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Auditory time-intensity cues in the binaural interaction component of the auditory evoked potentials

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Abstract

Binaural interaction in the brainstem and middle latency auditory evoked potentials to intensity (dI) and timing differences (dT) between the two ears was studied in 10 normal hearing young adults. A component reflecting binaural interaction in the brainstem potentials occurred at approximately 7 ms and was of largest amplitude when dI and dT were 0. The latency of the binaural interaction component gradually shifted and its amplitude decreased as dI or dT increased and binaural interaction became undetectable when $dI = 16$ dB or when $dT \geq 1.6$ ms. In the middle latency potentials binaural interaction components peaking at 20, 32, and 45 ms were defined that were also largest when dI and $dT = 0$. The latency of the interaction did not shift with changes in dT and dI whereas the amplitude gradually decreased but binaural interaction components were still evident even at the largest values of dI (30 dB) and dT (3 ms). Psychophysical judgments of binaural perceptions showed binaural fusion of the stimuli to persist with dT values up to 1.6 ms and that lateralization of the intracranial image was complete when either $dT = 1.6$ ms or when $dI = 16$ dB. The results suggest that the presence of a binaural interaction component of auditory brainstem potentials correlates with the fusion of binaural click stimuli and the amplitude of the binaural interaction component correlates inversely with the degree of lateralization of the intracranial image. Binaural interaction components of middle latency potentials persist and continue to change even after the binaural stimuli cannot be fused.

Keywords: Binaural interaction; Evoked potential; Brainstem auditory evoked potential; Middle latency auditory evoked potential; Binaural fusion; Lateralization

1. Introduction

Binaural interaction (BI) in the auditory evoked potentials may be defined as the difference between the algebraic sum of the monaural evoked potentials and the binaurally evoked potential. BI can be demonstrated in brainstem (5–8 ms), middle (20–40 ms) and long-latency (90–200 ms) auditory evoked potentials as a reduction in the amplitude of the binaurally evoked potentials compared to the sum of the monaurally evoked potentials. BI has been demonstrated in studies of both human (Hosford et al., 1979; Dobie and Norton, 1980; Wrege and Starr, 1981; Dobie, 1982; Berlin et al., 1984; McPherson et al., 1989; McPherson and Starr, 1993) and animals (Wernick and Starr, 1968; Dobie and Berlin, 1979, Gardi and Berlin, 1981; Özdamar et al., 1986; Wada and Starr, 1989).

There are several studies that have examined the relationship between perceptual features of binaural signals and the BI components. Furst et al. (1985) found that the first major peak of the BI component to click stimulation occurring at about 7.5 ms was observable only when the binaural signals were perceptually fused and localized intracranially. Thus, the BI component was absent for interaural timing differences greater than 1.2 ms or for interaural loudness differences greater than about 30 dB, values at which Furst and colleagues reported that their subjects no longer fused the images or, if fused, the images were not localized intracranially but were completely lateralized. The authors suggested that whenever the BI component is present, the binaural stimuli are perceived as a single fused image within the head.

Jones and Van der Poel (1990) in normal subjects showed that changes in latency of the BI component were observed for interaural timing differences out to 1.0 ms, with no further latency shift noted beyond 1.0 ms. They did not report a similar response for amplitude changes.

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Table 1
Lateralization (degrees from midline) of the acoustic image for intensity disparities between the two ears ($n = 10$)

dI (dB)	S_1	S_2	S_3	S_4	S_5	S_6	S_7	S_8	S_9	S_{10}	Mean	SD	Yost (1981)
0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	10	0	0	10	10	0	0	0	0	0	3	4.8	NR
2	20	10	10	10	20	0	10	10	20	10	12	6.3	10
4	30	30	40	30	20	30	40	40	30	20	31	7.4	30
8	60	60	50	70	60	70	50	50	60	40	57	9.5	50
16	90	90	90	80	90	90	90	90	90	90	89	3.2	90
32	90	90	90	90	90	90	90	90	80	90	89	3.2	NR

NR: not reported.

They observed that the shift in latency of the BI component was equal to approximately one-half the interaural time difference. In addition they showed that the topography of the BI component and that of wave IV were similar, suggesting their origin to be from similar generator sites, perhaps the lateral lemniscus. The latency shift of the BI component with changes in interaural time differences was attributed to a presynaptic delay line at the level of the superior olivary complex similar to the model of Jeffress and McFadden (1971).

