Brain Stem Auditory Evoked Potentials as a Tool in the Clinical Assessment of Children With Posterior Fossa Tumors

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Abstract

The use of brain stem auditory evoked potentials (BAEP) as a diagnostic modality in children with posterior fossa neoplasms is described. Thirty-one patients were examined; their diagnoses were medulloblastoma (12), brain stem glioma (9), cerebellar astrocytoma (6), and ependymoma (4). Distinct differences in the type and severity of waveform abnormalities were observed among the different tumor types, possibly related to location and invasiveness. Medulloblastomas frequently demonstrate normal waveforms, while brain stem gliomas demonstrate severe disruption of BAEP patterns. Ependymomas may result in a variety of abnormal studies, while cerebellar astrocytomas induce mild abnormalities or result in a normal exam. The use of brain stem auditory evoked potentials in the diagnosis of pediatric brain tumors, as well as the underlying mechanisms of the abnormalities, is discussed. (*J Child Neurol* 1987;2:272–275)

Brain tumors constitute the most common form of solid tumor among children.[1] About 50% of the brain tumors in children occur in the posterior fossa and are primarily cerebellar astrocytomas, primitive neuroectodermal tumors (medulloblastoma), ependymomas, and brain stem gliomas.[2] In the past decade, the most effective diagnostic tool for brain tumors, aside from clinical examination, has been computed tomographic (CT) scans. The posterior fossa, however, because of its bony artefacts and small, narrow structures is the area of the brain least amenable to visualization by CT scan.[3] More recently, magnetic resonance imaging (MRI) has become a new powerful modality for assessing and quantitating the posterior fossa tumors of childhood.[3] Both CT scans and MRI provide detailed anatomical data about size and localization of posterior fossa tumors but do not provide any functional information about posterior fossa structures. The brain stem auditory evoked potential (BAEP) has been shown to be useful for obtaining functional information about pathways within the brain stem.[4] Several authors have described, anecdotally and in small series, changes in the BAEP due to posterior fossa tumors of childhood.[5,6] As yet, however, there has been no study involving a large number of children that has assessed the role of BAEP as a diagnostic tool for posterior fossa tumors. Particularly, the possible specificity of BAEP with regard to the different tumor types, and its role in predicting outcome, recognizing recurrence, and monitoring the effects of chemotherapy have not been addressed. This report discusses our experience during a 5-year period measuring BAEPs in a series of 31 children with posterior fossa tumors. A further longitudinal study, correlating changes in patterns of BAEP with the patient's clinical course and therapeutic manipulation, is in process.

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Patient Population

Children who were patients at either M. D. Anderson Hospital or The University Children's Hospital at Hermann Hospital between 1981 and 1985 and who had a diagnosis of posterior fossa tumor participated in this study. Diagnoses were confirmed histologically; pathologic material obtained outside these

institutions was routinely reviewed by our pathologists, and, in cases of disagreement, our diagnosis was used for classification. Rarely, in a case of brain stem glioma, a typical clinical and neuroradiologic picture without biopsy was sufficient to make a diagnosis. These patients were either prior to treatment or remote from treatment at the time of performance of the electrophysiologic study. Patient characteristics are summarized in Table 1.

Methods

The BAEP studies were obtained on a Grass Model 10 ERS four-channel instrument with 1,024 points per channel or on a Tracor Northern 3500 machine with a sampling rate of 40 kHz. Four overlapping trials of 1,024 stimuli each were performed for each ear, and measurements were made from the composite waveforms. Monaural stimulation was performed with rarefaction clicks and contralateral masking; intensity was 85 dB hearing level in sedated children or 70 dB sensation level with cooperation. A vertex to ipsilateral ear derivation was used, with additional derivations employed as needed to confirm the waveforms. A trial of condensation and one of alternating clicks was usually added for comparison. When necessary, patients were sedated with chloral hydrate at a dose of 50 to 75 mg/kg, although recordings were attempted without sedation when possible.

