Brainstem auditory evoked response
II. Clinical applications in the assessment of patients with organic hearing loss.

The Annals of Otology, Rhinology & Laryngology,

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This paper studies the effect of otologic disease on the brain stem auditory evoked response (BAER). Both conductive and neurosensory hearing losses are analyzed by plotting wave V latencies and amplitudes as a function of sound intensity. It was found that the BAER is elicited primarily by frequencies greater than 2000 Hz. Conductive hearing losses produce a latency intensity function which approaches the norm with high decibel stimulation. Neurosensory losses produce a variety of latency intensity functions. In determining the degree of hearing loss, wave threshold is found to be the best index. Wave latency at high decibels is found to have little correspondence to degree of neurosensory loss; wave amplitude is highly variable among subjects but still a useful indicator for detecting pathology. Between-ear comparisons of wave latencies elicited by high decibel sound stimulation suggest that unilateral nonrecruiting or partially recruiting hearing losses will result in a latency difference. This method can be used to detect unilateral acoustic neuromas, and the false positives found by this technique are probably caused by other unilateral nonrecruiting hearing losses. In evaluating neurological disease, and especially when testing for a second occult lesion in multiple sclerosis (MS) an audiogram should be obtained because the criterion of a normal wave latency with decreased amplitude for the diagnosis of MS can be mimicked by peripheral hearing loss.

INTRODUCTION

Most experimental papers dealing with brain stem auditory evoked response (BAER) have focused on the ways in which central neurological diseases alter the latencies and interpeak differences of the component waves contained in the BAER. The first five waves are produced by various levels of the auditory pathway. Waves I through V are produced at the level of the acoustic nerve, cochlear nucleus, superior olive, nuclei of the lateral lemniscus (although this is disputed), and inferior colliculus respectively.1,2

Wave V is the strongest wave and many types of lesions both central and peripheral will prolong its latency and reduce its amplitude. The time interval between wave I and wave V is commonly used to separate central from peripheral diseases. Cochlear lesions were found to shorten the I-V interval; retrocochlear lesions have been found to prolong it.3,4 Brain stem lesions associated with a prolonged I-V interval can be produced by vascular insufficiency, mass effects, and demyelinating diseases.2,5,7 Mass effects can be produced by a shift of the brain stem,5 herniation,2 or invasive tumors.2,5,7 Acoustic neuromas prolong wave V's latency by more than 0.4 msec when the latency of the affected ear is compared to that of the opposite ear.8 Using this criterion, only 3% of acoustic neuromas are not diagnosed. However, there is a 12% false positive rate.8 Wave I is usually absent but can be normal even in the presence of an abnormal wave V.8 Multiple sclerosis (MS), a demyelinating disease, affects only the CNS and wave I thus remains normal.8,9,10 Of 30 MS patients studied by Robinson,9 22 had abnormal BAER responses, 16 had decreased wave V amplitudes, 10 had increased wave V latencies, and 4 had both.

The BAER cannot distinguish the type of lesion, only the location. For example, if waves I through III are normal and waves IV and V are not present, then the lesion must be located above the superior olive. In clinical usage, this technique is limited to the detection of occult lesions such as acoustic neuromas,8 the finding of a second
Fig. 1. Tone specificity of the BAER elicited by a click. Normal mean latency of 15 control subjects is represented by the center solid line; ± 2 SD are shown by the two outer solid lines. (Audiograms show air conduction with the vertical axis representing decibels plotted by ISO standards.)

Fig 2. BAER in subjects with severe hearing loss. Normal mean latency (a) and amplitude (b) for the 15 control subjects are shown by solid lines. The center line represents the mean and the two outer lines are ± 2 SD. (Audiograms show air conduction with the vertical axis representing decibels plotted by ISO standards.)
occult lesion for the diagnosis of MS, the establishment of brain death, or the evaluation of hearing. It makes little sense to study and bill a patient who has a known brain stem lesion when the results of the study will not affect patient management.

The auditory evoked response to clicks can also be used to evaluate both functional and organic hearing losses. Both neurosensory and conductive hearing losses can prolong wave V latencies. This paper deals only with organic losses and the parameters of the BAER which should be measured. Also, data regarding the frequency specificity of the click and the significance of a flat BAER are presented.

