Evaluation of Brainstem Stroke Using Brainstem Auditory Evoked Responses

BARNEY J. STERN, M.D., ALLAN KRAMHOLZ, M.D., HOWARD D. WEISS, M.D., PHILIP GOLSTEIN, M.D., AND KENNETH C. HARRIS, EEG. T.

SUMMARY Brainstem stroke syndromes are primarily determined by clinical criteria. There are few diagnostic procedures which are of benefit for the evaluation of brainstem ischemic events. Brainstem auditory evoked responses (BAERs) are a new electrophysiologic technique for assessing brainstem function. To evaluate the use of BAERs in patients with brainstem ischemic events, 35 individuals with recent brainstem strokes, selected by strict clinical criteria, were evaluated with BAERs. The initial BAER was abnormal in 22 of 35 patients (63%). When the clinical course and site of the lesion are correlated with the BAER results, several trends emerge. An unstable course, characterized by progression or remission and relapse, was present in 19/35 (54%) of patients, and 15/19 (79%) of these individuals had an initially abnormal BAER. The other 16 brainstem stroke patients with a stable clinical course had an initially abnormal BAER in 7 instances (44%). This difference is statistically significant at the p = 0.04 level. The principal sites of ischemia were mesencephalic in 11/35, pontine in 13/35, and medullary in 11/35. The association of an abnormal BAER with an unstable clinical course seemed independent of the site of the lesion. However, of the 9 deaths that occurred, all were in patients with mesencephalic or pontine lesions, and 8 of these individuals had an initially abnormal BAER.

Abnormal BAERs in patients with brainstem ischemic lesions correlate with an unstable clinical course. Furthermore, individuals with pontomesencephalic infarction and abnormal BAERs have an especially poor prognosis. The BAER may be of prognostic value in the early evaluation of patients with brainstem ischemic strokes.

THERE ARE FEW DIAGNOSTIC AIDS in the evaluation of brainstem ischemic events. Localization of intrinsic brainstem pathology depends on clinical evaluation, while the etiology of the dysfunction can often be defined by further neurodiagnostic methods. Brainstem stroke syndromes are defined by clinical criteria alone, and conventional diagnostic aids such as EEG and CSF evaluations add little to the clinical definition of the problem or in determining prognosis. Angiography may reveal atheromatous lesions and vessel occlusions, but this may not provide insight into the degree of parenchymous dysfunction or patient prognosis. Computerized tomography may be of assistance in evaluating brainstem hemorrhagic or neoplastic lesions but is usually of little value in the assessment of brainstem ischemic lesions.

Brainstem auditory evoked responses (BAERs) are a new electrophysiologic method of evaluating brainstem dysfunction. BAERs have been found to be useful for detecting or confirming the brainstem dysfunction associated with, for instance, brainstem glioma, central pontine myelinolysis, "locked-in" syndrome, and multiple sclerosis. Brainstem strokes have been included in series evaluating the use of BAERs, but there has not been a systematic study evaluating the BAER in a large population of patients with brainstem strokes.

In this study, 35 patients with ischemic brainstem strokes were studied with BAERs. The clinically determined principal site of ischemia was correlated with the BAER examination and clinical course of the patient. The correlations obtained may prove helpful in defining a group of patients at increased risk for an unstable clinical course and death.

Materials and Methods

The charts of 35 consecutive patients referred to the Neurology Division of The Sinai Hospital of Baltimore and identified as having an ischemic brainstem infarct were retrospectively reviewed. Other patients with a principally cerebellar lesion or with a hemorrhagic brainstem infarction as documented by CT scan and/or CSF evaluation were excluded from the study. Furthermore, patients with a likely embolic stroke (patients with atrial fibrillation and/or valvular heart disease) were also excluded from the study. All patients had a CT scan and most had a CSF evaluation. Thus, the 35 patients that formed the study group were thought to have a brainstem stroke on a principally thrombotic basis. Many patients were treated with anticoagulants but this factor was not specifically included in the analysis. The principal site of brainstem ischemia was identified as to midbrain, pontine, or medullary involvement. In some patients there was anatomical overlap, but the patient was assigned an anatomical site based on the site of maximal clinical impairment. The principal lateralization of the lesion was determined when possible. The clinical course of the patient was determined and the patient was characterized as having a stable course (unchanging or steadily improving) or unstable course (remitting-relapsing or progressively deteriorating). Patient outcome in terms of complete or incomplete recovery or death was noted. All 35 patients underwent a BAER evaluation. Several patients had serial BAER studies. Major conclusions are based on the first BAER evaluation. The clinical course of the patient was reviewed without specific knowledge of the BAER results.

