Brainstem Auditory Evoked Responses In Brainstem Infarction

Edward Faught, M.D. and Shin J. Oh, M.D.

SUMMARY Brainstem auditory evoked responses (BAERs) were recorded in 40 patients with clinically definite brainstem infarction, and results were compared to localizations from physical signs and CT scans. The BAER was abnormal in 92% of patients with evidence on physical examination of dysfunction of lateral structures in the pons or midbrain. Normal BAERs were seen with medially-situated or medullary lesions. When both rostrocaudal level and laterization were considered, the BAER indicated damage in additional areas not evident on physical examination in 25% of patients. However, physical signs indicated damage in areas not reflected by the BAER in 22% of patients. Therefore, the BAER complements the localization obtained from physical findings. BAERs were abnormal in more of these patients than were CT scans, and thus are useful for confirmation of bedside impressions.

Brainstem auditory evoked responses are sensitive indicators of demyelinating and neoplastic lesions involving auditory pathways in the pons and midbrain. Abnormal BAERs have also been described in brainstem strokes.1,2 We wished to compare the accuracy and sensitivity of the BAER method with that of the physical examination and of computerized tomography in patients with brainstem infarction. Most patients with brainstem strokes are readily identified as such at the bedside. However, on occasion a test to confirm the diagnosis or to evaluate further the extent and location of the lesion is desirable. Computerized tomography (CT) reliably identifies hemorrhage, but may not demonstrate an ischemic lesion. For these reasons, we believed that BAERs might serve a useful complementary role in the area of brainstem stroke.

Methods

Patient Population

The study population consisted of fifty-four consecutive patients with clinical diagnoses of brainstem infarction who were referred to the Evoked Potential Laboratory of the University of Alabama Medical Center for testing. They were referred by physicians of the Neurology Services for confirmation of the diagnosis. The clinical records of these patients were reviewed by one of us (E.F.), and 40 patients were selected in whom the diagnosis of brainstem infarction appeared very secure on clinical grounds. On the basis of their physical findings, patients were classified as to whether they had medullary, pontine, or midbrain syndromes, whether these were medial or lateral, and whether they were unilateral or bilateral. Cases with indeterminate clinical findings were excluded. BAER results had been interpreted for all of these patients by the same physician (S.J.O.). CT scans were available for 30 patients, and autopsy data were available for 5.

Scans were done on a General Electric 8800 scanner in 13 patients, and on an EMI 1010 in 17. Hemorrhagic strokes, by CT or autopsy, were excluded. The mean age was 61, ranging from 46 to 80. Eight were female, 32 male. In-hospital mortality was 20%.

BAER Method

BAERs were recorded simultaneously from a central vertex (Cz) electrode referred to the ear ipsilateral to the click stimulus (Ai), and in a separate channel to the ear contralateral to the stimulus (Ac), using a Nicolet CA 1000 or Tracor 3000 averager. Stimuli were square-wave clicks of alternating rarefaction-condensation lasting 100 µsec each, delivered at a rate of 10 per second to one ear at 75 dB HL intensity. The reference for noise intensity was a group of 30 persons with normal hearing for clicks. This was also the control group for BAER latencies: mean age was 35.1, SD 10.8, range 18–61 years. Two thousand repetitions were averaged, twice for each ear. The other ear was masked with white noise. For paper records, the standard EEG polarity convention was observed: positivity at the grid 1 electrode (in this linkage, Cz) relative to the grid 2 electrode (Ai or Ac) produced a downward pen deflection. Latencies were considered abnormal if they exceeded two standard deviations from our normal mean values. Amplitude deviations were interpreted with caution: generally no comment was made unless a wave was totally absent. Figure 1 illustrates a normal BAER, table 1 lists our normal values with this method.

Results

Abnormal BAERs were recorded in 70% of these 40 patients. They were abnormal in 87% (20/23) of patients with pontine clinical syndromes, 73% (8/11) of those with mesencephalic syndromes, and none of 6 patients with medullary syndromes.

Rostrocaudal Level

The auditory structures are for the most part laterally placed in the pons and midbrain. Thirty-one patients had clinical signs of dysfunction of lateral structures in the pons or midbrain such as sympathetic tracts, cochlear or vestibular nuclei, cerebellar peduncles, spinalthalamic and trigemino-thalamic tracts, and lateral
Absence of Wave IV or V

**Figure 1.** Example of normal BAER with designation of waves. We advocate recording from reference sites at both the ipsilateral ear (the one stimulated), and the contralateral ear because wave morphology may vary. Cz-Ai: ear reference ipsilateral to stimulus. Cz-Ac: ear reference contralateral to stimulus.