METHODS AND MATERIALS

This study employed the same equipment described on page 3. A total of 33 subjects (37 ears) were tested. Twenty-four subjects had a neurosensory hearing loss, eight had primarily a conductive loss, and one had a mixed loss. One subject with a conductive hearing loss was tested the day of and six weeks after stapedectomy. No subject had a history of neurological disease and each had an otologic examination prior to the test.

The subjects were tested with 0.1 msec clicks presented at a rate of 31/sec at varying sound intensities (HL). 2000 click presentations were summed per trial. If a subject produced a low amplitude BAER, a maximum in
Sound Intensity (decibels)

Fig. 4. BAER in subjects with neurosensory hearing losses involving both high and low frequencies. Normal mean latency of 15 control subjects is represented by the center solid line; ± 2 SD are shown by the two outer solid lines. (Audiograms show air conduction with the vertical axis representing decibels plotted by ISO standards.)

tensity of 95 dB was used. Wave V's latency, amplitude and threshold were recorded. Both ears of a subject were tested with 80 dB HL clicks at a rate of 31/sec for comparison of interear latencies. All recordings were repeated and averaged for reliability. The auditory threshold to the click was determined and compared to the BAER threshold; the clicks used for stimulating the BAER had an average auditory (subjective) threshold in normal subjects of 8.33 dB. In all subjects the opposite ear was masked with 40 dB white noise.

All subjects were tested without sedation when seated in a reclining chair to reduce neck muscle artifact. Only one subject had to be retested because of artifact. Four children between the ages of seven and eight were tested. They tolerated the procedure well and produced very little muscular artifact. In two subjects tested early in this experiment, auditory thresholds and contralateral BAERs were not obtained. All subjects had a prestudy audiogram, and masking was used if an interear difference greater than 20 dB was present. All audiograms presented in this paper are plotted by ISO criteria. Normative data were obtained from 15 control subjects described by Kavanagh and Beardsley.13

RESULTS

For each of the subjects tested, wave V's latency as a function of stimulus intensity was plotted to form a latency intensity function (Figs. 1-4, 6 and 7).

Figure 1 illustrates that the BAER to a click is elicited mainly by high
frequencies above 2000 Hz. Severe hearing losses are illustrated in Figure 2; two of the five subjects shown had no BAER but still had residual hearing. Figure 3 shows eight subjects with various degrees of high-frequency hearing loss and demonstrates that latency at high decibels is a very poor predictor of auditory function. Threshold appears to be the best predictor.

If a subject with abnormal hearing is tested with high decibel stimuli, a wave V with a normal latency but abnormal amplitude may be elicited. A reduction in amplitude along with an increased BAER threshold may be the only indications of a hearing abnormality (Fig. 5).

Various latency-intensity functions for patients with neurosensory and conductive hearing losses are shown in Figures 4, 6, and 7. Comparisons of these figures show that both conductive and neurosensory hearing losses can produce similar curves.

A predicted latency intensity function for a 20 dB conductive hearing loss is shown in Figure 6. At high decibel levels, latency approaches the norm; abnormalities are most marked a threshold. One subject (RA-L) who had a conductive hearing loss underwent stapedectomy. Figure 6 shows that improvement in this subject's audiogram was accompanied by a corresponding decrease in her BAER threshold and a shortening of wave V's latency.

None of the subjects tested had an auditory threshold above his or her BAER threshold (Table 1). Table 2 shows the difference in wave V latency between a subject's right and left ears when tested at 80 dB HL. Only 2 of the 19 subjects with a neurosensory loss had an interear difference greater than 0.40 msec; both of these patients had asymmetrical peripheral hearing losses.

DISCUSSION

Figure 1 plots wave V latency as a function of intensity level for three subjects with a neurosensory hearing loss. Subject CM-R had a bilateral familial low-frequency hearing loss and ES-R had a high-frequency hearing loss of equal severity. Only ES-R produced an abnormal latency-intensity curve.
Fig. 6. BAER in a patient with conductive hearing loss. Normal mean latency of 15 control subjects is represented by the center solid line; ± 2 SD are shown by the two outer solid lines. (Audiograms show air conduction with the vertical axis representing decibels plotted by ISO standards.)