Testing was done in an IAC sound-attenuated room...
if the patient was stable and could be transported. Otherwise, the test was performed at the patient’s bedside. All patients were studied in a supine position, with head support as necessary to minimize postural muscle activity. Sedation was not used.

Silver-silver chloride disc electrodes were placed according to the international 10-20 system at the vertex (Cz) and both mastoids (M1 and M2). Potentials were recorded between Cz and the mastoid ipsilateral to the click stimulus. The contralateral mastoid served as the patient ground. Electrode impedances were maintained below 5,000 ohms.

The auditory stimulus was a constant polarity, rarefaction click with a pulse duration of 0.1 msec. The stimulus was presented monaurally to each ear. Click intensity was 65dB above the patient’s subjective threshold (65dBSL). If the patient’s threshold was not obtainable, the stimulus was set 65dB above our standard for normal hearing adults in a similar environment (65dBHL). Masking was only used when there was a greater than 15dB difference in hearing threshold between a patient’s ears or when the threshold could not be determined. The ear contralateral to the click stimulus was masked with white noise at 20dBHL less than the intensity of the click stimulus. Clicks were presented at a rate of 10 Hz.

The amplifier gain was set at 10^4 with a filter bandpass of 150 to 3,000 Hz (3dB down). The responses were processed by a Nicolet CA-1000 clinical averager. The amplifier output could be monitored on an oscilloscope and averaging was not performed when the EEG was significantly contaminated by artifact. Each trial consisted of 2,000 to 4,000 individual responses which were averaged. Each averaged response was then repeated, at least once, to demonstrate reproducibility. Responses were recorded and superimposed by an X-Y Plotter. Vertex positivity was recorded as an upward deflection. Wave peak latencies were marked for waves I, III, V, and V N (negative V) (fig. 1). When a peak was not well defined, a midpoint of the wave was estimated. When waves IV and V were fused into a single complex, V N was used to measure interpeak latencies. This was necessary because of difficulty in accumulating adequate normative data for the relatively rare IV-V complex.

Interwave latencies for waves I, III, and V were determined and compared to data from 31 normal adult control subjects (table 1). Normative values were similar to those in previously published series.23,24 Interpeak latency was judged to be prolonged when the normal mean was exceeded by 3 standard deviations. Inter-ear latency differences were assessed for the wave I-V, I-III, III-V, I-V n, and III-V n intervals. The complete absence or absolute prolongation of waves I, III, V, or V N was also considered abnormal. Responses were interpreted as abnormal on the basis of amplitude only when the wave V or IV-V to I ratio was below 0.5. Wave amplitude was measured from the positive peak of a wave to the succeeding negative trough. When necessary, our standard techniques were modified to clarify wave forms. These modifications included: using click intensities up to 95dBHL, using stimulus rates up to 60 Hz, and recording from bipolar recording derivations such as Cz to the contralateral mastoid for better definition of wave V.

Results

Thirty-five patients with brainstem ischemic stroke were evaluated with BAERs. The principal site of ischemia was mesencephalic in 11/35, pontine in 13/35, and medullary in 11/35. There were 9 deaths, all in patients with a mesencephalic or pontine infarction. At the time of hospital discharge 5 patients had made a complete recovery (with resolution of neurological signs) and 21 patients had made an incomplete recovery. An initially progressively deteriorating or remitting-relapsing (unstable) course was apparent in 19 patients (54%) and 14 of these 19 patients had a principally mesencephalic or pontine lesion. A progressively improving or essentially unchanging (stable) course was present in 16 patients (46%) and 10 of these 16 patients had a principally mesencephalic or pontine lesion. Only one patient with an unstable course made a complete recovery at the time of hospital discharge.

Of the 19 patients with an initially unstable clinical course, 9 stabilized within 4 days of presentation. An additional 7 stabilized within one week of presentation and 3 patients were unstable for greater than one week.

