Chronic Conductive Hearing Loss in Adults

Effects on the Auditory Brainstem Response and Masking-Level Difference

Michael O. Ferguson, MD; Raymond D. Cook, MD; Joseph W. Hall III, PhD; John H. Grose, PhD; Harold C. Pillsbury III, MD

Objective: To determine whether chronic conductive hearing loss in adults results in changes in the auditory brainstem response (ABR) similar to those observed in children with histories of otitis media with effusion.

Design: Test of effect of unilateral conductive hearing loss on adult ABR using age-matched control group and subjects as their own controls.

Subjects: Twelve adults with a history of unilateral conductive ear disease. An age-matched control group of 21 adults was also tested.

Methods: The ABR, an electrophysiologic test of auditory brainstem functioning, was used to evaluate possible brainstem abnormalities in the impaired listeners. In addition, the masking-level difference, a behavioral test of binaural auditory processing in the brainstem, was used.

Results: When comparing the patients’ diseased ears with their healthy ears, significant delays were seen for wave V as well as for the I-V and III-V interwave intervals. For comparison with the control population, significant prolongations were again seen for wave V and for the III-V interwave intervals. In addition, reduced masking-level differences and significant correlations between the masking-level differences and the ABRs, independent of hearing threshold, were noted.

Conclusions: The results suggest that chronic conductive impairment in adults leads to changes in the ABR similar to those observed in children with histories of otitis media with effusion. As such, these changes do not appear to be related to a critical period of development.


Several investigations1-7 have demonstrated that the auditory brainstem response (ABR) often shows abnormalities in children having a history of otitis media with effusion (OME) and associated hearing loss. The hypothesis underlying these investigations is that fluctuation of hearing levels (HLs) during development may result in changes in auditory neural structure and function, particularly if there is a critical period during maturation in which the central neurophysiologic condition is relatively labile.3

A common finding in these ABR studies of the juvenile population is an increase in the interwave intervals compared with a control population, despite resolution of effusion and the presence of normal audiometric thresholds at the time of testing. Although findings of altered brainstem electrophysiologic features have been universal among these studies, the specific nature of the interwave changes reported have been somewhat inconsistent. Whereas Anteby et al1 found abnormally long wave III-V and wave I-V latencies for children with a history of OME, Folsom et al3 noted significant increases in the I-III and III-V interwave intervals, with significant increases in the absolute latencies of waves III and V. Similarly, Gunnarson and Finitzo5 found significant delays in the absolute latencies of waves III and V as well as in the I-III and I-V interwave intervals when comparing controls with children with OME. In addition, they noted an abnormality in the binaural interaction response in children with OME. An investigation by Hall and Grose6 also found increases in the I-III and I-V interwave intervals and significant delays in the absolute latencies of waves III and V. Chambers2 noted prolongations in the I-III interwave interval, but no increase in either the III-V or I-V intervals. A general synopsis of these studies is that early conductive impairment results in significant increases in the absolute latencies of wave III or V (or both) and 1 or more of the interwave intervals.
SUBJECTS AND METHODS

SUBJECTS

Control Group

The control group consisted of 21 adults (10 women and 11 men) ranging in age from 24 to 50 years (mean age, 31.4 years). Use of the adults as subjects was approved by the human subject institutional review board. Adults older than 51 years were excluded from the study because of the possible changes in the ABR secondary to the degenerative effects of age on the auditory system.21 Control subjects had no history of hearing impairment, ear trauma, or ear surgery. Inclusion in the control group required that an audiogram with normal results be obtained at the time of ABR data collection.

Experimental Group

This group was composed of 12 adults (10 women and 2 men) ranging in age from 19 to 49 years (mean age, 37.3 years). The subjects were drawn either from a list of patients with conductive hearing impairment scheduled to undergo correlative middle ear surgery or from conductively impaired patients seen for a routine visit in the outpatient clinic. The cause of impairment was varied, including otosclerosis, cholesteatoma, tympanic membrane perforation, and chronic infection. Inclusion in the experimental group required a unilateral chronic conductive hearing loss between 25 and 55 dB HL at 2000 Hz and 4000 Hz, confirmed by repeated audiograms at the time of the study. Individual patient audiograms (air conduction) are shown in Table 1. Bone conduction thresholds were always 15 dB HL or better. Average length of time for hearing impairment was about 14 years (range, 2-25 years). None of the patients acquired the hearing loss prior to 12 years of age.