comparing these two curves illustrates that the click-elicited BAER is primarily derived from the basilar cochlea. Davis and Hirsh, in experiments utilizing narrow band masking, have reached similar conclusions.14 Subject RG had an asymmetrical hearing loss more severe in his left (RG-L) than right (RG-R) ear. The RG-R line in Figure 1 demonstrates that losses between 2000 and 6000 Hz will produce an abnormal BAER. Audiograms of RG-L and RG-R were very similar below 4000 Hz, but above this level, RG-L had a more severe loss. The BAER for RG-L had a longer latency and higher threshold than the BAER for RG-R. Thus frequency losses above 4000 Hz will also produce an abnormal BAER response. Other experimenters have found that the BAER is maximally correlated with frequency band from 2 to 4 kHz15 and 4 to 8 kHz3,4.

The BAER is very sensitive to high frequency hearing losses, and minor losses can be detected.13 A normal latency-intensity function is very good evidence for normal hearing, especially because low-frequency hearing loss without a high-frequency component above 2000 Hz is rare.

If no BAER can be obtained from an individual, a question arises about whether the patient might be totally deaf. Figure 2 shows audiograms and BAER for five subjects with severe hearing loss. FK-R and FS-L had neurosensory hearing losses caused by an ototoxic drug and a Pantopaque®-proven acoustic neuroma, respectively. RS-L had a
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Fig. 7. Various BAER in patients with conductive hearing loss. Normal mean latency of 15 control subjects is represented by the center solid line; ± 2 SD are shown by the two outer solid lines.

mixed hearing loss from otosclerosis. DW-R had a pure conductive loss from congenital malformation of the middle ear, and MG-L had a neurosensory loss of undetermined etiology. Even though none of these ears was totally deaf, two (FK-R and FS-L) produced no BAER. Therefore a laboratory must be cautious in diagnosing deafness by BAER techniques because some residual hearing may still be present.

Data from eight subjects with varying degrees of high-frequency hearing loss are shown in Figure 3. In evaluating their BAER to determine hearing acuity, three measurements were made.

The first measurement is the threshold of the BAER. BAER threshold is defined by the authors as that decibel level at which the BAER is first discernable. In no studied subject with a significant high-frequency hearing loss was this parameter normal. It is unfortunate that BAER threshold values do not correspond closely with the degree of hearing loss present, as illustrated by GS-L (Fig. 3). This subject had the highest BAER threshold in Figure 3 but only a 35 dB loss. Two facts explain this. First, at threshold only wave V is detected and its amplitude is highly variable. Thus subjects with a normally low wave V amplitude will have a high BAER threshold. Second, at threshold wave V's amplitude is very small- and electrical and muscular artifact can mask a subject's response. Patients should therefore be relaxed and preferably asleep or sedated with chloral hydrate before testing. All subjects with an organic hearing loss had a BAER threshold below
their auditory threshold (Table 1). In functional hearing disorders, the reverse was often found.\(^{13}\)

Wave V latency is the second measurement of acoustic function. As illustrated by CH-L and BR-L in Figure 3, latency at high decibels has very little correlation with hearing loss. These subjects had a 50 to 60 dB high-frequency hearing loss, but at high decibel levels of stimulation had normal latencies. The authors believe that recruitment is responsible for this effect. Picton\(^{6}\) states that recruiting ears produce an “L” shaped latency-intensity function. The base of the “L” is produced by a very rapid increase in latency with a slight drop in sound intensity. Two subjects in Figure 3 (BR-L, CH-L), and one in Figure 4 (DS-L) have such a curve. Jerger\(^{15}\) also found that threshold is a better predictor of acoustic function than latency and noted that latency correlated with audiometric configuration.

At threshold, the latency of wave V is much more indicative of acoustic function. However, when elicited by low-level stimuli, the detection of wave V is markedly affected by artifact, and finding the exact BAER threshold could become quite time consuming.

Amplitude is the third measurement used in evaluating hearing. Because of its great standard deviation,\(^{13}\) it is the least reliable parameter. Nevertheless at high intensities when wave V latency is normal, the amplitude is often abnormal. Four subjects (DS-L, DV-L, CH-L, and BR-L) with neurosensory hearing loss show this pattern. Three of these four subjects had abnormal wave amplitudes above their threshold responses (Fig. 5). One subject, DS-L, had an audiogram which showed a significant hearing loss, but his only significant abnormality was an elevated BAER threshold of 50 dB and a reduced wave V amplitude at this level (Figs. 4 and 5). This subject demonstrates that the BAER threshold response must always be obtained when evaluating hearing.

