Action Potential Latency Shift by Rarefaction and Condensation Clicks in Meniere’s Disease

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Abstract
Transtympanic electrocochleography using rarefaction and condensation clicks was performed on 122 ears of 112 patients, including 98 ears of 89 patients with Meniere’s disease (MD) and 24 ears of 23 patients without Meniere’s disease (NMD). The mean action potential (AP) latency difference between rarefaction and condensation clicks was 0.40 ± 0.37 msec in the MD group and 0.06 ± 0.07 msec in the NMD group (p < .01). An AP latency shift > 0.2 msec was found more often in the MD group (62.2% vs 8.3%) and in ears with an enlarged summating potential and AP ratio (69.7% vs 36.4%). The results suggest that comparison of rarefaction and condensation latencies may serve as a useful addition to electrocochleography in the diagnosis of MD.

Key Words: Action potentials, condensation clicks, electrocochleography, Meniere’s disease, rarefaction clicks, summating potential

Abbreviations: AP = action potential; CON = condensation; ECoG = electrocochleography; MD = Meniere’s disease; NMD = non-Meniere’s disease; PTA = average of the pure-tone threshold hearing levels at 500, 1000, 2000, and 3000 Hz; RAR = rarefaction; SP = summating potential; SRAP = summating potential and action potential ratio

Electrocochleography (ECoG) has been used in the diagnosis of Meniere’s disease for more than 20 years (Portmann and Aran, 1971; Gibson et al, 1977; Ruth et al, 1988). Clinical evaluation of cochlear function has focused on the amplitude ratio of the summating potential (SP) and action potential (AP) derived from alternating polarity clicks.

Margolis et al (1992, 1995) have reported normal response patterns using rarefaction (RAR), condensation (CON), and alternating polarity clicks and tone bursts as stimuli in ECoG using a tympanic electrode. The authors concluded that one of the abnormalities in patients suspected of having endolymphatic hydrops was a latency difference between CON and RAR click-evoked APs.

Levine et al (1992) have reported normative data for ECoG amplitudes and latencies using a tympanic electrode and 13 normal subjects. The mean AP latency difference between RAR and CON clicks was 0.12 msec. Whitaker et al (1994) reported that in normal-hearing subjects, using a transtympanic needle electrode, the RAR and CON AP shift never exceeded 0.13 msec with a click stimulus.

The present study was designed to investigate the AP latency shift using RAR and CON clicks during transtympanic ECoG in a large sample of patients with Meniere’s disease and to observe the relation between the latency shift and an enlarged SP and AP ratio (SP:AP). Finally, the latency shift in patients with Meniere’s disease (MD) was compared to that found in a group of non-Meniere’s disease (NMD) patients.

PATIENTS AND METHOD

Subjects
Transtympanic ECoG using RAR and CON clicks was performed on 122 ears of 112 patients. There were 57 males and 55 females. The age
Table 1 Diagnosis in the Non-Meniere's Disease Group

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of Ears (n = 24)</th>
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<tbody>
<tr>
<td>Sensorineural hearing loss</td>
<td>10</td>
</tr>
<tr>
<td>Sudden hearing loss</td>
<td>6</td>
</tr>
<tr>
<td>Benign paroxysmal positional vertigo</td>
<td>3</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>3</td>
</tr>
<tr>
<td>Hyperacusis</td>
<td>1</td>
</tr>
<tr>
<td>Dysequilibrium</td>
<td>1</td>
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</tbody>
</table>

range of the subjects was from 19 to 84 years with a mean of 58.1 years. The procedure was performed in 66 right ears and 56 left ears, 102 unilateral and 10 unilateral. The patients were clinically divided into two groups, an MD group, which included 98 ears of 89 patients diagnosed with “definite Meniere's disease” defined by the Committee on Hearing and Equilibrium of the American Academy of Otolaryngology—Head and Neck Surgery (Committee on Hearing and Equilibrium, 1995), and an NMD group, which included 24 ears of 23 patients with various inner ear disorders (listed in Table 1). In the MD group, all ears presented one or more subjective symptoms including fullness, tinnitus, fluctuant hearing loss, and dizzy spells and 84 of 98 ears presented two or more symptoms at the time of EC0G testing.

Data Acquisition

The procedure of EC0G has been published in detail (Orchik et al, 1993). Briefly, the EC0G recordings were obtained using an auditory evoked potential system (Nicolet Pathfinder II, Nicolet Biomedical Instruments, Madison, WI). Biologic signals were amplified and bandpass filtered using a low-frequency cutoff of 5 Hz and a high-frequency cutoff of 1500 Hz. Test stimuli consisted of clicks in three polarity conditions: condensation, rarefaction, and alternating. The clicks were produced by 100-μsec square pulses and presented at a 90 dB nHL. The clicks were presented at a rate of 11.1 per second and an analysis time of 12 msec was employed. The noninverting electrode was a 5-cm Teflon-coated needle, manufactured by TECA Corporation, Pleasantville, NY. The needle electrode is placed on the promontory through an expandable foam earplug with a central plastic tube that connects to a remote earphone by another plastic tube. The inverting and ground electrodes were placed on the ipsilateral mastoid and forehead, respectively. Each signal average consisted of 128 or 256 stimulus presentations. The summed tracings were displayed on an oscilloscope. The AP latency measurements were made from the stored tracing by positioning the digital cursor available on the signal average. The AP latency was measured from the onset of the click stimulus to the N1 peak.