ABR STIMULI AND PROCEDURE

Audiological testing took place in a single-walled sound suite. Otoscopic examination was performed by 1 of 2 investigators (M.O.F. and R.D.C.). The ABR evaluation was conducted in a quiet examination room after the procedures were fully explained and signed consents were obtained. Subjects were awake and encouraged to relax with their eyes closed. They were tested in the prone position in a comfortable reclining chair. The ABR evaluation was performed using a Nicolet Spirit evoked potential system (Nicolet, Madison, Wis). Electroencephalographic activity was recorded for each ear by a midline forehead (non-inverting) electrode (Cz) and an ipsilateral ear canal (inverting) electrode (A1 or A2), with a ground electrode placed 1 to 2 cm above the nasion. Nicolet gold foil TiPtrodes were used in the ear canal since it was determined that they optimized the recording of wave I amplitudes, otherwise silver electroencephalographic electrodes were used.22 Inter-electrode impedance was maintained at 5000 Ω or less.

Click stimuli were produced via 100-microsecond rectangular electrical pulses transduced through tube phones (ER-3A tube phones, Eymotic Research, Elk Grove Village, Ill). Insert earphones were deeply inserted to obtain maximal interaural attenuation.5,25-27 Clicks had peak energy at 3000 Hz. Each ear in the control group was stimulated at click intensity levels of 60, 70, and 90 dB nHL. The diseased ears in the patient group were additionally stimulated at 100 dB nHL, and the healthy ears in this group were

Animal research has provided additional support for an association between attenuated auditory input and abnormalities in the auditory brainstem development. Experimentally induced conductive hearing impairments during critical periods are known to produce neural alterations central to the cochlea. Specific studies have demonstrated abnormalities in the development of binaural neural elements in the auditory brainstem, especially in cases of unilaterally induced conductive hearing loss.8,11

In addition to the use of the ABR as a means of evaluating auditory brainstem function, behavioral evidence of abnormalities in the brainstem auditory processing can be obtained through the use of the masking-level difference (MLD).15 The MLD is a psychoacoustic test that measures the sensitivity of the auditory system to interaural differences of time and amplitude. In the basic configuration, the masking noise is presented in phase to the 2 ears (No). The signal is presented either in phase to the 2 ears (So) or π radians out of phase at the 2 ears (Sπr). The MLD is the difference in the levels of the signal at masked threshold in these 2 configurations. It is assumed that the MLD is primarily dependent on auditory brainstem neurons receiving binaural input. Thus, the MLD too has been used as a tool for analyzing the effects of early conductive hearing loss on the auditory brainstem in children with recurrent OME.6,13-15 These studies have shown that the MLD is typically reduced when OME is present, remains significantly decreased even after the placement of tympanostomy tubes and the subsequent return of normal bilateral pure tone audiometric thresholds in quiet (ie, no noise present), but often returns to normal 1 to 2 years following restoration of normal hearing thresholds.16 Further study6 examining both MLDs and ABRs in children having a history of OME with hearing loss showed both reduced MLDs and abnormalities in the ABRs. In addition, the study showed a significant correlation between the decreased MLDs and the degree of ABR waveform asymmetry. Although the MLD is based on low-frequency stimulation and the ABR is based primarily on high-frequency stimulation, both rely critically on brainstem function, and previous studies17,18 of listeners with presumed brainstem pathologic features have shown a significant relation between ABR and MLD results.