Even when using all of these criteria, a BAER evaluation cannot reliably duplicate an audiogram because many combinations of auditory losses involving different frequencies can produce similar curves, and because a BAER elicited by clicks, mainly tests high-frequency hearing. Although normality can be determined with a high degree of accuracy, any loss present can only be described as mild, moderate, profound, or severe and only in reference to high-frequency hearing.

The latency-intensity functions for 10 subjects with various neurosensory hearing losses in both high and low frequencies are shown in Figure 4. Many of these curves, and those in Figure 3, are either parallel to or slowly approach the

### Table 1. Difference Between BAER Threshold and Subjective Hearing Threshold in Subjects with Periphera

<table>
<thead>
<tr>
<th>Change In Decibels</th>
<th>N euro</th>
<th>Conducive</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>40</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>35</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>30</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>25</td>
<td>1</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>20</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>15</td>
<td>4</td>
<td>2</td>
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<tr>
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<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>&lt;0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Total Ears\(^{a}\) 23 9 1

\(^{a}\)In 2 subjects the auditory (subjective) hearing threshold was not tested, and 2 subjects produced no BAER waves.

33 ears were tested in 219 subjects. Change in Decibels - BAER threshold minus auditory (subjective) hearing threshold.

### Table 2. Difference of Wave V Latency Between Right and Left Ears in Subjects with Periphera

<table>
<thead>
<tr>
<th>Latency Between Right and Left Ears, msec</th>
<th>Neurosensory</th>
<th>Conductive</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.00-0.09</td>
<td>8</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>0.00-0.19</td>
<td>5</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>0.20-0.29</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>0.30-0.39</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>0.40-0.59</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>0.60-0.69</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>&gt;0.70</td>
<td>0</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

\(^{a}\)Four subjects produced BAER in one ear only, and in two subjects contralateral testing was not performed.
norm. One might theorize that these subjects had nonrecruiting or partially recruiting ears in the frequencies eliciting the BAER. However, there is no reliable method of testing this hypothesis because a click stimulates a band of frequencies and the BAER may be composed of both recruiting and nonrecruiting frequencies. Also, the best objective means of evaluating recruitment in subjects with a bilateral symmetrical hearing loss is the Short Increment Sensitivity Index, and this method is usually not calibrated for frequencies above 4000 Hz.

Conductive hearing losses are equivalent to decreasing the sound intensity used in stimulation. In theory, a conductive hearing loss will result in a shifting of the latency-intensity function to the right, so that wave V's latency at threshold is unchanged. The amount of shift should correspond to the degree of hearing loss. A predicted latency-intensity function for a 20 dB loss is shown in Figure 6. This curve slowly approaches the norm, and at higher decibel levels, normal latencies are elicited. The preoperative audiogram of RA-L (Fig. 6) shows a typical conductive hearing loss; the loss is not equal in all frequencies and is maximal in the lower frequencies. (Preoperative and postoperative bone conduction were not significantly different.) The latency-intensity function shows a marked displacement to the right compatible with a severe hearing loss. The patient had a stapedectomy and six weeks after the operation, her audiogram showed complete closure of the air-bone gap with persistence of a neurosensory hearing loss. The patient's latency-intensity function also showed marked improvement with a shorter latency and a BAER threshold 40 dB lower (Fig. 6).

Latency-intensity functions for seven other subjects with conductive hearing losses are shown in Figure 7. The mildest loss was GB-R, who had only a 20 dB high-frequency loss from a resolving serous otitis media. The most severe losses were SD-R, who had a 40 dB loss in all frequencies from ossicular discontinuity; and DW-L, who had a 40 dB loss in all frequencies from a congenital deformity of his middle ear.

In all subjects, the displacement of the curve corresponds roughly to the degree of conductive hearing loss. However, the extent of displacement was not an exact indicator of the deficit. This imprecision is to be expected because conductive hearing losses are often maximal in the low frequencies (RA-L, Fig. 6) and because the click-elicited BAER measures high frequencies. The same degree of low-frequency hearing loss is often associated with different magnitudes of high-frequency losses, and one would expect different latency intensity functions to be produced.

