Cochlear Microphonics and Recruitment

CHAN LIU, XIUWU CHEN and LI XU

From the Research Department of Physiology, Beijing Institute of Oto-Rhino-Laryngology, Beijing, China

In this study, bilateral cochlear microphonics (CM) were evoked by tone burst simultaneously. A speaker was put in head-foot axis 2 m from the mid-point of a given line connecting the bilateral external meatus. Five normal persons and 68 cases (34 cases of Meniere's disease, 27 cases of sudden hearing loss, and 7 cases of low-tone sensory hearing loss without vertigo) with unilateral sensory hearing loss and recruitment, in addition to 2 cases of bilateral Meniere's disease with recruitment were examined. CM shifted in normal and hearing loss ears and was absent in profound and totally deaf ears. When recruitment was present, CM at corresponding frequencies were enlarged and prolonged in 50 cases. Some of the enlarged and prolonged CM decayed slowly, others quickly. Meanwhile the CM of the opposite normal ear decreased obviously. The presence of enlarged and prolonged CM may indicate an increase of abnormal excitability of the hair cells caused by some pathological stimulations. This would cause excitability of the hair cells in the opposite cochlea to be inhibited by the effect of the different system. In such a condition, the patients complained that the stimulating sound was heard louder in the disordered ear than that in the opposite normal ear. CM was slightly enlarged during sleep. *Key words: cochlear microphonics, recruitment.*

INTRODUCTION

Cochlear microphonics (CM) can directly reflect the functional status of cochlear sensory cells thus having a prospective diagnostic value in clinical audiology. However, so far clinical application of CM is still limited. In this study, bilateral CM were evoked simultaneously in cases with unilateral or bilateral sensory hearing loss, and in persons with normal hearing. CM were found to be enlarged and prolonged at some frequencies with recruitment. Recruitment has for many years been taken as an important sign by which to differentiate cochlear hearing loss from retrocochlear lesion. However, this sign is subjective and the mechanism is still obscure. We carried out this study in order to find an objective sign and the mechanism of recruitment.

MATERIAL AND METHODS

Five normal persons (2 male, 3 female, age range 20–49 years, average 38), 68 cases with unilateral sensory hearing loss, i.e. 34 cases with Meniere's disease, 27 with sudden hearing loss and 7 with low-tone sensory hearing loss without vertigo, in addition to 2 cases of bilateral Meniere's disease were tested. In all of the affected ears there was recruitment in the alternate binaural loudness balance (ABL) test and the score of short increment sensitivity index (SISI) test ≥ 75%. These two tests were performed at 0.5, 1, 2 and 4 kHz. The diagnosis of Meniere's disease was made after Pearson & Brackmann (1).

Electrocochleogram (CM), summating potential (SP) and action potential (AP), was evoked and recorded in a large, soundproof and electrically isolated room. Of the 68 cases with unilateral hearing loss, 47 were recorded intratympanically with needle electrodes and 21 extratympanically with ball electrodes. Five normal persons and 2 cases with bilateral Meniere's disease were all recorded intratympanically. The needle electrode was always pierce through the tympanic membrane at the mid-point of a given line extending horizontally from the umbo to the posterior margin of the tympanic membrane. The ball electrode was placed on
the clean skin surface of the posterial wall of the external auditory canal close to the attachment margin of the tympanic membrane. The reference electrode was placed on the homolateral ear lobe and the ground electrode on the root of nose. A signal processor was used in this experiment to give tone bursts, pick up and record the cochlear responses, and a sound-level meter B & K 2209 to measure the intensity of tone-bursts at the external auditory meatus. Clicks were delivered from an earphone of TDH 49 for evoking SP and AP. Basic noise was 16 dB SPL when the room was quiet, and 30 dB SPL when the instrument working. A speaker was placed in the head-foot axis, 2 m away from the mid-point of a given line connecting the bilateral ears. Tone bursts were given with frequencies of 0.5, 1, 2, 4, and 8 kHz in different intensities. The time of rise and decay of a tone burst was 2 ms, respectively, and 4 ms for maintenance. Bandpass filter was set from 320 Hz to 12 kHz for CM, and 80 Hz to 1.2 kHz for SP and AP. In order to compare bilateral CM, the binaries were stimulated simultaneously. The signals were summed up 512 times by an averager with an interval time of 75 ms and sweep time of 20 ms for 0.5 and 1 kHz, and 10 ms for 2, 4 and 8 kHz. Under such conditions, CM was readily evoked from ears with normal hearing or mild, moderate or even severe hearing loss, but could not be evoked from profound and totally deaf ears.