**TABLE 1 Normal BAER Values**

<table>
<thead>
<tr>
<th>Wave</th>
<th>Mean</th>
<th>+ 2 SD</th>
<th>+ 3 SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave I</td>
<td>1.70</td>
<td>2.08</td>
<td>2.26</td>
</tr>
<tr>
<td>Wave II</td>
<td>2.81</td>
<td>3.21</td>
<td>3.41</td>
</tr>
<tr>
<td>Wave III</td>
<td>3.85</td>
<td>4.20</td>
<td>4.37</td>
</tr>
<tr>
<td>Wave IV</td>
<td>5.06</td>
<td>5.48</td>
<td>5.68</td>
</tr>
<tr>
<td>Wave V</td>
<td>5.66</td>
<td>6.22</td>
<td>6.50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Interpeak latencies (msec)</th>
<th>I-III</th>
<th>III-V</th>
<th>I-V</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.11</td>
<td>2.19</td>
<td>2.37</td>
</tr>
</tbody>
</table>

**TABLE 2 Absent Waves or Prolonged Latencies (measured from wave I) in Patients with Lateral Brainstem Infarctions**

<table>
<thead>
<tr>
<th>Wave</th>
<th>Midbrain syndromes (Wallenberg) patients</th>
<th>Medullary syndromes (Ponto- medullary syndromes patients</th>
<th>Mid-upper pontine syndromes patients</th>
<th>Midbrain syndromes patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0</td>
<td>4 (80%)</td>
<td>13 (81%)</td>
<td>4 (40%)</td>
</tr>
<tr>
<td>II</td>
<td>0</td>
<td>4 (80%)</td>
<td>13 (81%)</td>
<td>3 (30%)</td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>5 (100%)</td>
<td>13 (81%)</td>
<td>5 (50%)</td>
</tr>
<tr>
<td>IV</td>
<td>0</td>
<td>1 (20%)</td>
<td>13 (81%)</td>
<td>8 (80%)</td>
</tr>
<tr>
<td>V</td>
<td>0</td>
<td>1 (20%)</td>
<td>13 (81%)</td>
<td>8 (80%)</td>
</tr>
<tr>
<td>III-V</td>
<td>0</td>
<td>1 (20%)</td>
<td>13 (81%)</td>
<td>8 (80%)</td>
</tr>
</tbody>
</table>

N = 30, mean age 35.1 ± 10.8. Alternating polarity clicks, 75 dB HL, Cz-Ai linkage.
ipsilateral to the stroke. An autopsy series would be helpful in determining the anatomical explanation for this lateralization, and whether it applies to pure midbrain lesions as well as to pontine lesions.

Normal BAERs

Most of the 12 normal BAERs among these patients were readily understandable. Six were in patients with medullary syndromes, 5 Wallenberg and one medial. Three others were in patients with only paramedian signs. Interestingly, 2 had bilateral internuclear ophthalmoplegias and 1 unilateral. The latter patient was also “locked in”: conscious but unable to move. The remaining 3 patients had some evidence of lateral pontine or midbrain dysfunction, so that the normal BAERs were not expected. The presence of a Horner’s syndrome was the single clinical sign most predictive of an abnormal BAER, except where it was part of a Wallenberg syndrome. The sympathetic tracts lie close to the auditory pathways in the upper brainstem, and only 2 of 18 patients with Horner’s syndrome from lesions at these levels had normal BAERs.

CT Scanning and Other Testing

Computerized tomographic scanning demonstrated the infarction in only 27% of cases. In the one instance in which the CT demonstrated a low density lesion while the BAER was normal, an autopsy showed encephalomalacia in the midbrain and pontine tegmentum in the midline, sparing the auditory pathways. In the largest group of patients, 47%, the BAER was abnormal and the CT normal.

Somatosensory evoked potentials were not done routinely in this study, but there were 3 patients — 2
TABLE 3  Lateralization of Brainstem Infarctions by Brainstem Auditory Evoked Potentials

<table>
<thead>
<tr>
<th>BAER abnormality</th>
<th>Clinical syndrome*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unilateral pons</td>
</tr>
<tr>
<td>Ipsilateral</td>
<td>9</td>
</tr>
<tr>
<td>Contralateral</td>
<td>1</td>
</tr>
<tr>
<td>Bilateral</td>
<td>0</td>
</tr>
</tbody>
</table>

*Lateral syndromes only.
†BAER abnormal on one side only.