Wrege and Starr (1981) observed that when the interaural timing differences increased from 0 to 500 μs there was a proportional shift in the latency of the BI consistent with the shift in wave IV–V of the brainstem auditory evoked potential. They also reported that when the delays exceeded 500 μs the BI component was no longer identifiable.

Furst et al. (1990) studied localization of binaural click trains in patients with multiple sclerosis as a function of interaural time or intensity differences. The amplitude of the BI component correlated with both interaural loudness differences and interaural timing differences. In both patients who could not utilize interaural time or intensity differences for the discrimination judgments, the BI component was absent. Van der Poel et al. (1988) similarly observed that many MS patients have abnormal thresholds for detecting movement of a binaurally fused intracranial image using interaural timing differences.

The purpose of the present study in normals was to examine the effect of interaural timing differences and interaural intensity differences of binaural clicks on BI in both early- and middle-latency auditory evoked potentials and to relate the findings to subjects' localization of the intracranial binaural image.

2. Methods

Ten normal hearing young adults with no history of neurologic or chronic ear disease were used. Each subject had normal pure tone hearing thresholds, normal middle ear acoustic impedance measurements and a centrally fused image for the acoustic stimulus presented at 70 dBnHL for both intensity (dI) and timing differences (dT) equal to zero (the null condition). Informed consent was obtained from each subject and the investigation was performed in accordance with the principles of the Declaration of Helsinki.

Auditory evoked potentials were recorded between electrodes positioned at Cz (positive) and a non-cephalic electrode positioned over Cvii. A ground electrode was placed at Fpz. Brain potentials were amplified and filtered between 1 and 3000 Hz (3 dB down, 6 dB/octave) for both the brainstem auditory evoked potentials and the middle latency auditory evoked potentials. Rarefaction (100 μs) acoustic clicks were presented at 11.1/s at an intensity

Table 2
Lateralization (degrees from midline) of the acoustic image for timing disparities between the two ears ($n = 10$)

dT (ms)	S_1	S_2	S_3	S_4	S_5	S_6	S_7	S_8	S_9	S_{10}	Mean	SD	Yost (1981)
0	0	0	0	0	0	0	0	0	0	0	0	0	0
0.05	10	10	0	20	10	0	0	10	10	0	7	6.7	NR
0.10	40	30	40	40	40	30	20	30	40	10	32	10.3	30
0.20	60	60	50	60	60	70	50	60	60	50	58	6.3	60
0.40	70	60	70	80	70	80	70	70	70	70	71	5.7	70
0.80	80	70	80	80	80	80	80	70	80	80	78	4.2	80
1.60	90	80	90	80	90	90	90	80	90	90	87	7.8	90
3.20	90	90	90	80	90	90	90	90	80	90	88	4.2	NR

NR: not reported.

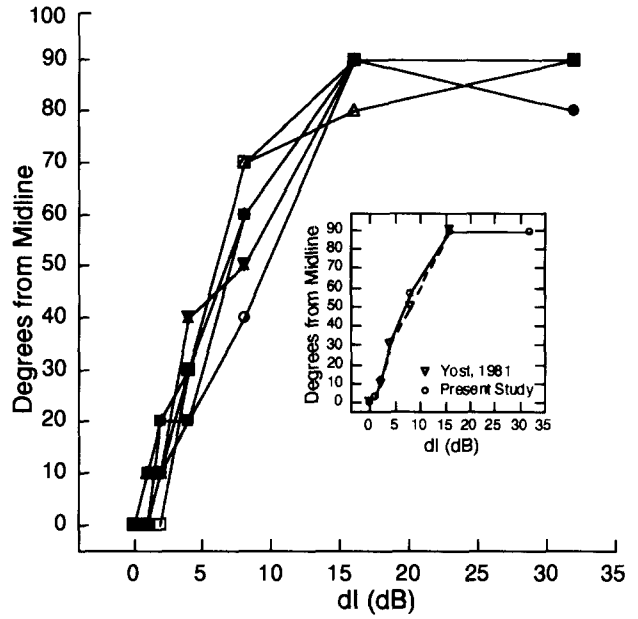

Fig. 1. Degrees of lateralization from midline as a function of interaural intensity differences ($n = 10$). Inset shows the mean from our study plotted along with data from Yost (1981).

