable change in threshold before the attack but not during the attack of vertigo.

Keywords: Rupture theory, dizziness, fluctuating hearing loss, hearing aids

Introduction

Changes in audiometric thresholds during the attacks of vertigo that occur in the course of Meniere’s disease have not been described previously. Nevertheless there is a general assumption that the hearing is worse during the attack. Change or lack of change of audiometric thresholds during attacks may help in the understanding of the mechanisms that cause the vertigo. Several possible theories have been suggested. The ‘rupture theory’ put forward by Schucknecht [1] was based on histological findings at autopsy, suggesting that the Reissner’s membrane ruptures due to increased endolymph volume with subsequent mixing of endolymph and perilymph. The potassium-rich endolymph temporarily paralyses the afferent nervous activity, causing an attack of vertigo. Ion pumps quickly restore the ionic equilibrium so that the vertigo ceases. The ruptured Reissner’s membrane is able to repair itself but some histological evidence of a past rupture can still be observed.

As Reissner’s membrane is within the cochlea, it can be assumed that a rupture has an effect on the cochlear function as well as the vestibular function. If the rupture theory is correct, hearing levels should deteriorate significantly while the attacks of vertigo are occurring. The purpose of this study was to compare audiometric thresholds during an attack of vertigo with an audiogram obtained within the previous 24 h and the audiogram obtained after the attack.

Patients and methods

Patients with Meniere’s disease were enrolled who had positive electrocochleography (EcochG) [2] results for endolymphatic hydrops, scored 7 or more on the Gibson’s scale [3] and were classified as ‘definite’ Meniere’s. in the AAOHNS scale [4].
Ethical approval for this study was obtained from Macquarie University Ethics Review Committee (Human Research).

The patients were fitted with Widex Senso Diva or Inteo hearing aids in the affected ear following McNeill’s procedure [5]. They were supplied with a portable programmer (SP3 to use with the Diva and an IP5 to use with the Inteo hearing aid), which enabled them to measure their own hearing thresholds via the hearing aid. Patients were asked to measure their own hearing up to three times a day for a period of 8 weeks.

All the patients were asked to try to record their hearing thresholds during a significant attack of vertigo and also to describe the features of the attacks. For many patients this was too gruelling a task but six did manage to record their audiograms during the episodes, often with the help of their partner. The history of each attack and the audiometric results are described.

Results
The severity of the attack of vertigo in each case is described as follows.

Case 1 (Figure 1)
A 54-year-old female had an attack of rotational vertigo with nausea but no vomiting that lasted for 1 h. She felt unsteady afterwards for 4 h. She did not notice any subjective change in her hearing during the attack.

Case 2 (Figure 2)
A 41-year-old female had an attack of rotational vertigo with nausea but no vomiting that lasted for 1 h. She felt unsteady afterwards for 2 h. She did not notice any subjective change in her hearing during the attack.

Case 3 (Figure 3)
A 42-year-old female had an attack of rotational vertigo with nausea but no vomiting that lasted for 2 h. She felt unsteady afterwards for 6 h. She did not notice any subjective change in her hearing during the attack.

Case 4 (Figure 4)
A 57-year-old male had an attack of rotational vertigo with nausea but no vomiting that lasted for 20 min. He felt unsteady afterwards for 1 h. He did not notice any subjective change in his hearing during the attack.

Case 5 (Figure 5)
A 51-year-old female had an attack of rotational vertigo with nausea and vomiting that lasted for 4 h. She felt unsteady afterwards for 12 h. She did not
notice any subjective change in her hearing during the attack.

Case 6 (Figure 6)
A 47-year-old female had an attack of rotational vertigo with nausea and vomiting that lasted for 4 h. She felt unsteady afterwards for 6 h. She noticed a deterioration of hearing a few minutes before the attack commenced but no obvious change during the attack. Hearing was better 12 h after the attack.

Discussion
The audiograms recorded by five patients showed no obvious changes in the thresholds during the attack of vertigo. One subject (case 6) had a deterioration of hearing but it is suspected that this occurred before the attack commenced.

The findings of this study do not support the rupture of Reissner’s membrane occurring within the cochlea in this group of patients. An influx of potassium should have drastically altered the endocochlear potential and should have caused a marked
deterioration of hearing levels. The audiometric thresholds were obtained at narrow sound frequency intervals and did not show an isolated drop at any frequency in any of these subjects.

Alternative theories must be developed. One theory is that the rupture or an ionic change only occurs within the vestibular portion close to the cristae of the semicircular canals [6]. Another theory was suggested by Gibson and Arenberg that when there was excess endolymph within the cochlea, episodes of longitudinal drainage of fluid to the endolymphatic sac could cause an attack of vertigo if there was reflux of endolymph past the valve of Bast into the utricle [7].

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