RESULTS

Biophysical properties of CM. In normal hearing ears, the wave form and frequencies of CM were similar to those of the stimulating tone. The amplitude and onset time of CM were correlated with frequency and intensity. At the same frequency; the larger the intensity, the higher the amplitude and the shorter the onset time, with the same intensity; the higher the frequency, the lower the amplitude and the shorter the onset time (Fig. 1). This phenomenon behind the change of onset time is CM shift (2). The detected threshold of CM was positively correlated to the subjective threshold of the pure tone audiogram in normal and affected ears without recruitment.

CM detected from normal ears. The amplitude of CM in 5 normal persons is shown in Fig. 2.
The absolute amplitude of CM varied widely among individuals but the amplitude was similar on both sides. For these reasons we did not adopt the mean value, but bilateral amplitudes were compared to evaluate the magnitude of CM.

Enlarged and prolonged CM at recruitment frequencies: Of 68 cases with unilateral hearing loss, CM were enlarged and prolonged at corresponding frequencies with recruitment in 60 cases (88%). These included 30 cases of Meniere’s disease, 23 cases of sudden hearing loss, and 7 cases of low-tone sensory hearing loss without vertigo. The enlarged and prolonged CM decayed slowly in 40 cases, i.e. the detected threshold of CM of the affected ear was equal to or lower than that of the opposite normal ear, while in another 20 cases the detected threshold of CM of the affected ear was higher than that of the opposite normal ear, or CM decayed rather quickly. In the meantime, the amplitude of CM in the opposite normal ear decreased. The external canal of the affected ear was obstructed with a sound-proof ear plug after which CM of the opposite ear were detected again. The result was the same as before. In 2 cases of bilateral Meniere’s disease with recruitment, CM were enlarged and prolonged at some frequencies in left ears and at other frequencies in right ears, in accordance with predominant recruitment (Fig. 3). This indicates that the presence of enlarged and prolonged CM at corresponding frequencies depends upon the existence of recruitment, irrespective of technical error. When the intensity of the stimulating tone is 80 dB SPL, the ratio of total amplitude of CM in the affected ear and the opposite normal ear is 53/28 at 0.5 kHz and 53/27 at 1 kHz. SP and AP were also detected in 30 cases of unilateral Meniere’s disease with enlarged and prolonged CM in the affected ears. There were 15 cases (50%) with enlarged SP (SP/AP > 40%) and 17 cases (57%) with enlarged AP in the affected ears.

Enlarged CM during sleep. If the patient fell asleep, CM were also slightly enlarged in both the affected and normal ear, and the detected threshold of CM during sleep is often (about 10 dB SPL) lower than that of the awake condition. When the patient was awakened, the amplitude of CM decreased to original level.
Fig. 4. CM type I. The CM of the affected (right) ear were enlarged with the detected threshold at 10 dB SPL, while in the opposite normal ear, the threshold of CM was also at 10 dB SPL.

Fig. 5. CM type II. The amplitude of CM of the affected (left) ear was obviously larger than that of the opposite normal (right) ear, but decayed quickly, with detected threshold at 50 dB SPL while in the opposite normal side the threshold was 10 dB SPL.

DISCUSSION

Gibbin et al. (3) reported on the prolongation of CM in man and termed this phenomenon CM ringing. They found in the cochlear and Meniere's pathology subgroup that there was a significant difference in CM amplitude between obvious ringing and no ringing, the former being significantly larger than the latter. Kumagami & Osawa reported in 6 cases with low-tone hearing loss that all AP, CM and -SP values were quite satisfactory as compared with 10 normal subjects (4). In this study, we found CM enlarged and prolonged at frequencies with recruitment in the affected ears as compared with the opposite normal ears. We also found enlarged AP and -SP in the affected ears though the positive percentage was not large, especially in those cases with a rather long remission period during CM detecting.

