MISMATCH NEGATIVITY IN AUDITORY NEUROPATHY PATIENTS

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ABSTRACT
Mismatch negativity (MMN), is a negative component in the auditory event-related potential. There has been increased interest in using the MMN as a clinical diagnostic tool because it might provide an objective neural measure of auditory discriminability. Auditory neuropathy (AN) is characterized by a paradoxical absence of auditory brainstem evoked potentials with presence of otoacoustic emissions, in patients whose pure-tone thresholds were slightly elevated. The present study was designed to investigate the detectability of MMN in cochlear hearing loss and AN patients and to test the effectiveness of MMN as an indicator of auditory discrimination at cortical level, particularly in patients with AN, if any. This study consisted of sixty subjects divided into three groups: (Group 1) 20 AN patients, (group 2) 20 patients with bilateral moderate SNHL of cochlear origin and (group 3) 20 normal peripheral hearing subjects. All participants were submitted to: full medical history, otoscopy, basic audiological evaluation, TEOAEs, ABR for neurotologic diagnosis and MMN testing. The results of the present study demonstrated that SNHL had a significant impact on the timing of the brain processes involved in the detection and discrimination of stimuli. Moreover, no significant differences were found between AN patients and patients with cochlear hearing loss as far as MMN latencies.

Key words: Mismatch negativity test, auditory neuropathy.

INTRODUCTION
Mismatch negativity (MMN), a negative component in the auditory event-related potential (Naatanen et al. 1978), is thought to index automatic processes involved in sensory or
solve this problem by stimulating the spiral ganglion cells directly (Shallop et al. 2001).

There has been increased interest in using the MMN as a clinical diagnostic tool because this deviant-evoked negativity might provide an objective neural measure of auditory discriminability. One idea has been to employ the MMN as a test of the auditory system's ability to transmit the acoustic information important for understanding spoken language (Picton, 1995; Naatanen, 1995; Kraus et al., 1995). To date, the MMN has been used to assess the efficacy of phoneme discrimination training and related neural plasticity (Kraus, et al., 1995; Tremblay et al., 1997), and some investigators have proposed using MMN to monitor the effectiveness of hearing aid therapy (Picton, 1995) and cochlear implants (Ponton and Don, 1995).

The present study was designed to investigate the detectability of MMN in cochlear hearing loss and AN patients and to test the effectiveness of MMN as an indicator of auditory discrimination at cortical level, particularly in patients with auditory neuropathy, if any.

SUBJECTS AND METHODS

A. Subjects:
This study consisted of sixty subjects divided into three groups:

Group 1 : 20 patients diagnosed as AN, selected according to the following criteria:

a- Bilateral mild to moderate low frequency SNHL.

b- Poor speech discrimination scores disproportionate to the degree and configuration of hearing loss.

c- Normal middle ear pressure with elevated or absent acoustic reflexes.

d- Preserved transient evoked otoacoustic emissions (TE-OAEs).

e- Bilateral absent or severely distorted auditory brainstem responses (ABR).

f- Normal MR imaging of the brain.

Group 2 : 20 subjects with bilateral moderate cochlear hearing loss.

Group 3 : 20 subjects with bilateral normal peripheral hearing.

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MMN response parameter measurements:
1. Onset latency (L1): measured (in msec) from the stimulus onset to the onset of the response.
2. Peak latency (L2): measured (in msec) from the stimulus onset to the maximum negative peak of the response.
3. Offset latency (L3): measured (in msec) from the stimulus onset to the offset of the response.
4. Peak amplitude (A1): measured (in microvolts) from the baseline to the maximum negative peak of MMN.
5. Onset to peak amplitude (A2): measured (in microvolts) from the onset point to the maximum negative peak of MMN.
6. Peak to offset amplitude (A3): measured (in microvolts) from the maximum negative peak of MMN to the offset point.

Statistical procedure:
Results were entered into a computer and analyzed statistically using SPSS version 10.0. Descriptive statistics was performed for the three groups. One way ANOVA test was done to compare MMN latency and amplitude, among the three groups. Post Hoc test was done to validate the correlation between the three groups. Probability of $P < 0.05$ was considered statistically significant.

RESULTS
Demographic data of the studied three groups were illustrated in table (1). They were age and sex matched. Their mean pure tone thresholds were illustrated in figure (2).

Speech discrimination, TEOAEs, and ABR findings:

Group I (AN patients):
Their speech recognition scores ranged from 0% to 40% correct with an average of 16% correct. All subjects had measurable TEOAEs, with absent ABR. In addition, MR imaging of the brain was normal in all 20 patients tested.

Group II (Cochlear HL patients):
Their speech recognition scores ranged from 76% to 100% correct with an average of 84% correct. All subjects had absent TEOAEs, but ABR revealed well identifiable and repeatable waves with normal absolute and interpeak latencies.