Interestingly, studies19-22 of adults with acquired conductive hearing impairment have also indicated reduced MLDs. Research20,22 has shown that reduced MLDs persist even after the postsurgical restoration of normal audiometric thresholds, but often recover over a 1- to 2-year period. This pattern of results is similar to that found by Hall et al19 for children with a history of OME. The
stimulated at the intensity level necessary to compensate for the hearing loss in the impaired ear. The intensity level at which the healthy ear was stimulated was determined by first calculating the interaural difference of the hearing thresholds of the healthy and diseased ears at 2000 Hz and 4000 Hz. This interaural difference was then subtracted from 100 dB nHL (the stimulus intensity for the diseased ear) to determine the level of stimulation for the healthy ear. This ensured that the stimulus level reaching the cochlea of each ear was matched for intensity. Clicks were presented at a rate of 15.1 per second.

The electroencephalographic response was amplified and bandpass filtered between 100 Hz and 3000 Hz, then sent to a signal averager set to scan a 10-millisecond epoch. The final averaged response, the result of 1500 stimulus presentations, was graphically displayed on the system’s video monitor.

For each subject, peak latencies of waves I, III, and V were measured and from these values interwave interval values between waves I and III, waves III and V, and waves I and V were calculated. The ABR tracings were judged independently by the 2 investigators (M.O.F. and R.D.C.). Differences in measurements between investigators were reevaluated. For each group of subjects the average values of peak latencies were computed, and measurements were compared both between groups and interaurally within each subject.

STIMULI FOR MLD

The masking noise for the MLD test was a 300-Hz wide-band arithmetically centered on 500 Hz and presented in phase to the 2 ears (No). The digitally generated 400-millisecond 500-Hz pure tone signal had 50-millisecond sinusoidal rise-fall times and was presented either interaurally in phase (So) or 180° out of phase (Sπ) to the 2 ears. All stimuli were presented binaurally using earphones (MDR V6 earphones, Sony Electronics, Tokyo, Japan) mounted in circumaural cushions. Stimulus timing and response collection were controlled by a microcomputer (Gateway microcomputer, Gateway 2000, North Sioux, SD). The MLD was determined by subtracting the masked threshold level of the interaurally phase-shifted signal condition (NoSπ) from the masked threshold level of the interaurally in phase signal condition (NoSo). The 300-Hz wide masker was presented continuously at a pressure spectrum level of 60 dB.

PROCEDURE FOR MLD

Data were collected using a 3-alternative, forced choice, 3-down, 1-up adaptive strategy that estimated the 79.4% detection threshold. There were 3 observation intervals with the signal presented in only 1 interval at random. With 3 successive correct responses, the signal level was reduced; after 1 incorrect response, the signal level was increased. The threshold run was stopped after 12 reversals in the direction of signal attenuation, and the average of the last 8 reversals was taken as the threshold for the run. An initial step size of 8 dB was reduced to 4 dB after the first 2 reversals and reduced to 2 dB after the second 2 reversals. The subject was provided with visual feedback after each response. At least 2 thresholds were obtained and averaged for each signal condition, with a third condition added and included in the average if the range of the first 2 thresholds was greater than 3 dB. Ten of the 12 patients underwent MLD testing. Normative data were drawn from a previous study in our laboratory of 14 listeners with normal hearing who were tested under identical conditions.

RESULTS

AUDITORY BRAINSTEM RESPONSE

Control Group

Preliminary analyses of the ABR tracings, including the absolute wave latencies of waves I, III, and V as well as interwave intervals I-V, I-III, and III-V, showed no significant interaural differences between the left and right ear for the control group. Therefore, data from both ears of an individual control listener were averaged. The mean ABR absolute wave and interwave latencies at the stimulus levels of 60, 70, and 90 dB nHL are shown in Table 2. As the click stimulus increased in intensity, the mean latencies for waves I, III, and V decreased, demonstrating the expected effect of stimulus intensity on ABR wave latency. The I-III interwave interval increased proportionally to stimulus intensity, while the III-V and the I-V
interwave intervals were inversely related to intensity. The variation in the I-V interwave interval across stimulus level is shown in Figure 1. The graph shows a monotonic relationship between interwave interval and stimulus intensity, demonstrating a need to compensate for HL discrepancies when comparing healthy ears with conductively impaired ears.