The recruitment phenomenon has been considered to have a close relationship with outer and inner hair cells (OHC & IHC). Positive summing potential (SP) from OHC presents inhibition to IHC in the normally functional cochlea (5). During endolymphatic hydrops SP turns negative which would cause a relief of the inhibition effect of IHC. When a strong stimulating tone was given, e.g. 80 dB SPL, the released IHC would give responses larger than normal. The result was revealed as enlarged and prolonged CM.

According to the detected threshold the enlarged CM were classified into two types. Type I including 40 cases revealed a lower threshold (20 cases) or the threshold equal to that of the opposite ear (20 cases), shown as Fig. 4. The pure tone audiogram often showed mild or moderate sensory hearing loss with complete recruitment. The hearing threshold fluctuated, and could be recovered or improved in most of the cases after proper treatment. All these
revealed that when the disease is in its early stage, the lesion of OHC is still slight. The enlarged CM took their source from both OHC and IHC. Type II, including 20 cases, had a higher threshold around 55 dB SPL (Fig. 5). The pure tone audiogram showed moderate or moderate-severe hearing loss with complete or incomplete recruitment. The hearing threshold could fluctuate and hearing of the affected ear could be improved. In this type, the enlarged CM would take their source from IHC only. Because of the high threshold of IHC, the CM from the affected ear decayed quickly. The number of cases of CM type I and type II presented in various types of sensory hearing loss is shown as Table I. Table II shows that the prognosis of 49 cases of sensory hearing loss with CM type I was much better than that of type II after 6 months’ follow-up.

In 8 of 68 cases with unilateral hearing loss (4 cases with Meniere’s disease and 4 with sudden hearing loss) the amplitude of CM of the affected ears was lower than that of the opposite normal ears. The average pure tone hearing threshold of the affected ears of all 8 cases was over 75 dB nHL, and the recruitment phenomenon appeared at 100 dB nHL or so. The maximum intensity of the stimulating tone for CM detection in this experiment was 80 dB SPL. This might be the reason why no enlarged CM could be found in these cases.

The enlarged and prolonged CM indicate that there exists some pathological excitability of the hair cells causing increased excitability in upper auditory neurons. Thus, by the effect of the efferent system, the negative feed-back is given to the OHC in the contralateral side, for the excitability of OHC in the opposite normal ear is inhibited so that low CM are exhibited. This effect of inhibition of the opposite OHC would come about because of the existence of recruitment whether the sound stimulation is present or not. Thus, when the affected ear is obstructed with a sound-proof plug there is no change of CM in the opposite ear. Patients often stated that the stimulating sound was heard louder in the affected ear with enlarged and prolonged CM than in the opposite normal ear.

A low hearing level with enlarged and prolonged CM if recruitment is present shows that CM can reflect the functional status of the hair cells but not the hearing.

CM was also slightly enlarged during sleep. Possible explanations would be one or both of

Table I. The number of cases of CM type I and type II in 60 cases of sensory hearing loss

<table>
<thead>
<tr>
<th></th>
<th>Total cases</th>
<th>Type I</th>
<th>Type II</th>
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<tbody>
<tr>
<td>Meniere’s disease</td>
<td>30</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Sudden hearing loss</td>
<td>23</td>
<td>16</td>
<td>7</td>
</tr>
<tr>
<td>Low-tone hearing loss</td>
<td>7</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>without vertigo</td>
<td>60</td>
<td>40</td>
<td>20</td>
</tr>
</tbody>
</table>

Table II. Prognosis of 49 cases of sensory hearing loss with CM type I and type II after 6 months’ follow-up

<table>
<thead>
<tr>
<th></th>
<th>Recovery (cases)</th>
<th>Markedly improved (≥30 dB nHL)</th>
<th>Improved (≥15 dB nHL)</th>
<th>Unchanged</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>9</td>
<td>6</td>
<td>5</td>
<td>9</td>
<td>29</td>
</tr>
<tr>
<td>Type II</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>19</td>
<td>20</td>
</tr>
</tbody>
</table>
following: A) During sleep the tonic activity of the muscles and the activities of the internal organs are reduced; B) An increase of the excitability of the hair cells induced by inhibition of cerebral cortex through some unknown neural pathway. How other factors such as general anaesthetics, excitation drugs, and retrocochlear lesions affect CM should be studied further.

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REFERENCES


Address for correspondence: Chan Liu, Beijing Institute of Oto-Rhino-Laryngology, 17 Hougou Lane, Chong-nai, Beijing, China