level of 70 dBnHL. The ear contralateral to stimulation was masked with broadband noise at 35 dBHL for monaural recordings. Although this may activate the efferent pathway our results do not appear to be influenced by this since they are in agreement (for $dI = 0$ and $dT = 0$) with previous studies (McPherson and Starr, 1994). In our laboratory this intensity is approximately 12 dB below the threshold of the acoustic middle ear reflex for click stimulation. Both contralateral masking and insert earphones were used to reduce acoustic crossover. A 10 ms sample was obtained for the brainstem auditory evoked potentials and a 100 ms sample for the middle latency auditory evoked potentials. Two samples consisting of 2000 trials each for the brainstem auditory evoked potentials and 1000 trials each for the middle latency auditory evoked potentials were obtained for right monaural, left monaural and binaural click presentation for each stimulus condition.

Table 3
Equivalent dI, dT and lateralization values (mean of 10 subjects)

Degrees from midline	dI (dB)	dI (ms)
0	0	0
10	2	0.05
30	4	0.10
50	8	0.20
90	16	1.6

The stimulus sequence consisted of random presentations of interaural intensity differences of 0, 1, 2, 4, 8, 16 and 32 dB; and for interaural time differences of 0, 0.05, 0.10, 0.20, 0.40, 0.80, 1.6 and 3.2 ms. The ear of presentation and the type of evoked potential (auditory brainstem, middle latency) were randomly selected. The recording session was 4 h with a rest period after the second hour.

The amplitude at 0.9 ms served as the baseline for making amplitude measures of the peaks. This was done for the brainstem components, the middle latency components N20, P30, N40 and P60, and the BI components.

The insert earphones were switched between ears according to a random number assigned each subject. In addition, calibration for intensity and phase were com-

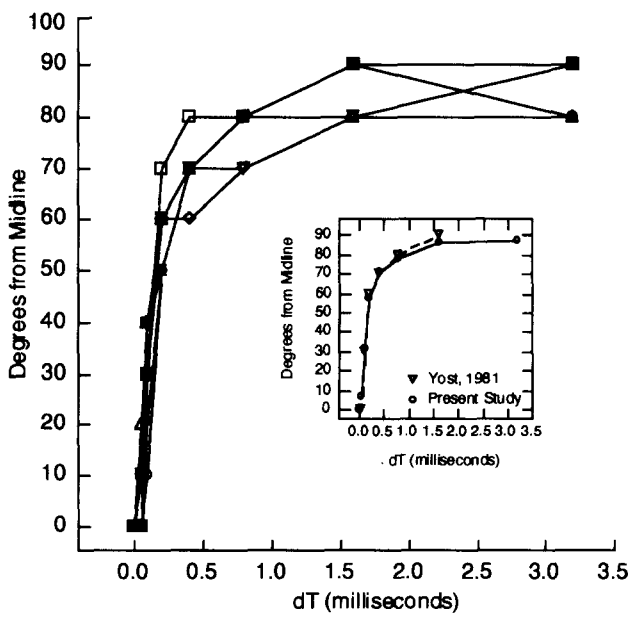

Fig. 2. Degrees of lateralization from midline as a function of interaural timing differences ($n = 10$). Inset shows the mean from our study plotted along with data from Yost (1981).

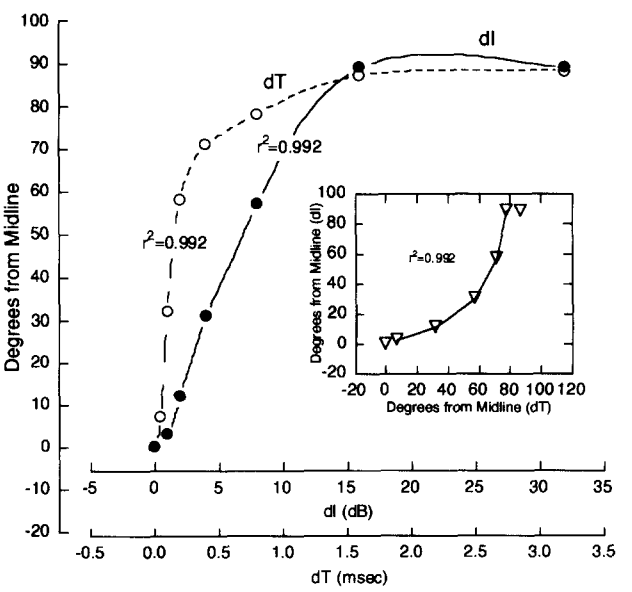

Fig. 3. Curve-fit for the acoustic image for interaural intensity and timing differences.

pleted before, during and following the study and no significant differences were found.