At least one BAER examination was obtained on all patients. The mean duration from admission to BAER

![Figure 1](http://stroke.ahajournals.org/)

**Figure 1.** A. Brainstem auditory evoked response from a normal control. The wave latencies are: wave I = 1.72 msec, wave III = 3.96 msec, and wave V = 5.96 msec. B. Brainstem auditory evoked response from a patient with a brainstem stroke causing a 'locked-in' syndrome. The wave latencies are: wave I = 1.60 msec, wave III = 3.96 msec, and wave V = 6.60 msec. The wave I-V and wave III-V latencies are prolonged.
The predominant BAER abnormality was a prolongation of the wave I-V interval (fig. 1). An isolated wave I-III or III-V abnormality without an associated I-V delay was not found. It was not possible to establish a pattern consistently relating medullary, pontine, or mesencephalic lesions to specific BAER abnormalities. In a few individuals there was correlation of the pattern of the BAER abnormality, the site of the clinical lesion, and the hypothesized sites of origin for the BAER. 16, 25, 26 One patient with a medullary lesion and 3 patients with pontine lesions had unilaterally absent responses or a delayed wave I. Two additional patients with pontine lesions and 1 with a medullary lesion showed primarily a wave I-III delay. One patient with a pontine lesion demonstrated a predominant III-V delay. No patient with a mesencephalic lesion had primarily a III-V delay.

In no patient was the only abnormality a prolongation of the inter-ear wave I-V latency without an associated absolute wave I-V latency delay. Amplitude ratios never constituted the only basis of abnormality.

The difference in proportion of abnormal BAERs between the stable and unstable groups is statistically significant at p = 0.04.

**Table 2**

<table>
<thead>
<tr>
<th>Wave &amp; wave interval</th>
<th>Latencies to a 65 dB SL click (msec)</th>
<th>Inter-ear differences (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD*</td>
</tr>
<tr>
<td>I</td>
<td>1.70</td>
<td>0.12</td>
</tr>
<tr>
<td>III</td>
<td>3.79</td>
<td>0.17</td>
</tr>
<tr>
<td>V</td>
<td>5.72</td>
<td>0.19</td>
</tr>
<tr>
<td>V_N</td>
<td>6.38</td>
<td>0.23</td>
</tr>
<tr>
<td>I-V</td>
<td>4.01</td>
<td>0.18</td>
</tr>
<tr>
<td>I-III</td>
<td>2.10</td>
<td>0.12</td>
</tr>
<tr>
<td>III-V</td>
<td>1.91</td>
<td>0.13</td>
</tr>
<tr>
<td>I-V_N</td>
<td>4.68</td>
<td>0.27</td>
</tr>
<tr>
<td>III-V_N</td>
<td>2.59</td>
<td>0.17</td>
</tr>
</tbody>
</table>

*Standard deviation.

**Table 3**

<table>
<thead>
<tr>
<th>Clinical localization of infarct (total)</th>
<th>BAER abnormality lateralization*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bilateral</td>
</tr>
<tr>
<td></td>
<td>Total</td>
</tr>
<tr>
<td>Midbrain (8)</td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>3</td>
</tr>
<tr>
<td>Bilateral</td>
<td>5</td>
</tr>
<tr>
<td>Pons (7)</td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>3</td>
</tr>
<tr>
<td>Bilateral</td>
<td>4</td>
</tr>
<tr>
<td>Medulla (7)</td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>7</td>
</tr>
<tr>
<td>Bilateral</td>
<td>0</td>
</tr>
<tr>
<td>Total (22)</td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>13</td>
</tr>
<tr>
<td>Bilateral</td>
<td>9</td>
</tr>
</tbody>
</table>

*Lateralization refers to the ear from which an abnormal response was obtained relative to the side of the clinical lesion.

examination was 3 days (range 0–11 days). Twelve patients had 2 or more BAER examinations and in 9 patients the results of examinations one and two were in agreement; 8 patients had 2 abnormal tests and one patient had 2 normal tests. Three patients revealed changing results on successive tests: 2 patients abnormal to normal, one patient normal to abnormal.

If the first BAER examination is considered, 22/35 (63%) patients had an abnormal BAER. Eight of 9 patients who died had an abnormal initial BAER. Two of 5 patients who had a complete recovery at time of hospital discharge had an abnormal BAER; one of these patients had a lateral medullary syndrome and the other patient had a midbrain lesion. Eight of 11 patients with a mesencephalic lesion, 7/13 patients with a pontine lesion, and 7/11 patients with a medullary lesion had an abnormal BAER on initial testing.