Analysis

An SP:AP of 0.40 and greater was employed as the diagnostic criterion of endolymphatic hydrops (Orchik et al, 1993). An AP latency difference of RAR and CON greater than 0.2 msec was defined as prolonged in this study. The use of an AP latency difference of RAR and CON greater than 0.2 msec is based upon data from a group of 24 ears of 23 patients with a diagnosis of sensorineural hearing loss, tinnitus, etc. The range of the AP latency difference is from 0 to 0.24 msec with a mean of 0.06 and standard deviation of 0.07. The AP latency difference of 0.2 represents two standard deviations above the mean.

Statistics

Student's t-test was used to compare groups. Statistical significance was set at a relatively stringent alpha level of .01 in order to minimize the probability of falsely rejecting the null hypothesis. Tests were two-tailed. Data were analyzed using Microsoft Excel software Version 4.0. The chi-square test was used to examine the relationship between the SP:AP ratio and AP latency shift. Statistical analyses were performed using Stata 4.0 for Windows.

RESULTS

The AP latency difference of RAR and CON clicks was prolonged in MD (Fig. 1). There was no significant difference of AP latency between RAR and CON clicks in NMD (Fig. 2). The mean AP latency of RAR clicks was 2.63 ± 0.27 msec and the mean AP latency of CON clicks was 3.00 ± 0.49 msec in the MD group (p < .01). The mean AP latency of RAR clicks was 2.68 ± 0.37 msec and the mean AP latency of CON clicks was 2.64 ± 0.39 msec in the NMD group (p > .01). The mean AP latency difference between RAR and CON clicks was 0.40 ± 0.37 msec in the MD group and 0.06 ± 0.07 msec in the NMD group (p < .01) (Fig. 3).

In the MD group, an SP:AP ratio > 0.40 was found in 76 of 98 ears (77.6%) and an SP:AP
A prolonged AP latency shift was related to an enlarged SPAP ratio ($X^2 = 4.753, p < .05$). In the NMD group, an SPAP ratio > 0.40 was found in 4 of 24 ears (16.7%) and an SPAP ratio < 0.40 was found in 20 of 24 ears (83.3%). An AP latency shift > 0.2 msec was found in 2 of 24 ears (8.3%) and an AP latency shift < 0.2 msec was found in 22 of 24 ears (91.7%). In the group with an enlarged SPAP, the mean AP latency of RAR clicks was 2.64 ± 0.28 msec and of CON clicks was 3.09 ± 0.51 msec ($p < .01$), and a latency shift of > 0.2 msec was found in 53 of 76 ears (69.7%).

In the group without an enlarged SPAP, the mean AP latency of RAR clicks was 2.59 ± 0.19 msec and of CON clicks was 2.68 ± 0.22 msec ($p > .01$), and a latency difference of > 0.2 msec was found in 8 of 22 ears (36.4%).

In the MD group, according to the staging system recommended by the Committee on Hearing and Equilibrium (1995), at stage 1, a pure-tone average (PTA) ≤25 dB, was found in 24 ears; stage 2, 26 to 40 dB, in 17 ears; stage 3, 41 to 70 dB, in 52 ears; and stage 4, >70 dB, in 5 ears. The mean AP latency differences between RAR and CON clicks in the MD group with different hearing thresholds are in Table 2. The mean AP latency shifts were similar across groups with the exception of stage 4. However, there were only five ears in this group and the implication of the smaller shift in this group is unclear.

**DISCUSSION**

In the clinical application of ECoG, two approaches of electrode placement have been used: transtympanic and extratympanic. Each

<table>
<thead>
<tr>
<th>PTA</th>
<th>Latency Shift (Msec)</th>
<th>SD</th>
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<tr>
<td>≤25 dB (n = 24)</td>
<td>0.43</td>
<td>0.46</td>
</tr>
<tr>
<td>26-40 dB (n = 17)</td>
<td>0.44</td>
<td>0.33</td>
</tr>
<tr>
<td>41-70 dB (n = 52)</td>
<td>0.39</td>
<td>0.36</td>
</tr>
<tr>
<td>&gt;70 dB (n = 5)</td>
<td>0.10</td>
<td>0.16</td>
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</table>
has advantages and disadvantages, which have been discussed in detail in a previous publication (Orchik et al, 1993). AP amplitudes obtained with a trans tympanic electrode are 5 to 10 times greater than those obtained with an extratympanic electrode (Coats, 1986). Even though responses obtained with extratympanic ECOC diminish rapidly with increasing electrode distance from the tympanic membrane, the latency is not affected with the presence of AP. For the purpose of study of AP latency, both approaches present similar results. In a recent study, Margolis et al (1995) placed the electrode on the tympanic membrane with microscopic visualization and obtained substantially larger responses. The authors reported that a latency difference between CON and RAR click-evoked APs is one of the indicators for patients with endolymphatic hydrops. Transtympanic ECOCG is regularly used to obtain reliable responses in the diagnosis of MD in our clinic and was the approach used in this study.