Prior to the electrophysiological testing each subject was shown a head of a mannequin and, using a random presentation of stimuli, asked to indicate by touching a point along a line traversing the cranium and joining the ears the locus of the perceived image. For each value of dT the subjects were asked to indicate whether they heard one or two clicks. A grid was used and the sites selected converted into degrees lateral to the midline. This procedure was repeated twice and the mean of the two trials are reported.

The monaural evoked potentials were digitally added to obtain the sum of the monaural potentials (e.g., right ear + left ear). The BI component was determined by subtracting the binaurally evoked waveforms from the sum of the monaural waveforms. Grand average waveforms were constructed by averaging across all subjects for each condition.

The latencies of the brainstem and middle latency auditory evoked potentials were defined for each subject according to their polarities and latencies. Since the insert earphones created a 0.9 ms delay from stimulus onset to stimulation at the tympanic membrane this value was subtracted from each of the latency values. The 0.9 ms delay served as the baseline point of reference. Peak-to-following trough amplitudes were measured for waves I, II, III, IV and V. Baseline-to-peak measurements were made for the amplitudes of N10, N20, P30, N40 and P60. The amplitude of the BI was measured from baseline-to-peak. The duration (i.e., width) of the BI component was measured from where the component first intersected the baseline to where the component intersected the baseline on the distal side of the response.

Means and standard deviations were used to describe the results. Correlation analysis was used to describe the correspondence between the psychoacoustic measures and the measures of the BI components. Correlation coefficients that did not pass the Kolmogorov-Smirnov test for normality were not considered to be drawn from a normal distribution of the population and hence not used even if they were found to be statistically significant.

3. Results

3.1. Psychoacoustic measures

Tables 1 and 2 show the data for the lateralization of the acoustic image for intensity and time disparities between the two ears. On the extreme right of each table are results from an experiment by Yost (1981) on lateralization of continuous sinusoids. These results are illustrated in Figs. 1 and 2.

Fig. 1 shows that complete lateralization occurs with an intensity difference (dI) of 16 dB and greater. Fig. 2 shows that complete lateralization occurs with a time difference (dT) between the two ears of 1.6 ms and greater. Fusion of the two clicks into a single image persisted up to and including dT values of 1.6 ms. At 3.2 ms fusion was lost and two clicks were heard which lateralized to the ear receiving the initial stimulus (i.e.,