If the clinical course is correlated with the BAER results, several trends emerge (table 2). An unstable course was present in 19 patients and 15 of these individuals (79%) had an initially abnormal BAER. However, of the 16 patients with brainstem strokes and a stable clinical course, only 4 (40%) had an initially abnormal BAER. Four of the 5 (80%) patients with a medullary lesion and an unstable course had an initially abnormal BAER. Three of the 6 (50%) patients with a medullary lesion and a stable course had an initially abnormal BAER. No patient with a medullary lesion died.

<table>
<thead>
<tr>
<th>clinical course</th>
<th>Normal BAER</th>
<th>Abnormal BAER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pontomesencephalic infarct (24)</td>
<td>stable 6</td>
<td>unstable 3 (1 death) 11 (8 deaths)</td>
</tr>
<tr>
<td>Medullary infarct (11)</td>
<td>stable 3</td>
<td>unstable 1</td>
</tr>
<tr>
<td>Total (35)*</td>
<td>stable 9</td>
<td>unstable 14</td>
</tr>
</tbody>
</table>

*The difference in proportion of abnormal BAERs between the stable and unstable groups is statistically significant at p = 0.04.
ear ipsilateral to the side of clinical involvement. The other 5 patients with unilateral brainstem disorders had abnormalities of their BAERs from both left and right ear stimulation (bilateral abnormalities). In our series, no patient with a unilateral brainstem lesion, based on clinical criteria, had an abnormal BAER only on stimulation of the ear contralateral to the lesion. Of the 9 patients with bilateral, clinical brainstem dysfunction, 5 had bilateral BAER abnormalities while in 4 the abnormality was unilateral.

Discussion

There are few diagnostic aids to assist the clinician in the evaluation of brainstem ischemic strokes. Most EEG patterns are nonspecific though the rare pattern of alpha or spindle coma may be of value in lesion localization or prognosis. CT scans often do not visualize ischemic brainstem strokes because of the small tissue volume involved as well as the artifact common on posterior fossa views.

BAERs are an electrophysiological phenomenon thought to occur as a result of the activation of brainstem “generators” along the auditory pathway (fig. 1). Preliminary studies suggested wave I originated from the 8th nerve, wave II from the cochlear nucleus, wave III from the superior olivary complex, wave IV from the nucleus of the lateral lemniscus and/or region of the inferior colliculus, and wave V from the inferior colliculus. Recent investigations have raised doubts about the specificity of these previously postulated BAER generators. Despite controversy as to the precise brainstem site of origin for specific components of the BAER, current theories still approximate the initial hypotheses. A lesion affecting the function of any of the hypothesized generators, or pathways between generators of the BAER, might affect the BAER. Disruption of the BAER response may not only be due to a pathologically definable lesion but may also be caused by a physiological, subclinical alteration involving the BAER generators or pathway.

Because the BAER is a reflection of brainstem function, 35 patients with clinically defined brainstem ischemic strokes were examined with BAERs. Twenty-two of 35 (63%) patients had an abnormal BAER on initial evaluation. Unlike prior reports, not all patients with brainstem strokes had abnormal BAERs. When subgroups were examined, several trends, which may have therapeutic implications, became apparent.

BAERs may be of value in identifying patients at high risk for an unstable clinical course. Two recent studies have stressed the early unstable course in vertebrobasilar infarction. Jones et al. found a progressive disability or remitting-relapsing course in 54% of patients whereas Patrick et al. found 51% of their patients exhibiting a similar course. The current series is in substantial agreement with these studies in that 54% of our patients were unstable. Furthermore, we agree with Patrick et al. that fluctuation can occur for at least one week after presentation. In particular, 19 of 35 patients with a brainstem ischemic lesion exhibited an unstable course, and 15 of these 19 patients (79%) had an abnormal BAER. The other 16 brainstem stroke patients with a stable clinical course had an initially abnormal BAER in 7 instances (44%). This difference is statistically significant at p = 0.04 using the one-tail Fisher's exact test. All patient deaths involved mesencephalic or pontine lesions and 8 of 9 of these patients exhibited abnormal BAERs on the first evaluation. This striking finding for patients with pontomesencephalic strokes may be the major reason for the overall correlation of an unstable clinical course with an abnormal BAER. Of the 3 patients who had clinical fluctuation for more than one week, all had abnormal BAERs in the setting of pontine or mesencephalic lesions.