Absolute latency values were measured by cursor for BAEP waveforms I through V, and interwave latency values were measured for I–III, III–V, and I–V intervals. Absolute amplitude values were mesured only for waveforms I and V, and the amplitude ratio of I to V (I:V) was calculated. The interwave latency and I:V results were compared with laboratory normal values, and abnormal results were placed into two categories—delayed and dispersed. Delayed responses were those in which the I-V interwave latency measurement exceeded 4.6 milliseconds (msec). This value exceeded 3 standard deviations from the mean for all normal subjects above the age of 9 months. This absolute value was chosen in order to simplify comparison with results from other studies.

Dispersion was defined as a significant reduction in the amplitude of wave V compared with that of wave I. A I:V amplitude ratio value of 2 or greater was considered abnormal. The I:V value is less than 2 for all normal subjects, and values greater than 2 are seen in subjects with confirmed brain stem pathology. In some cases, wave V was not present and the wave pattern was considered "disrupted." When both delay and dispersion were present, the response was considered dispersed. If one side demonstrated an interwave latency delay and the other side was normal, the responses were considered delayed. If one side showed a dispersion pattern, the responses were considered dispersed regardless of what the other side showed.

TABLE 1
Characteristics of 31 Children With Posterior Fossa Neoplasms

Tumor Type	No. of Patients	Sex (M/F)	Age Range at Diagnosis (yr)
Medulloblastoma	12	9/3	< 1–9
Astrocytoma	6	2/4	< 1–13
Ependymoma	4	3/1	< 1–9
Brain stem glioma	9	5/4	< 4–16

Results

Figure 1 shows representative BAEP waveforms of the normal, delayed, and dispersed patterns. Table 2 shows the distribution of the three types of BAEP waveform in this population of patients. As seen in Table 2, most patients with medulloblastomas presented with a normal study (9/12), and abnormalities, when present, consisted of increased I–V interwave latency. In contrast, no child with ependymoma had a normal study, and both increased interwave latency (delay) and decreased wave V amplitude (dispersion) were seen. Cerebellar astrocytomas resulted in a variety of BAEP pattern changes, while the majority of children with brain stem gliomas displayed a dispersed pattern (7/9), and none had a normal exam.

Discussion

This report describes a study of BAEP as a diagnostic tool in children with posterior fossa tumors.

As is evident from Table 2, posterior fossa tumors seem to have varying degrees of interference with impulse conduction through brain stem pathways. Thus, the least disruptive to BAEP patterns are the medulloblastomas, followed by cerebellar astrocytomas. In our experience, normal BAEP waveforms were seen only in patients who had one of these tumor types.

The two patients with astrocytomas who had dispersed waveforms (I:V ratio greater than 2) had increased intracranial pressure at the time of examination; in one of them who was reexamined after

TABLE 2
Distribution of Types of BAEP Waveform in Patient Population

Tumor Type	No. of Patients	Normal	Delayed*	Dispersed†
Medulloblastoma	12	9	3	0
Brain stem glioma	9	0	2	7
Cerebellar astrocytoma	6	1	3	2‡
Ependymoma	4	0	2	2

*Delayed = I:V interwave latency > 4.6 msec.
†Dispersed = I:V ratio > 2.
‡These patients had markedly increased intracranial pressure at the time of their study.

