SUMMARY Brainstem auditory evoked responses (BAERs) were recorded in 40 patients with clinically defined 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 lateralization 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.

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, spinothalamic and trigemino-thalamic tracts, and lateral
served at more caudal levels than the clinical level of damage. An example of this “caudal shift” of BAER abnormality is seen in figure 2.

Five patients with infarctions judged clinically to be around the pontomedullary junction displayed an interesting BAER pattern. All had abnormal I–III latencies on stimulation of the ipsilateral ear, and normal III–V latencies bilaterally. Rather surprisingly, 4 of the 5 also had normal I–IV and I–V latencies bilaterally. This suggests that transmission through fibers crossing low in the auditory pathway is fast enough to produce normal latencies at higher levels, even when the anatomically shorter uncrossed pathways are delayed. This “skip” pattern of wave delay, seen usually with low pontine syndromes, is also seen occasionally with more rostral lesions. This pattern is illustrated by figure 2.

**Lateralization**

Abnormal BAERs were usually observed upon stimulation of the ear ipsilateral to the clinical lesion. In table 3, laterality of the clinical syndrome is compared to lateralized BAER abnormalities. Results are listed for 31 patients with clinical syndromes involving lateral structures on one or both sides of the pons and midbrain. In 78% of cases (14 of 18) with unilateral clinical signs, the BAER abnormalities were seen only on stimulation of the ipsilateral ear. The BAER indicated additional damage on the contralateral side in 3 patients. The single patient whose BAER was abnormal only on stimulation of the ear contralateral to the lesion also had questionable clinical evidence of bilateral damage. The BAER was often more sensitive than table 3 indicates: in 7 of the 13 patients with bilateral clinical signs, the signs were greater on one side, and in each case the BAER abnormalities were worse on that side. Figure 3 is from a patient with a unilateral clinical midbrain syndrome, demonstrating ipsilateral BAER abnormalities. However, this patient may have had subclinical involvement of the pons on the same side, since the ipsilateral wave III is also delayed. In patients who had only delays of the later waves, IV and V, the same laterization held true: the delay was observed only with the stimulation of the ear.

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

<table>
<thead>
<tr>
<th>Wave</th>
<th>Medullary syndromes (Wallenberg)</th>
<th>Pontomedullary syndromes</th>
<th>Mid-upper pontine syndromes</th>
<th>Midbrain syndromes</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>II</td>
<td>0</td>
<td>4 (80%)</td>
<td>2 (12%)</td>
<td>0</td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>5 (100%)</td>
<td>8 (50%)</td>
<td>3 (30%)</td>
</tr>
<tr>
<td>IV</td>
<td>0</td>
<td>1 (20%)</td>
<td>13 (81%)</td>
<td>5 (50%)</td>
</tr>
<tr>
<td>V</td>
<td>0</td>
<td>1 (20%)</td>
<td>14 (88%)</td>
<td>8 (80%)</td>
</tr>
<tr>
<td>III-V</td>
<td>0</td>
<td>2 (12%)</td>
<td>16 (96%)</td>
<td>10 (67%)</td>
</tr>
<tr>
<td>All waves</td>
<td>normal 4 (100%)</td>
<td>0</td>
<td>2 (20%)</td>
<td>1 (10%)</td>
</tr>
</tbody>
</table>

**Table 1**  Normal BAER Values

<table>
<thead>
<tr>
<th>Absolute latencies (msec)</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>

| Interpeak latencies (msec) |   | | |
| I–II                      | 2.11 | 2.43 | 2.59 |
| III–V                     | 1.82 | 2.19 | 2.37 |
| I–V                       | 3.94 | 4.42 | 4.66 |

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

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**Figure 2.** Left midbrain stroke. This figure illustrates several points: 1) "Caudal shift" of BAER abnormality. The BAER indicated more extensive dysfunction than the clinical exam. Clinically, the patient had a Weber syndrome with a III nerve palsy and contralateral hemiparesis. However, there is prolonged I–III latency and delay of waves III and IV on the affected side, indicating pontine dysfunction by BAER criteria. 2) BAER lateralization. The abnormal waveforms are seen with stimulation of the ear ipsilateral to the stroke, the left ear. 3) "Skip" latency delay. The left I–III interpeak latency is 2.66, >3 SD from normal, the absolute III latency is 4.38, >3 SD from normal. However, the latency of waves V (6.14), and the I–V interpeak latency (4.42), are normal. This pattern is most striking with pontomedullary junction infarctions, but can be seen with more rostral lesions, as in this case. Mean latencies, 2 trials, Cz-Ac linkage. Alternating polarity clicks, 75 dB HL.
with Wallenberg syndromes and 1 with lateral pontine syndrome — in whom BAERs were normal but SEPs were abnormal.

**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 latter 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 lateral lemniscal 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

![Right ear stimulation, Cz-Ai linkage](https://example.com/right耳朵刺激，Cz-Ai连接)

![Left ear stimulation, Cz-Ai linkage](https://example.com/left耳朵刺激，Cz-Ai连接)

**Figure 3.** Right midbrain infarction by clinical signs, possible subclinical pontine involvement. Wave III and later waves are distorted and delayed in the BAERs produced by right ear stimulation at all intensities. All waves appear normal when the ear contralateral to the side of the stroke is stimulated.
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 anatomical 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 anatomically definitive. 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

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