Although 7/11 patients with a medullary lesion had an abnormal BAER, none of these patients died. This possible discrepancy between the value of BAERs in identifying patients at high risk for death with pontomesencephalic infarcts as opposed to medullary infarcts may be due to the regional anatomy of the auditory pathway and its relation to the blood supply of the medulla as compared to the pontomesencephalic regions.

The lateral medullary syndrome tends to be secondary to vertebral and/or posterior inferior cerebellar artery occlusion, whereas pontine or mesencephalic infarctions are often related to atherosclerotic disease of the basilar artery or its branches. The lateral medullary syndrome patient may thus have a different natural history than patients with primarily basilar artery disease. The natural history of the lateral medullary syndrome is generally benign in the acute stage, and all of the patients in this series survived. However, an occasional patient presenting with a lateral medullary infarct will progressively deteriorate, and 5/11 (45%) patients in this series did have an initially progressive or remitting-relapsing course.

Although the eighth nerve enters the pons and the cochlear nuclei lie within the pons, the perfusion of these structures may be dependent on some of the same vessels perfusing the lateral medullary region. The eighth nerve is usually perfused via the anterior inferior cerebellar artery (AICA) and/or the internal auditory artery. However, if the AICA is hypoplastic or absent, the ipsilateral posterior inferior cerebellar artery may perfuse the areas normally served by the AICA. As described by Duvernoy, the cochlear nuclei are perfused by rami of the vertebral and posterior inferior cerebellar arteries. These have superficial anastomoses with rami of the AICA and rami arising directly from the initial segment of the basilar artery. The AICA is probably the dominant vessel to the cochlear nuclei but rami of the vertebral and proximal basilar arteries are involved. The involvement of the cochlear nuclei and eighth nerve is not stressed in reviews of the lateral medullary syndrome, but dysfunction of these structures would not be surprising given the potential dependence of these structures on blood supply from the vertebral and/or posterior inferior cerebellar arteries. Therefore, a high proportion of patients (7
of 11 in this series) might be expected to have abnormal BAERs in the setting of a lateral medullary infarct and yet display the relatively benign course characteristic of many, but not all, patients with a lateral medullary syndrome.

Review of the vascular anatomy supplying the remaining BAER ‘‘generators’’ may help explain the correlation of clinical outcome in pontomesencephalic infarction to the BAER. According to Duvernoy,44 the superior olivary nuclei are principally perfused via the AICA. The nucleus of the lateral lemniscus and the pontine portion of the lateral lemniscus are perfused via direct branches of the basilar artery and via rami arising from the AICA and superior cerebellar arteries. The mesencephalic portion of the lateral lemniscus is supplied by rami arising from the superior cerebellar artery and the collicular and posteriomedial choroidal arteries (which originate from the posterior cerebral artery). The inferior colliculus is perfused via the superior cerebellar and collicular arteries. Therefore the integrity of the hypothesized BAER generators for waves III, IV, and V is dependent on perfusion arising in large part from the basilar artery. Although a patient with a pontine or mesencephalic infarct may not clinically demonstrate widespread dysfunction, if an area of the auditory pathway is physiologically disrupted, an abnormal BAER may result. Thus, an abnormal BAER may indicate vascular compromise more widespread than clinically apparent and thus may also be an indicator of more severe basilar artery disease. This may account for the correlations noted between an abnormal BAER and a high risk of death for infarction in the basilar artery dependent pontomesencephalic areas.

Prediction of clinical course in patients with brainstem strokes is difficult. Coma is commonly associated with a poor outcome25, 26, and although other patterns of neurological deficits66 have been associated with a poor survival they have not been correlated with the clinical course. Two patients in this series were admitted to the hospital in coma; one progressively deteriorated and died and the other improved and survived. Both of these patients had an abnormal BAER. If these patients are excluded from the series, the correlations noted between an abnormal BAER and clinical course are still statistically significant (p = 0.03, one tail Fisher’s exact test). Four patients underwent a BAER examination while comatose. All of these patients died and in all the BAER was abnormal. If patients in coma at the time of the BAER are excluded from the series, the correlations noted are no longer statistically significant, but this may be related to the small size of the remaining sample. However, there continues to be an apparent trend relating an abnormal BAER to an unstable clinical course in patients who are not comatose at the time of the BAER evaluation. A larger series will be needed to establish definitive correlations between the clinical course and the BAER in the non-comatose brainstem stroke patient.