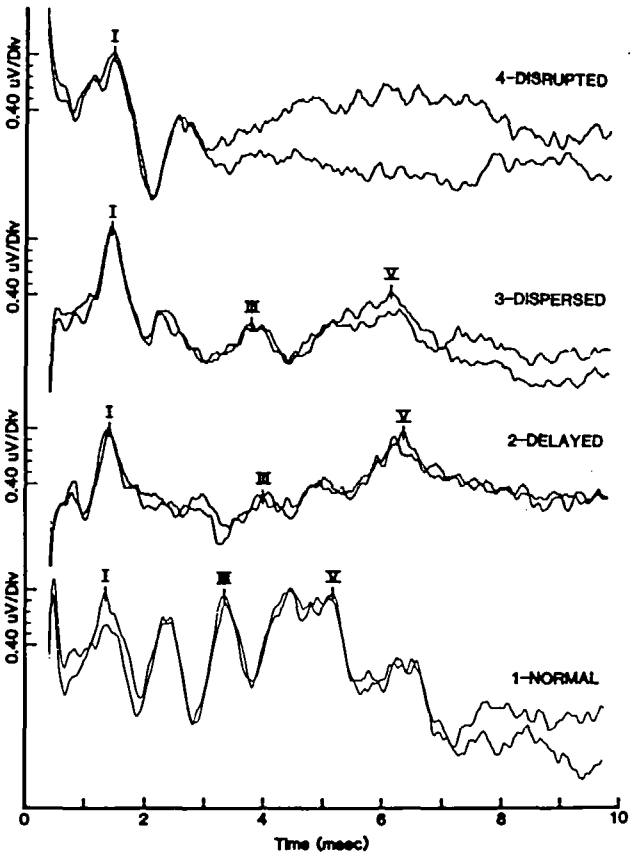

FIGURE 1
Brain stem auditory evoked potential waveforms from tumor patients demonstrating the patterns used for classification. All waveforms are from 1024 sweeps with two overlapping trials, band pass 30 to 3000 Hz, with intensity determined as outlined in the text. Example 1 represents a normal study. Example 2 represents a delay with a I–V interval greater than or equal to 4.6, a value 3 standard deviations beyond the mean for most laboratories. Example 3 represents a wave I to wave V amplitude ratio (I:V) equal to or greater than 2, a value beyond that seen in any normal patient. Example 4 represents a I:V value of infinity due to the absence of an identifiable wave V.

shunting, the BAEP pattern returned to normal. This is compatible with a reversible effect of increased intracranial pressure on conduction through the brain stem.

As expected, brain stem gliomas manifested the most severe disruption of the BAEP waveforms, with delayed conduction and attenuation of later waveforms. In fact, in several of our patients, wave V was not discernible at all.

Interestingly, ependymomas seemed to cause a more severe alteration of BAEP than did medulloblastomas (Table 2). The numbers are small, but of all our patients (studied at presentation or later) we found no child with ependymoma who had a normal BAEP. Because ependymomas and medulloblastomas may present with similar clinical and neuroradiologic findings, a normal BAEP examination is highly suggestive of a medulloblastoma. Conversely, a dispersed waveform pattern most likely denotes an ependymoma.

The electrophysiologic mechanisms underlying the appearance of delayed or dispersed BAEP waveforms are, at present, unknown. Delayed interwave intervals are commonly seen in disorders affecting myelination of the auditory pathways (such as demyelinating and degenerative disorders).[7,8] Symmetric delays suggest diffuse processes such as increased intracranial pressure, irradiation, or drugs.[9] Asymmetric delays may denote a discrete intrinsic lesion, usually on the same side as the stimulated ear.[9]

Dispersion (ie, the loss of amplitude of waveform V) is more difficult to explain. Loss of amplitude is usually considered to indicate axonal damage[10] or a lack of coherence of conduction among nerve fiber bundles. Nunez[11] points out that "far field" transients need to be conducted coherently within nerve fiber bundles to demonstrate a reasonable amplitude. This coherence depends on identical velocity of

conduction toward a specific point, with consequent summation of impulse activity. If, because of such factors as intrinsic damage and loss of myelin, the conducting fibers do not allow for identical conduction velocity, summation is less efficient.[12] The result is a "smear effect" with blunting and dispersion of the peak and a loss in amplitude. Such dispersion has been seen in patients who have intrinsic lesions of the brain stem that include infarcts, tumors, hemorrhage, and degenerative or demyelinating disorders.[4,7–9,12,13]

Thus, dispersion (decrease or loss of waveform V) may imply a more profound intrinsic disruption of impulse conduction through the brain stem than does delay (increased interwave latency) of BAEP waveforms.

Summary

This study presents data on brain stem-evoked potentials from a series of 31 children who were found to have posterior fossa tumors. We demonstrated a tendency for the various tumors to affect differentially neural pathways in the posterior fossa. BAEP may be a helpful adjunct to neuroradiologic studies in the diagnosis of posterior fossa tumors in children.

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