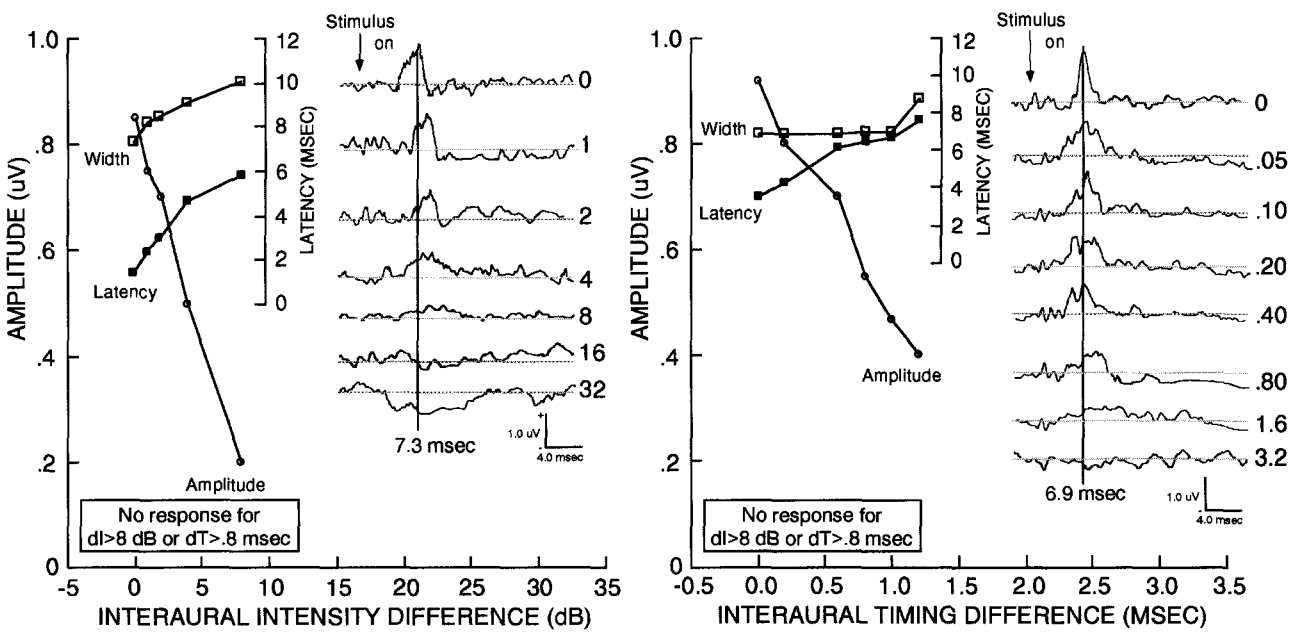

Fig. 4. Interaural intensity and timing differences in the BI component of the brainstem auditory evoked potentials (grand average).

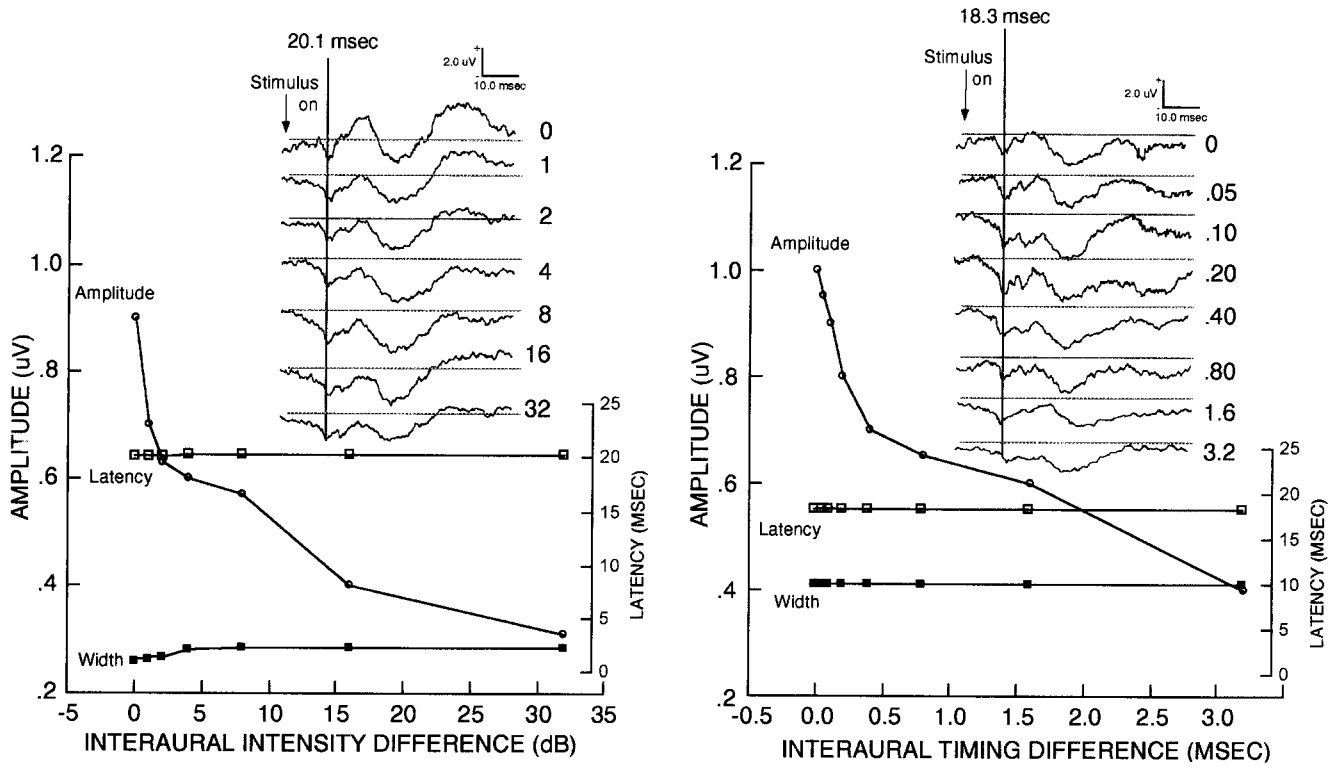

Fig. 5. Interaural intensity and timing differences occurring at 20 ms in the BI component of the middle latency auditory evoked potentials (grand average).

leading ear). The insets to Figs. 1 and 2 show the mean dI and dT plotted alongside the results obtained from Yost (1981). Even though the methods were somewhat different, there is relatively good agreement of both interaural timing differences and interaural loudness differences on lateralization between the two studies.