Several other observations of patient subgroups deserve mention. Six patients had an internuclear ophthalmoplegia (INO). Two of these patients had normal BAERs and no hemiplegia and both survived whereas 4 of the patients with abnormal BAERs had an associated hemiplegia and 2 of these 4 patients died. Again these findings lend support to the value of BAERs in detecting more widespread physiological dysfunction. The incidence of BAER abnormality for patients with an INO is similar to that reported in multiple sclerosis.37, 48

Three patients developed the ‘‘locked-in’’ syndrome. Pathologically this is usually characterized by a basilar artery thrombosis with an associated ventral pontine infarction with variable extension to the pontine tegmentum.49-51 Though other etiologies of ‘‘locked-in’’ syndrome are known,52-56 the 2 surviving patients had nothing to suggest a pathophysiology other than basilar artery occlusion and in one case a basilar artery occlusion was demonstrated angiographically. The 2 survivors had an initially abnormal BAER whereas the deceased patient had an initially normal BAER. On post-mortem examination of the one non-survivor there was no overt infarction of the pontine tegmentum. In this limited patient subgroup BAERs did not seem to be helpful for patient prognosis. Two patients had a prolonged wave I-V interval, and in one patient this was predominantly due to a wave III-V interval delay. A characteristic wave III-V interval delay in ‘‘locked-in’’ syndrome has been suggested previously.12, 13 The nonsurvivor with an initially normal BAER subsequently developed a prolonged wave III-V interval. As mentioned, the pontine tegmentum was overtly intact on microscopic evaluation, thus lending support to a hypothesized more widespread physiological dysfunction involving the pontine auditory pathways.

The value of serial BAERs is not evident in this series. Nine of 12 patients had similar results on consecutive tests. However, this study was not designed to evaluate the usefulness of serial BAERs and we cannot comment on this further. Ideally the BAER should have been obtained as early as possible during the patient’s clinical course. The mean delay of 3 days in obtaining the BAER is within the period of high risk for an unstable course and is probably a fair indicator of the acute clinical status.

The ability of the BAER abnormality to consistently localize the level of a brainstem lesion was not impressive in our study. No patient with a mesencephalic lesion had a primarily III-V interval delay. Wave I-III interval abnormalities and abnormalities of wave I or the initial portion of the BAER were more commonly seen with medullary and pontine lesions. But these abnormalities were not always consistent, and it was not possible to develop a catalogue of specific BAER abnormalities based on the location of lesions. This could be explained, in part, by the fact that recent evidence suggests that the brainstem generators for the BAER are complex and the various components of the BAER may actually be dependent on multiple generators.36, 32, 33 Furthermore, in patients with abnormal responses, wave forms, especially wave III, were dis-
ruptured making interwave latency measurements difficult. In several patients with pontine or medullary lesions, it was difficult to obtain any response on stimulation of the ear ipsilateral to the brainstem lesion. This may have localizing value, but it prevents evaluation of interwave intervals in this group.

There was a tendency to lateralize the BAER abnormality to the ear ipsilateral to the clinical brainstem disorder (table 3). This was perhaps most dramatic with pontine lesions, but seemed true at all brainstem levels. This suggests that ipsilateral brainstem auditory pathways may be most important in producing abnormalities in the BAER in patients with brainstem vascular disease, regardless of the level of the lesion. A recent study of lateralized brainstem lesions reached similar conclusions.21 However, further studies will be necessary to confirm this, and the phenomenon may be more specific to vascular disease of the brainstem than to other types of pathology.

There have been few previous studies relating the results of evoked response examinations to patient outcome.57-59 This series demonstrates the unstable course of many patients with a vertebrobasilar infarction and emphasizes the potential value of BAERs in identifying a subgroup of patients at high risk for an unstable clinical course. The BAER also may define a subgroup of patients with pontomesencephalic lesions who are at especially high risk of dying. We agree with Patrick, et al. that "there is a need for a randomized study of therapy may be of benefit.

especially high risk of dying. We agree with Patrick, et al. that "there is a need for a randomized study of therapy may be of benefit.

References

34. Muller AR, Jannetta P, Bennett M, et al.: Intracranially recorded responses from the human auditory nerve: New insights into the
Evaluation of brainstem stroke using brainstem auditory evoked responses.
B J Stern, A Krumholz, H D Weiss, P Goldstein and K C Harris

*Stroke*. 1982;13:705-711
doi: 10.1161/01.STR.13.5.705

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

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/13/5/705

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Stroke* is online at:
http://stroke.ahajournals.org//subscriptions/