TRANSTYMPANIC ELECTROCOCHLEOGRAPHY FOR EVALUATION OF MENIERE’S DISEASE

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ABSTRACT

35 patients with unilateral sensorineural impairment and hearing levels within the normal range in the opposite ears were investigated by bilateral transtympanic electrocochleography (TTECoG). Only 19 patients (54%) satisfied the criteria for Meniere’s disease and 16 patients (46%) showed sensorineural impairment due to other etiologies. Clinical studies have focused exclusively on the amplitude ratio of the summating potential (SP) and the action potential (AP) derived from alternating polarity click responses (i.e., SP/AP). In only clinically defined ears with Meniere’s disease have abnormally large SPs been found. Neither normal ears nor sensorineural impaired ears of other etiologies exhibited abnormal SPs, hence we used normal ears as a control in this study.


INTRODUCTION

A number of reports on electrocochleography (ECoG) performed in clinical studies have shown that changes in the electrocochleogram can be specific for Meniere’s disease. The changes most frequently described are small and distorted cochlear microphonics (CM), high AP thresholds, steep increase of AP amplitude with stimulus level, prolonged AP latency, increased negative SP and wide AP-SP complex.1-6

In recent years, the amplitude ratio SP/AP has been reported to be increased in many cases of Meniere’s disease.7 Hypothetically the SP/AP can be enlarged because Meniere’s disease may alter the pressure gradient across the basilar membrane by increasing endolymphatic pressure. Durrant and Dallos8 reported the enlargement of SP on displacement of the cochlear partition toward the scala tympani. Biochemical mechanisms for alterations in cochlear potentials in endolymphatic hydrops have also been suggested.9

The technique we have used presently referred to as TTECoG was developed concurrently and independently in France.10 TTECoG, by means of a needle electrode passed through the tympanic membrane onto the promontory, produces excellent signal quality, requiring a minimum of signal averaging to obtain repeatable responses. In this study, we present results obtained with TTECoG in patients suspected of having unilateral endolymphatic hydrops with normal hearing in the opposite ears.

PATIENTS AND METHODS

Between 1990 and 1993 at the Khalili Hospital, affiliated to Shiraz University of Medical Sciences, 35 patients suspected of having unilateral endolymphatic hydrops with normal opposite ears were investigated by TTECoG. There were 22 females and 13 males, and their ages ranged between 22 and 75 years (mean age 47).

TTECoG was performed bilaterally and safely without using an anesthetic agent in the hands of an experienced otolaryngologist. Tympanograms (Madsen ZO 174) were performed after each TTECoG measurement bilaterally, and in all cases an intact tympanic membrane without any complication was noticed. The evoked potential equipment used for this study was the Madsen 2250.
TTECoG in Meniere's Disease

Fig. 1. Diagram to show the placing of the electrode on the promontory, and how it is held in place by means of an elastic band. On the left, the arrow points to the area in the tympanic membrane through which the electrode is introduced.

Fig. 2. Measurement of the summating potential (dotted line), and the action potential (continuous line) amplitudes in normal patients (A), those with sensorineural hearing loss (B), and those with Meniere's disease (C).

During testing the patients were awake and comfortably rested on an examining couch in a sound-proof room. The three electrode system was used. The sterilized active electrode on the promontory was a fine stainless steel needle 5 cm long and 0.2 mm in diameter insulated along its whole length except for the two ends. The electrode was held in angled forceps and under binocular operating microscope control, was lowered and made to pierce the tympanic membrane at a point half-way along a line joining the umbo to the sulcus at eight o'clock in the right ear (Fig. 1). The elastic band is then positioned on the electrode carrier. This active electrode was referenced to a hypodermic needle electrode pressed to the ipsilateral ear with a ground electrode on the forehead. The clicks with 100 μsec duration were presented at 100 dBHL and at a rate of 10 stimuli/s by shielded TDH-39 earphones. The signal from the electrode was directed into a biologic preamplifier with a system band pass filter of 5 to 3000 Hz. The amplified responses are then summed by an averaging computer. Sample size was 512, with an analyzing time of 10 ms. The alternating click AP, SP and CM amplitudes were measured on the monitor with a digital cursor. All recordings were repeated for all conditions to test reliability, which was usually excellent.

RESULTS

The SPs, characterized by their initial deflection amplitude, were compared between hearing-impaired ears and normal opposite ears of the individual patients. As it was readily apparent that the smaller SP amplitudes tended to occur with the smaller AP amplitudes, the results were analyzed in terms of the ratio of the SP amplitude to the AP amplitude (peak to peak). Using this method of analysis, a highly significant difference emerged between the sensory group and the Meniere group with hearing losses between 40-80 dBHL. An SP/AP ratio value of 28% clearly divides the two groups so that using a threshold value of 30% or greater, 96% of the Meniere's group were correctly identified with no false-positives.