Another clinical problem is distinguishing conductive from neurosensory or mixed losses. All of these conditions can produce similar curves (Figs. 3, 4, and 7). In an attempt to differentiate these losses, the authors' laboratory has experimented with the BAER elicited by a mastoid oscillator. The authors have found this technique to be useful but not ideal because, as already mentioned, clicks often do not stimulate the cochlea in the area of maximal conductive loss. The sound energy output of a mastoid oscillator is limited and the best-defined waves only correspond to those produced by 40-50 dB air stimuli. Berlin, using electrocochleography, found bone conduction to be plagued with problems of artifact and calibration. Also, results are difficult to interpret because the type of hearing loss above 4000 Hz, on a subject's audiogram, cannot be reliably determined with mastoid oscillators, which are not calibrated for these frequencies. Finally, bone conduction doubles the time needed to evaluate hearing and makes the test approximately two hours long.

The expense and length of this procedure is often not justified because most middle ear pathology in children can usually be detected by tympanometry or by a good otologic examination. A rare need for the use of bone conduction may lie in the evaluation of cochlear function in the presence of known middle ear disease, for example, in an infant with external ear canal atresia.

The BAER is also used in the detection of acoustic neuromas. Sellers states that unilateral acoustic neuromas will usually cause a BAER interaural latency difference of more than 0.4 msec (83 dB HL clicks). He found that his method
had a 12% false positive rate. Of our 19 subjects with a neurosensory hearing loss (Table 2), only 2 (10.5%) had an interear latency difference greater than 0.40 msec (80 dB HL clicks).

A possible explanation for these false positive results is the presence of a unilateral nonrecruiting hearing loss. An acoustic neuroma usually causes this type of hearing loss, but there are many other etiologies (ie, conductive hearing losses). For this reason, patients with a conductive hearing loss cannot be evaluated for acoustic neuromas using this criterion. Also, the authors feel that patients with severe unilateral high frequency hearing loss, with complete dropout of several frequencies, should not be tested in the involved ear because the waveforms produced are very poor or absent.

Another application of the BAER is the detection of a second occult lesion for the diagnosis of MS. One of the criteria for the diagnosis of such a lesion is a normal wave V latency with a decreased wave amplitude. Stockard et al feel that a reduction in amplitude alone is not as significant as prolongation of wave latency. We agree, because at high decibel stimulation the sole abnormality in both conductive and neurosensory hearing losses may be an abnormal amplitude and the results may be mistakenly interpreted as suggestive of MS (Fig. 5).

More research is needed to determine if the BAER can effectively differentiate central versus peripheral etiologies of hearing losses shown on an audiogram. Until this research is done, we feel that whenever an abnormal BAER is elicited an audiogram, if possible, should be obtained.

The major drawback of the click-elicited BAER described in this paper is its lack of frequency specificity. The click stimulates a band of frequencies and the evoked response is generated from a large area of the cochlea. When restricted areas are stimulated, the responses have lower amplitudes and the waves produced are poor or absent. However, specific areas of the cochlea, both basilar and apical, can be evaluated with the use of derived potentials and theoretically tested by presenting a tone pip with the appropriate rise-fall times. The frequency following response also has great potential in obtaining tone specific information.

The major drawback with the BAER is machine time and cost. To test air and bone conduction using clicks, requires approximately two hours for one subject. To reconstruct an entire audiogram by testing several frequencies would theoretically require a great deal of time and the cost would be astronomical.

CONCLUSION

The BAER is a clinically useful tool for the objective measurement of both organic and nonorganic hearing loss. This technique is not affected by such subject variables as attention or many forms of sedation, which plaque cortical evoked response audiometry. Unlike electrocochleography, it is totally noninvasive. Disadvantages of the BAER are twofold. First, the recording of tone specific information is still in the research stage; second, a great deal of machine time is often required to evaluate fully a subject. The authors of this paper believe that the BAER shows great promise and that further research will develop the BAER technique into one of the major tools of the otolaryngologist.

REFERENCES

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ACKNOWLEDGMENT - We wish to thank the Department of Audiology and Otolaryngology at the Gundersen Clinic for their support in this project, and the Volunteer Office at LaCrosse Lutheran Hospital for supplying many of the volunteers presented in this paper.