### Patient Group

**Figure 2** shows an example of ABRs recorded in the healthy and diseased ears of a subject from the patient group. The subject’s tracings were selected to reflect typical waveforms and latencies for the patient group. The mean ABR wave latencies and interwave latencies for both the diseased ears and the healthy ears are shown in Table 2. Diseased ear mean wave latencies were derived from ABR recordings at a stimulus level of 100 dB nHL. Mean absolute wave and interwave latencies for healthy ears were calculated from ABR recordings at stimulus levels that were adjusted to compensate for hearing loss in the impaired ear on an individual basis. Thus, the stimuli reaching the cochlea of both diseased and healthy ears were matched with respect to intensity levels.

#### Comparison of Diseased Ears With Healthy Ears in Patients

A 2-factor repeated measures analysis of variance was performed on the absolute wave latencies (I, III, and V) of the healthy and diseased ears to determine if any significant differences existed. This analysis showed no effect of ear (F[1,11] = 1.56; P = .24) and the expected main effect of wave latency (F[2,22] = 8948.44; P < .001). The interaction between ear and wave latency was also significant (F[2,22] = 11.75; P = .001) and therefore, simple main effects were assessed.29 The results revealed no significant differences between the 2 ears for wave I latency or wave III latency, but the wave V latency for the diseased ear was prolonged in comparison with the healthy ear (F[1,11] = 37.65; P < .001). Mean absolute wave latencies (I, III, and V) of the diseased ears relative to the mean latencies of the healthy ears are shown in the first half of Figure 3.

These results suggest that differences in the derived interwave latencies should also emerge between ears. A 2-factor repeated measures analysis of variance applied to the interwave latencies (I-V, I-III, and III-V) for the 2 ears showed a significant difference between the ears (F[1,11] = 27.39; P < .001), demonstrating an increase in the overall interwave latencies in the diseased ears. As expected, there was also a significant main effect of interwave latencies (F[2,22] = 3337.96; P < .001). However, the interaction between ear and interwave latency was not significant (F[2,22] = 2.83; P = .08). Preplanned analyses of the individual interwave latencies were performed using independent t tests.29 Ear differences were significant for both the I-V and the III-V interwave intervals (T11 = −5.29, P < .05; T11 = −0.50, P < .05, respectively), but not significant for the wave I-III interval (T11 = −1.27; P = .23). Interwave intervals (I-V, I-III, and III-V) relative to the mean latency of the patients’ healthy ears are shown in the second half of Figure 3.

### Table 1. Summary of Patient Audiogram Data

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Ear</th>
<th>Frequency, Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>250</td>
</tr>
<tr>
<td>1</td>
<td>Diseased</td>
<td>40 25 25 30 25 35</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>−10 15 10 0 10 20</td>
</tr>
<tr>
<td>2</td>
<td>Diseased</td>
<td>−5 35 20 20 30 25</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>−10 −10 0 −5 5 5</td>
</tr>
<tr>
<td>3</td>
<td>Diseased</td>
<td>65 50 40 45 50 60</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>10 10 10 −5 10 5</td>
</tr>
<tr>
<td>4</td>
<td>Diseased</td>
<td>55 50 40 45 40 35</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>15 10 10 5 15 15</td>
</tr>
<tr>
<td>5</td>
<td>Diseased</td>
<td>25 25 30 30 30 45</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>30 35 15 10 5 20</td>
</tr>
<tr>
<td>6</td>
<td>Diseased</td>
<td>35 30 25 25 40 75</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>15 5 10 10 15 25</td>
</tr>
<tr>
<td>7</td>
<td>Diseased</td>
<td>20 20 30 25 45 65</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>10 10 10 5 20 20</td>
</tr>
<tr>
<td>8</td>
<td>Diseased</td>
<td>35 30 30 45 45 55</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>5 10 5 5 5 5 20</td>
</tr>
<tr>
<td>9</td>
<td>Diseased</td>
<td>20 25 35 40 40 70</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>−10 −10 0 −10 5 10</td>
</tr>
<tr>
<td>10</td>
<td>Diseased</td>
<td>45 30 25 20 35 50</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>15 5 10 5 0 15</td>
</tr>
<tr>
<td>11</td>
<td>Diseased</td>
<td>65 50 45 40 25 35</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>30 30 25 10 10 20</td>
</tr>
<tr>
<td>12</td>
<td>Diseased</td>
<td>65 50 50 35 40 60</td>
</tr>
<tr>
<td></td>
<td>Healthy</td>
<td>20 20 10 5 10 25</td>
</tr>
</tbody>
</table>