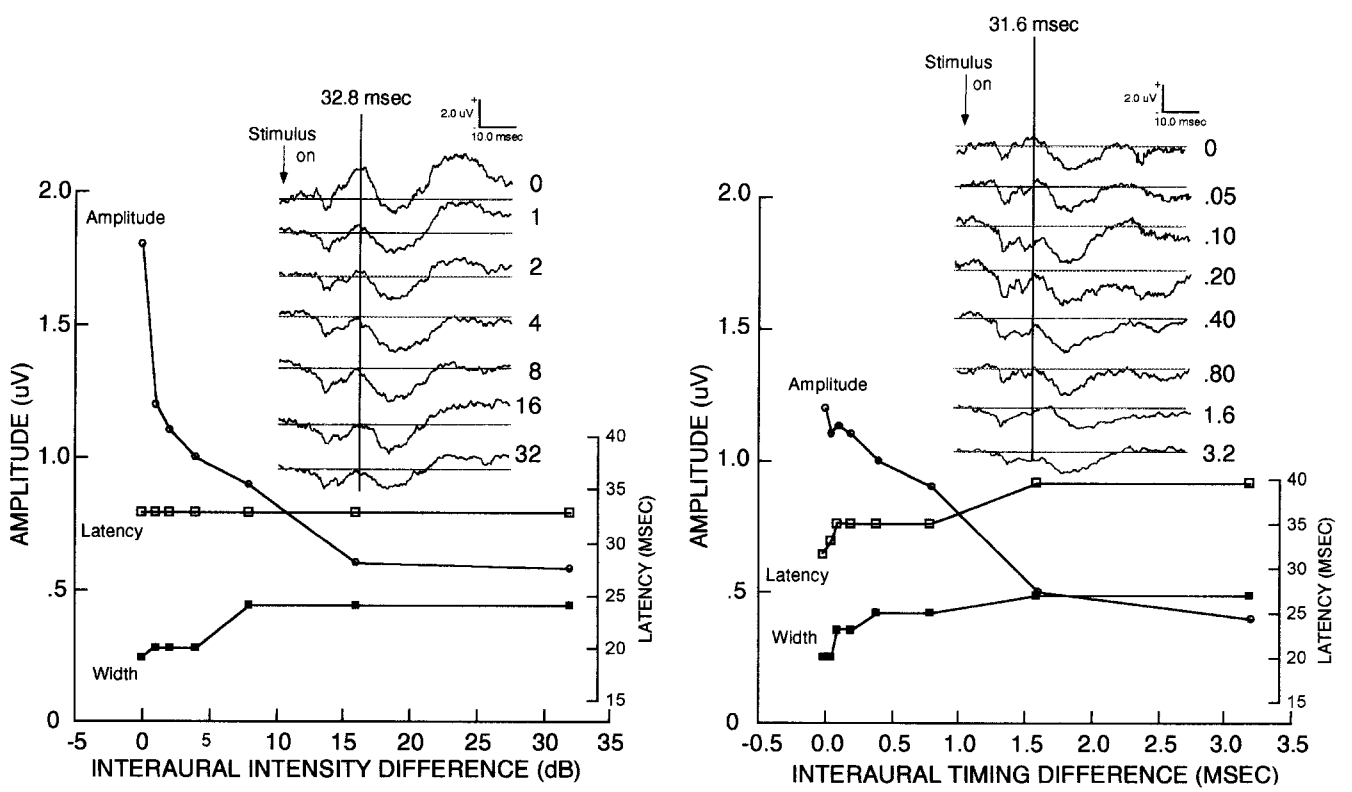

Fig. 6. Interaural intensity and timing differences occurring at 30 ms in the BI component of the middle latency auditory evoked potentials (grand average).

Table 3 shows the values of dI and dT that demonstrate correspondence with equal degrees of lateralization. The lines of best fit for these data are shown in Fig. 3 and illustrate the non-linearity of image lateralization accompanying intensity and timing disparities between the two ears.