The amplitude of the SP was compared with the AP using a 100 dB click stimulus regardless of the patient's hearing level. The SP was measured from the baseline, whereas the AP was measured from the N peak to the following P. A typical TTECoG to alternating polarity clicks from a normal, sensorineural impaired and Meniere's disease ears are shown in Fig. 2.

Three groups were studied: 1) 35 normal ears, 2) 16 ears affected by sensorineural hearing loss, in which endolymphatic hydrops was extremely unlikely, and 3) 19 ears affected by Meniere's disease, in which the hearing level varied from 40 to 80 dB for the click stimulus. In this

| Table I. Comparison of electrocochleography components in the three groups studied. |
|----------------------------------------|-----------------|-----------------|-----------------|
| Amplitude (μV) and ratio(%)            | Normals (0-20 dBHL) | Sensorineural Hearing Loss (40-80 dBHL) | Meniere's Disease (40-80 dBHL) |
| n=35                                   | n=16             | n=19             |
| SP                                     | Range     Mean  S.D. | Range        Mean  S.D. | Range        Mean  S.D. |
| 0.55-9.5                               | 3.95       2.68  | 0.1-7.5        1.13  1.6  | 1.3-19.54    4      3.3  |
| AP                                     | 2-52       18    12.4 | 0.6-29        6.95  6.5  | 3.5-31       8.1    6.3 |
| CM                                     | 2.1-16.5   9     4.4  | 0.2-19.5      6.8    4.7  | 2-16.5       7.5    4.6  |
| SP/AP                                  | 9.5-60     26    14   | 0.1-28        13     9.1 | 30-90.5      52     17  |

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last group there was good evidence that both endolymphatic hydrops and loss of hair cells were present (Table I) and all of them showed recruitment on short increment sensitivity index (SISI) and alternating binaural loudness balance (ABLB) testing. None of the seventy ears studied exhibited retrocochlear lesions on auditory brainstem response (ABR), tone-decay and reflex-decay tests.

As a result of this study, we are fairly confident that ECoG can be used to distinguish between conditions such as Meniere's disease that are associated with endolymphatic hydrops, and sensorineural hearing loss due to other etiologies.

**DISCUSSION**

In this investigation, we defined on clinical grounds a subset of patients with cochlear deficits whose conditions demonstrated endolymphatic hydrops based on audiometric findings, site of lesion tests, and ABR in order to differentiate cochlear lesions from retrocochlear or sensorineural impairment of other etiologies. Among these clinically defined ears with Meniere's disease, only those with recruitment showed a high incidence (54%) of abnormally large SP/AP amplitude ratios. Thus, we seem to have identified results of an objective diagnostic test, which correlates well with clinical evidence of endolymphatic hydrops.

With the use of absolute SP amplitudes, only about 13% of ears with Meniere's disease studied by Eggermont yielded positive results. However, with the use of the SP/AP amplitude ratio, his detection rates increased to about 42%. Therefore, for this reason we worked out our investigation results on the basis of SP/AP amplitude ratio which improves sensitivity and specificity. Eggermont's detection rates were nevertheless still below the rates we found. The following factors might have caused this difference: 1) different stimuli (rectangular-pulse click versus tone pip), 2) different recording sites (promontory versus ear canal), and 3) different clinical criteria for defining ears with Meniere's disease. Our investigation with regard to the incidence of enlarged SP/AP ratios among ears with Meniere's disease agrees more closely with the findings by Gibson et al. than with Eggermont's results. Hence, as the SP/AP amplitude ratio can discriminate between 19 ears with Meniere's disease, 16 ears with sensorineural impairments not associated with Meniere's disease and 35 normal hearing ears, its diagnostic value in any specific patient should be given paramount importance.

Because SP is a distortion product arising within the cochlea, it is very tempting to relate the negative SP encountered in TTTECoG recordings from patients with Meniere's disease to the actual presence of endolymphatic hydrops. With reference to this statement the following conclusions can be reached: 1) the SP/AP ratio appears to be the best diagnostic indicator of the presence of active Meniere's disease, 2) the SP/AP ratio increases as the hearing loss increases and as the disease becomes more established, and 3) when the CM were averaged in each group it was seen that CM diminishes as the disease becomes more established and less helpful clinically, as there is a wide range of values encountered in both normal and pathologic ears. Finally, from this investigation, it can be concluded that TTTECoG remains the best differential diagnostic tool for endolymphatic hydrops.

**REFERENCES**