### Table 2. Auditory Brainstem Response Absolute Wave and Interwave Latencies for Patient and Control Groups

<table>
<thead>
<tr>
<th>Absolute Wave and Interwave Latencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
</tr>
<tr>
<td>Patients</td>
</tr>
<tr>
<td>Diseased</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Controls</td>
</tr>
<tr>
<td>At 60 dB nHL</td>
</tr>
<tr>
<td>At 70 dB nHL</td>
</tr>
<tr>
<td>At 90 dB nHL</td>
</tr>
</tbody>
</table>

*All values are mean (SD) milliseconds. Results for the diseased ear were obtained at a stimulus level of 100 dB normal hearing level (nHL). Normal ear measurements were intensity matched to compensate for interaural hearing disparity.*

**ARCH OTOLARYNGOL HEAD NECK SURG/ VOL 124, JUNE 1998**

©1998 American Medical Association. All rights reserved.
Comparison of Diseased Ears With Control Group

As with the interaural analyses above, comparing the ABR data of the diseased ears of the patient group with the ABR data from the control group requires that the 2 groups be matched by intensity to compensate for the conductive hearing impairment in the patient group. The average pure tone threshold at 2000 and 4000 Hz in the impaired ear of the patient group was approximately 35 dB HL. The average pure tone threshold for the control group was approximately 5 dB HL. Thus, using the mean control data at 70-dB click stimulus to compare with the mean patient data at 100 dB will on average adjust for the disparity in hearing sensitivity between groups.

A 2-factor analysis of variance (one within, one between) performed on the absolute wave latencies of the 2 groups indicated a significant main effect of group (F[1,31] = 4.69; P = .04) and, as expected, a significant main effect of absolute wave (F[2,62] = 11.140; P = .001). No significant main effect was found for the interaction between group and wave (F[2,62] = 2.94; P = .06). Preplanned independent t tests were again performed to determine group differences at each independent wave. As with the results of the patients’ diseased vs healthy ears, the latency of the absolute wave V in the patient group was significantly prolonged compared with the control population (T31 = −2.46; P = .02). Again, consistent with the previous analysis comparing the healthy vs the diseased ear of the experimental group, no significant differences were found for wave I or III (T31 = −1.61, P = .12; T31 = −1.63, P = .11). Absolute wave latencies relative to the mean latencies of the control group are shown in the first half of Figure 4.

The analysis of variance on the interwave latencies showed no significant main effect of group (F[1,31] = 3.78; P = .06), an expected significant main effect of interwave latencies (F[1,31] = 3663.02; P = .001), and a non-significant interaction between group and interwave latencies (F[2,62] = 2.78; P = .07). Preplanned independent t tests indicated results that are fairly consistent with that seen in the interaural analyses. No group difference was
noted for the I-III interwave latency (T31 = −0.078; P = .86), while group differences were significant for the III-V interwave latency (T31 = −2.32; P = .03), again reflecting the longer intervals of the patient group. The only statistical result that differed from the previous analysis comparing the healthy vs the diseased ear of the experimental group was that for the I-V interwave latency. The present analysis found the group difference of this interwave latency marginally insignificant (T31 = −1.93; P = .06). The interwave intervals relative to the control group are shown in the second half of Figure 4.