There are two phases of a stimulus during sound stimulation of the ear. The RAR phase occurs while the earphone diaphragm moves away from the tympanic membrane; the CON phase occurs while the earphone diaphragm moves toward the tympanic membrane. During the RAR phase, the basilar membrane motion is toward the scala vestibuli, while during the CON phase the basilar membrane motion is initially toward the scala tympani (Pierson and Moller, 1980). The hair bundle deflection toward the tallest row of stereocilia, as the displacement of the basilar membrane is in the direction of the scala vestibuli, results in depolarization of the cell. Conversely, the deflection of the hair bundle moving away from the tallest stereocilia, as the displacement of the basilar membrane is in the direction of the scala tympani, causes hyperpolarization of the cell. In normal ears, hair cells are initially stimulated by RAR stimuli while initially inhibited by CON stimuli. The latency shift occurs because neurons discharge during the displacement of the basilar membrane toward the scala vestibuli, which occurs earlier for the RAR pulse than for the CON pulse (Peake and Kiang, 1962; Kiang et al, 1965). Simmons and Glattke (1975) observed that depolarizing responses were associated with a membrane resistance decrease, and hyperpolarizing responses with a resistance increase. In hydropic ears, an increase in endolymph volume alters the mechanical characteristic of the basilar membrane. When the pressure in the scala media is increased, vibration toward the scala tympani would be limited and the movement toward the scala media would be enhanced (Morrison et al, 1980). That is, during the CON phase, the initial downward movement of the basilar membrane is limited by the hydrops and hence slowed; the inhibition phase is prolonged and the latency is delayed. This is a possible explanation for the longer delayed response to CON stimuli.

The results of the present investigation indicate that there is a relationship between an enlarged SP:AP ratio, which indicates endolymphatic hydrops, and a prolonged AP latency shift ($X^2 = 4.753, p < .05$). The mean AP latency difference between RAR and CON was 0.40 msec in the MD group and 0.06 msec in the NMD group. Moreover, the mean latency shift of the patients with an enlarged SP:AP is 0.4 msec while that of the patient with SP:AP < 0.40 is 0.1 msec. This relationship is further supported by the fact that 69.7 percent of the patients with an SP:AP > 0.40 present a >0.2-msec latency shift, and only 36.4 percent with an SP:AP < 0.40 have a >0.2-msec latency difference between RAR and CON.
The stiffness due to hydrops has a different impact on the RAR than on the CON phase. During the RAR phase, the earphone diaphragm moves away from the tympanic membrane, and then the basilar membrane moves upward toward the scala vestibuli, which stimulates the spiral ganglion. During the CON phase, the earphone diaphragm moves toward the tympanic membrane and then the basilar membrane moves downward toward the scala tympani, which inhibits the spiral ganglion. In the hydropic ear, the downward movement of the basilar membrane is limited by the hydrops (Morrison et al, 1980); hence, the time required to generate the AP is prolonged. Subsequently, the CON phase will have an enhanced inhibition phase before stimulation. In the case of RAR, the basilar membrane moves upward and the stiffness decreases in relation to the initial stiffness with hydrops, so the impedance decreases. Thus, the conduction velocity is not significantly decreased compared to the nonhydropic condition. Therefore, the RAR phase will have a slightly prolonged stimulation period without prior inhibition. In consequence, the latency difference between these two phases is amplified.

CONCLUSION

The amplification of latency difference in response to RAR and CON clicks in Meniere's patients is due to endolymphatic hydrops. Endolymphatic hydrops causes a pressure increase in the scala media and pushes the basilar membrane downward toward the scala tympani. The extension of the basilar membrane increases its stiffness and, hence, limits its motion toward the scala tympani, while the membrane's motion toward the scala vestibuli will decrease its stiffness; therefore, the motion is not limited. The slower motion toward the scala tympani prolongs the inhibition phase and further delays the AP in response to the CON click since its initial motion is toward the scala tympani. There is no significant change in response to the RAR click. Therefore, the latency difference in patients with endolymphatic hydrops is amplified.

Clinical diagnosis of MD depends on a detailed history and the presence of classic symptoms. The Committee on Hearing and Equilibrium of the American Academy of Otolaryngology—Head and Neck Surgery (Committee on Hearing and Equilibrium, 1995) has devised a diagnostic scale based on clinical criteria. At the present time, the confirmation of endolymphatic hydrops in MD patients depends on the histopathologic studies of temporal bones. Recent advances in ECoG testing make it possible to evaluate endolymphatic hydrops in a clinical setting. Pou et al (1996) reported that ECoG has been used as an objective electrophysiologic test in the clinical diagnosis of endolymphatic hydrops. We have focused on the SP:AP ratio derived from alternating polarity click responses as a reliable test to detect the presence of endolymphatic hydrops to confirm the diagnosis of MD and to evaluate the results of treatment. The results of the present investigation suggest that comparison of the CON and RAR latency shift may serve as a useful addition to the application of ECoG in the diagnosis of MD.

REFERENCES