Discussion

We believe that BAERs have a useful place in the diagnosis of brainstem strokes. Like other electrophysiological techniques, they usually serve to confirm and extend the clinical impression, rather than to dispute it. In our series, the BAER abnormalities suggested that an additional side or level of the brainstem was involved, beyond that reflected by the symptoms and signs, in 25% of cases. However, in another 22%, clinical presentation indicated more extensive dysfunction than could be deduced from BAERs. The BAER therefore complements, rather than duplicates, the physical examination.

Several cases of abnormal BAERs in brainstem strokes have been reported previously. Kjaer reported a correspondence between clinical and BAER localization in about two-thirds of fifteen patients with infarctions. Hashimoto et al included twelve cases of pontine infarction among their 64 cases of varied brainstem pathology, but did not analyze them separately. Green and McLeod reported four patients, and Brown et al nine, with brainstem infarctions studied by BAERs. Stern et al found an abnormal BAER in 22 of 35 (63%) patients with brainstem infarctions, a figure close to our 70%. These authors reported that an abnormal BAER was predictive of an unstable clinical course.

We believe that this simply reflects the relatively good prognosis and low likelihood of progression of the two groups of patients most likely to have normal BAERs: those with lateral medullary infarctions, and with small midline tegmental infarctions caused by occlusion of perforating branches of the basilar artery. It is important in this regard to distinguish between lateral medullary and lateral pontomedullary or pontine syndromes: the later have a worse prognosis because they may be due to clot in the basilar artery which may propagate. The BAER is obviously helpful in this distinction.

Localization of lesions in our group generally conformed to that reported by Stockard, whose series included 8 cases with brainstem vascular lesions. However, we noted some additional features.

Firstly, prolongation of I—III and II—III latencies was seen with normal I—V and III—V latencies in some patients with pontomedullary junction infarctions. In these cases, some impulses originating in the ear ipsilateral to the lesion must reach the upper brainstem via contralateral pathways. They somehow escape delay as they traverse the region of the ipsilateral cochlear nuclei, then cross in the trapezoid body. A recrossing of impulses to the ipsilateral side may occur at higher levels via connections between the laterallemniscal nuclei and the commissure of the inferior colliculi. However, we can make no statements about function of the upper brainstem ipsilateral to the lower brainstem lesion in this situation, since a single functioning inferior colliculus may be quite sufficient to generate a
wave V of normal latency and amplitude to stimuli arriving from either ear.

We also observed a good correspondence between unilateral brainstem clinical signs and abnormal BAERs on stimulation of the ipsilateral ear, which we have reported in an earlier group of patients. This occurs despite anatomic studies showing that the majority of auditory pathway fibers decussate in the trapezoid body. This lateralization holds true for pontine lesions, but we are less certain about midbrain lesions. We have not seen enough patients with autopsy-proven lesions confined to one side of the midbrain to be sure about the side of the BAER abnormality.

As a diagnostic test to complement the physical examination, BAERs are more sensitive than CT scans in brainstem infarction. Of course, an abnormal CT scan, when obtained, may be more anatomic in definition. CT scans are often unrevealing: Campbell et al. reported normal scans in 9 of 17 patients with infratentorial infarcts, with no significant difference whether the scan was done during the first 24 hours, or 7–10 days later. Sensitivity may improve with technical advances in scanning, particularly when 5 mm slices of the posterior fossa are taken and magnified, a technique not always employed in our series. However, even in more recent series, the CT frequently failed to delineate brainstem infarctions. Finally, we see two areas where further investigation would be beneficial. A large autopsy series of patients with vascular disease who had BAERs would be helpful. Not only the location, but also the degree of pathological change correlating with wave changes could be elucidated. A related question is: can BAERs be used to diagnose ischemia of the brainstem in the absence of physical signs? Theoretically, the answer is “yes” since BAERs sometimes indicated dysfunction in areas clinically uninvolved. In fact, Ragazonni et al. have reported that 14 of 26 patients with reversible brainstem ischemic attacks had BAER abnormalities (latency delays > 2 SD from normal) when tested after recovery. Kjaer on the other hand, found only 1 of 9 patients had abnormal BAERs after vertebrobasilar transient ischemic attacks. All of the patients in the present series had deficits lasting well over 24 hours. Further studies of BAERs in equivocal syndromes of brainstem ischemia and in vertebrobasilar transient ischemic syndromes should be undertaken, since the test is likely to have its greatest utility when the clinical picture is not crystal clear.

References

Brainstem auditory evoked responses in brainstem infarction.
E Faught and S J Oh

Stroke. 1985;16:701-705
doi: 10.1161/01.STR.16.4.701

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/16/4/701