MASKING-LEVEL DIFFERENCE

As previously mentioned, the MLD is derived by subtracting the NoS\textsubscript{π} threshold from the NoSo threshold. Although the NoS\textsubscript{π} and the NoSo thresholds are included in the results, attention is focused primarily on the MLD as the main measure of binaural processing. Binaural cues influence the NoS\textsubscript{π} detection thresholds, and thus binaural hearing is measured by this threshold, but this threshold is also influenced by the general processing efficiency of the listener.\textsuperscript{30} This general efficiency factor is theoretically canceled out by the subtraction of the NoS\textsubscript{π} threshold from the NoSo threshold, since both thresholds are affected by processing efficiency. The thresholds for NoSo, NoS\textsubscript{π}, and the derived MLDs for individual impaired listeners are shown in Table 3. Also shown are control data from a group of adults with healthy hearing obtained from a previous study\textsuperscript{21} using identical stimulus parameters. The most notable distinction between the patient results and the control data is that both the patient NoS\textsubscript{π} thresholds and the MLDs were consistently poorer than those of the control group, while the NoSo thresholds were similar to the control results. Eight of 10 impaired listeners were outside of the 95th percentiles (estimated using ± 2 SDs) of the listeners with healthy hearing for the MLD and NoS\textsubscript{π} threshold. For the NoSo threshold, 9 of 10 patients were within the 95% confidence interval. Thus, the abnormality in the MLD measure was caused primarily by an abnormally high NoS\textsubscript{π} threshold.

RELATION BETWEEN MLD AND ABR

Because both the MLD and the ABR are related to auditory brainstem processing, the next step in the analysis was to examine possible relations between the reduced MLDs and the abnormality in the ABR latencies of the patient group. The most obvious correlation to investigate was that between the MLDs and the absolute wave latencies and interwave latencies of the impaired listeners. Because of the influence of HL on both the MLD and the ABR, it is possible that any relationship between the MLD and the ABR would be attributable simply to the association of each of those variables to HL. Therefore, MLD and ABR correlations were determined using a partial correlation analysis, with the effects of HL being controlled statistically (pure-tone average >500, 1000, 2000, and 4000 Hz). No significant correlations were noted between the MLD and the absolute wave latencies (wave I, r = 0.35; wave III, r = −0.03; and wave V, r = −0.08). With regard to interwave intervals, the I-V and the I-III interwave intervals correlated significantly (P < .05) with the MLD (r = −0.63 and r = −0.78, respectively), while the III-V interwave interval was not significantly correlated (r = −0.11).

| Table 3. Summary of Threshold and Masking-Level Difference (MLD) Data* |
|-----------------|-----------------|-----------------|
| Healthy group, mean (SD) | 77.9 (1.3) | 63.1 (1.6) | 14.8 (1.4) |
| Individual patients, mean | 77.4 (1.4) | 68.8 (2.4) | 09.6 (2.9) |

*Normative data from previous study evaluating adult masking-level difference. Thresholds are reported in decibel sound pressure level. See the introductory section for explanation of NoSo and NoS\textsubscript{π}.
†Data outside 95th percentile of normal listener.

COMMENT

This study was undertaken to determine whether chronic conductive hearing loss in adults results in similar ABR measurement abnormalities as seen in the juvenile population. If so, it could provide some evidence that a degree of lability or neuronal adjustment unrelated to any critical developmental period might exist in the mature auditory system. As described earlier, several studies investigating children with histories of OME found abnormalities in their ABRs that were not seen in control populations. Despite some individual variation among studies, it was generally noted that wave III or V (or both) were delayed, and several interwave intervals were prolonged. Our results obtained in adult listeners are in general agreement with most published reports on children with histories of OME. When comparing the patients’ diseased ears with their healthy ears, significant delays were seen for wave V as well as for the I-V and III-V interwave intervals. For comparison with the control population, significant delays were again seen for wave V and for the III-V interwave interval, whereas the difference for the I-V interwave interval did not attain significance. Overall, our results are most consistent with the study of Anteby et al,\textsuperscript{1} who found significantly prolonged interwave latencies between waves III and V and between waves I and V. Our results support an interpretation that conductive hearing loss in adults leads to abnormalities in their ABRs beyond those attributable to simply loss of hearing sensitivity.

A unique feature of this study is that the diseased ears in the patient group could be compared, in essence, with 2 different “control” groups. The similar findings between the 2 control comparisons suggest a relatively
robust effect. Perhaps the more powerful, or informative, of the 2 controls is the interaural comparison within each patient. With this interear evaluation, it was possible to match the intensity level reaching the cochlea of the diseased ear precisely with the intensity level of its control. Although the comparison with the control group of subjects with normal hearing was also intensity matched, it was done so on a group average basis, and thus exact intensity matching for each patient was not obtained. The interaural comparison represents a tighter control because any effect caused by a variable other than hearing impairment is essentially canceled out. This feature may explain why the results of the 2 control comparisons were not identical. In addition, individualized intensity matching of the interear comparison more closely reflects the equal hearing threshold between controls and patients that was present in the juvenile investigations. This may account for the fact that the interaural results agree more closely with the findings of the childhood OME studies.