3.2. Binaural interaction in the brainstem auditory evoked potential

Fig. 4 shows the changes in the BI component of the brainstem auditory evoked potential to interaural intensity and timing differences. The BI component peaks at approximately 7 ms and is of maximal amplitude for dI = 0 dB and dT = 0 ms. The insets in Fig. 4 illustrate the BI component to have a gradual broadening in duration, decrease in amplitude, and prolongation in peak latency as dI or dT increase. BI is not present at or above dI = 16 dB, the same value when there is complete lateralization of the stimulus (see Fig. 1). BI is also undetectable when dT = 1.6 ms, the value when fusion of the binaural signals is lost. The effects of changes of dT on the latency of the BI component are less than with dI (see Fig. 2).

3.3. Binaural interaction in the middle-latency auditory evoked potential

BI components for middle latency potentials occurred at 20.1 ms, 32.8 ms, and 45.9 ms. The change in their amplitude latency and duration are graphed accordingly in Figs. 5–7. The amplitude of the components gradually decrease without major changes in latency or in duration (see insets). Even at extreme separation of time (dT = 3 ms) or intensity (dI = 30 dB), when complete lateralization of the acoustic image has occurred, the BI components are still present.

Table 4
Correlation probabilities for correspondence between psychoacoustic measures and measures of the binaural interaction component

	ABR	N20	P30	N40
dI				
Amplitude	0.001	0.022	0.037	0.044
Latency	0.011	0.035	0.99 *	ND
Width	0.005	0.009	0.034	0.249
dT				
Amplitude	0.001	0.001	0.023	0.009
Latency	0.070 *	0.008 *	0.008 *	ND
Width	0.001	0.153 *	0.001 *	0.009

* Failure to pass the Kolmogorov-Smirnov normality test.

3.4. Correlations between stimulus parameters and binaural interaction components

Changes in dI and dT for amplitude show significant ($P < 0.05$) linear correlations between the psychophysical measures of dI and dT and the corresponding electrophys-