Group III (Normal hearing subjects):
Their speech recognition scores
Table (2): Group effect on MMN latency measures (msec) using ANOVA test.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Moderate SNHL</th>
<th>Auditory neuropathy</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L1</td>
<td>178.70 ± 11.41</td>
<td>213.50 ± 13.30</td>
<td>226.95 ± 16.55</td>
<td>35.19</td>
<td>0.000*</td>
</tr>
<tr>
<td>L2</td>
<td>212.00 ± 12.99</td>
<td>248.00 ± 17.49</td>
<td>253.81 ± 12.25</td>
<td>27.08</td>
<td>0.000*</td>
</tr>
<tr>
<td>L3</td>
<td>250.70 ± 12.75</td>
<td>276.63 ± 9.57</td>
<td>276.63 ± 13.07</td>
<td>17.39</td>
<td>0.000*</td>
</tr>
</tbody>
</table>

* P < 0.05

Table (3): Group effect on MMN amplitude measures (microvolts) using ANOVA test.

<table>
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<tr>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
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<tr>
<td>A1</td>
<td>5.69 ± 1.58</td>
<td>5.20 ± 0.88</td>
<td>5.12 ± 1.62</td>
<td>0.522</td>
<td>0.598</td>
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<tr>
<td>A2</td>
<td>3.62 ± 1.89</td>
<td>3.56 ± 1.08</td>
<td>4.19 ± 1.40</td>
<td>0.599</td>
<td>0.556</td>
</tr>
<tr>
<td>A3</td>
<td>5.88 ± 2.21</td>
<td>5.44 ± 0.75</td>
<td>4.20 ± 2.43</td>
<td>2.191</td>
<td>0.129</td>
</tr>
</tbody>
</table>

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DISCUSSION

Cortical event-related potentials has been successfully used to assess the cognitive processes involved in the detection and discrimination of complex stimuli as speech sounds, in normal-hearing subjects (Martin et al., 1999). There has been increased interest in using the MMN as a clinical diagnostic tool because this deviant-evoked negativity might provide an objective neural measure of auditory discriminability. MMN is used as a test of the auditory system’s ability to transmit the acoustic information important for understanding spoken language (Picton, 1995; Naatanen, 1995; Kraus, et al., 1995).

In the present study, MMN latencies were significantly prolonged for the two hearing-impaired groups (AN and cochlear hearing loss) in comparison to those obtained from the normal hearing subjects. This agreed with Korczak et al. (2005). They suggested that the brain is not processing the acoustic signals with the same degree of accuracy and effectiveness as it is in individuals with normal-hearing sensitivity. There was no statistically significant difference between the cochlear HL and AN patients as regards latency measures of MMN. This specific pattern of electrophysiological finding provide evidence that the signal has been neurally coded at the level of the cortex and the brain is able to discriminate the acoustic changes present in the signal on a preattentive level.

As regard the MMN amplitude, there was no statistically significant difference among the three groups. This agreed with previous studies that attributed this variability to the dependence of MMN amplitude on the level of alertness of the subject (Lang et al., 1995 and Morr et al. 2002).

The results of the present study demonstrated that SNHL had a significant impact on the timing of the brain processes involved in the detection and discrimination of stimuli. In AN patients, MMN provide useful information regarding higher-level (cortical) responsiveness to auditory stimuli. Further extensive studies of cortical evoked potentials are recommended in AN patients, specially MMN using speech stimuli in order to provide insight into the early and later cognitive processes that underlie the detection and discrimination of speech.

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الملخص العربي

موجة عدم التوافق السالبة في مرض إعتلال العصب السمعي

ملخص البحث:

تمثل موجة عدم التوافق السالبة وسيلة إكلينيكية وموضوعية لتقييم التمييز السمعي.

يتميز مرضى إعتلال العصب السمعي بوجود ضعف سمعي للنغمات النقية مع اختفاء الاستجابة السمعية المثارة لجذع المخ فيما يظل الإنباعات الصوتية للأذن الداخلية في حالتها الطبيعية.

صمم هذا البحث لدراسة موجة عدم التوافق السالبة في 10 شخص قسموا إلى ثلاث مجموعات: المجموعة الأولى: تشتمل على 20 مريض من مرضى إعتلال العصب السمعي. المجموعة الثانية: 20 مريض من مرضى ضعف السمع الحسي (القوقعي) والمجموعة الثالثة: من 20 شخص من ذوي السمع الطبيعي.

ولقد تم فحص كل هؤلاء الأشخاص على النحو التالي:

تاريخ مرضى شامل، فحص الأذن بالنظرة، تقسيم السمع الأساسي، الإنباعات الصوتية للأذن الداخلي، الاستجابة السمعية المثارة لجذع المخ واختبار تسجيل موجة عدم التوافق السالبة.

وأظهرت نتائج هذا البحث أن ضعف السمع الحسي / عصبي له تأثير بالغ على توقيت التفعيل الدماغي المسؤول عن التقسيم والتمييز للمؤثرات. بالإضافة لهذا لم يتلاحظ وجود إختلاف جوهري بين مرضى إعتلال العصب السمعي ومرضى ضعف السمع القوي من حيث زمن حدوث موجة عدم التوافق السالبة.