In speculating on the cause of the abnormalities in the ABRs seen in the conductively impaired ears, it is important to consider all possible sites along the auditory pathway. One obvious candidate is simply the acoustic attenuation resulting from the middle ear disease. Whereas individual ABR wave latencies generally increase in cases of conductive hearing loss, it is believed that this effect is simply related to the decreased level of stimulation reaching the cochlea. Furthermore, it is believed that interwave intervals are not influenced by conductive hearing loss when the sensation level of the stimulus is controlled. As mentioned previously, the conductive hearing loss in this study was controlled by adjusting the level of stimulation to each ear. The acoustic attenuation resulting from conductive hearing loss would therefore not appear to be a strong candidate to account for the abnormalities in ABR results found in the hearing-impaired listeners.

Possible effects of middle ear disease on cochlear function have also been suggested as a source of the observed increases in absolute wave latencies and interwave intervals. However, the literature is unclear regarding the effect of cochlear hearing loss on I-V interwave intervals. Although some reports suggest that cochlear loss is associated with an increase in the I-V interval, other reports suggest either no effect or even a decrease in the I-V interwave interval. However, the audiometric data obtained in our study suggest little evidence of cochlear hearing loss. Therefore, cochlear dysfunction is not considered to be a likely contributor to the results obtained in this study.

In keeping with the above discussion, the finding of wave V abnormalities in the presence of normal wave I characteristics suggests that the basis of the abnormality lies in the auditory brainstem and not in the auditory periphery. This divergence of wave latency in the progression from wave I-V in the ABR may be caused by a variety of reasons. The effect could be due to a decrease in the overall number of active neurons, a desynchrony in neuronal discharge, or it could represent a degree of lability in the neuronal connectivity of the auditory brainstem. Although our study does not allow a differentiation between these possibilities, it suggests that abnormalities in the ABRs in the presence of long-term conductive hearing loss should not be summarily attributed to a critical period of development in the immature system.

As with the ABR data, the MLD results are in general agreement with prior studies demonstrating a reduced MLD in listeners with a conductive hearing loss. The reduced MLDs demonstrated in our patients are likely to be due to not only poor use of binaural cues but also to the elevated hearing thresholds and threshold asymmetry. Hall et al demonstrated that MLDs will often improve considerably when tested under conditions of equal sensation level, indicating that the smaller MLDs measured with equal sound pressure level presentation are attributable to primarily the elevated thresholds in quiet rather than to diminished binaural processing. However, the same study found that even under conditions of equal sensation level testing, the resulting MLDs were often still reduced. In addition, a study by Hall and Grose demonstrated a persistence of reduced MLDs for up to 2 years following surgical correction that resulted in normal audiometric thresholds. Thus, the present MLD results are of greatest interest when hearing threshold is statistically controlled, as was done in the partial correlation between the MLD and ABR. The significant correlations obtained imply an association that is independent of hearing threshold. While the positive correlation of the MLDs to the I-V interwave intervals fits well with the significant differences seen interaurally for that particular metric, the positive correlation between the MLDs and the I-III interwave intervals is not as compatible with the current ABR results.

Overall, this study suggests that chronic conductive hearing impairment in adults leads to changes in the ABR unrelated to a critical period but similar to those observed in children with histories of OME. This similarity warrants the continued investigation of adult patients to determine whether the ABR and MLD patterns again change subsequent to the return of normal audiometric thresholds following corrective middle ear surgery.

Accepted for publication February 2, 1998.

Reprints: Michael O. Ferguson, MD, Division of Otolaryngology–Head and Neck Surgery, CB No. 7070, Burnett-Womack, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7070 (e-mail: fergusom@med .unc.edu).

REFERENCES