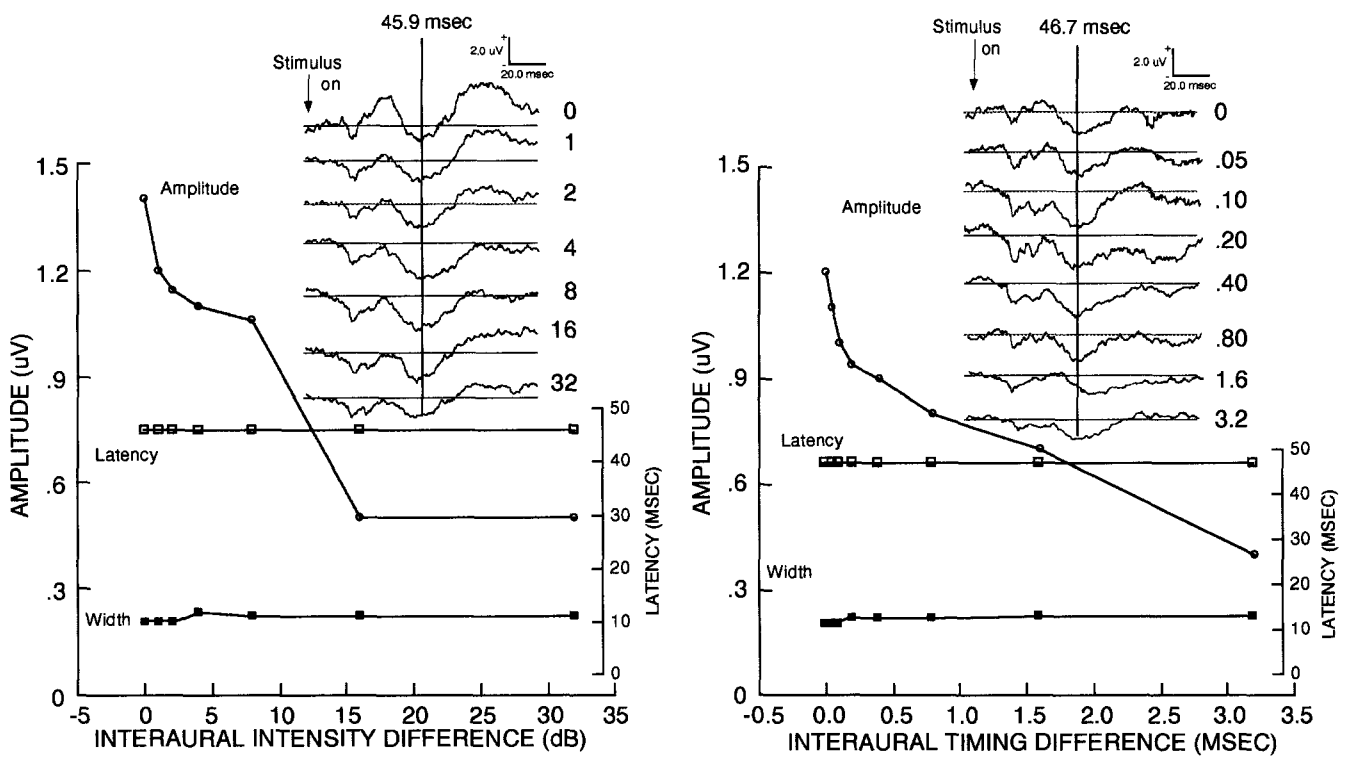

Fig. 7. Interaural intensity and timing differences occurring at 40 ms in the BI component of the middle latency auditory evoked potentials (grand average).

iological measures of BI (Table 4). For the brainstem auditory evoked potential, amplitude and width of BI components were correlated with stimulus parameters for both dI and dT. The change in BI latency correlated with stimulus parameters only for dI. For the middle latency BI components the correlations with stimulus parameters using dI was significant for all but one instance (latency of the interaction component at 30 ms). For dT significant correlations with amplitude were seen for all three subcomponents (i.e., 20, 30 and 45 ms) in the middle latency auditory evoked potentials. Only one of the remaining correlations for latency and width of the BI components were significant for dT at 45 ms in the middle latency auditory evoked potentials.

4. Discussion

The results of this study demonstrate a significant ($P \leq 0.05$) linear correlation between the changes in amplitude of binaural components of auditory brainstem and middle latency potentials and changes in the locus of the intracranial fused image accompanying interaural time and intensity differences. BI for the middle latency components are still evident even with interaural time disparities sufficiently large to disrupt binaural fusion. These results suggest that the amplitude of BI components reflect neural processes contributing to localization of the fused image, similar to the proposals of Furst et al. (1985) and Jones and Van der Poel (1990). However our data extends their

Table 5
Means and standard deviations in the binaural interaction component for amplitude (μV) and latency (ms) changes for dI

	dI (dB)						
	0	1	2	4	8	16	32
ABR							
Amplitude (μV)							
Mean	0.85	0.75	0.70	0.50	0.20	-	-
SD	0.26	0.29	0.30	0.27	0.36	-	-
Latency (ms)							
Mean	7.3	8.2	8.5	9.1	10.1	-	-
SD	0.61	0.60	0.69	0.81	0.79	-	-
Width (ms)							
Mean	1.4	2.3	3.0	4.7	5.8	-	-
SD	0.47	0.53	0.52	0.61	0.92	-	-
N20							
Amplitude (μV)							
Mean	0.91	0.73	0.63	0.60	0.57	0.42	0.31
SD	0.19	0.27	0.29	0.41	0.37	0.44	0.39
Latency (ms)							
Mean	20.1	20.1	20.1	20.2	20.2	20.2	20.2
SD	2.74	2.63	2.85	2.92	2.93	2.87	3.4
Width (ms)							
Mean	1.3	1.2	1.4	2.0	2.2	2.2	2.3
SD	0.51	0.51	0.59	0.63	0.64	0.68	0.66
P30							
Amplitude (μV)							
Mean	1.8	1.2	1.1	1.0	0.92	0.63	0.58
SD	0.59	0.61	0.68	0.66	0.73	0.69	0.70
Latency (ms)							
Mean	32.8	32.8	32.8	32.8	32.8	32.8	32.8
SD	3.3	3.0	3.4	3.7	3.9	4.0	4.0
Width (ms)							
Mean	1.9	2.0	2.0	2.4	2.4	2.4	2.4
SD	0.67	0.65	0.69	0.67	0.69	0.72	07.7
N40							
Amplitude (μV)							
Mean	1.9	1.2	1.1	1.1	1.0	0.57	0.52
SD	0.39	0.37	0.39	0.41	0.47	0.43	0.51
Latency (ms)							
Mean	46.0	46.0	46.0	46.0	46.0	46.0	46.0
SD	4.1	3.8	4.5	4.8	4.9	4.9	5.6
Width (ms)							
Mean	1.0	1.0	1.0	1.2	1.1	1.1	1.1
SD	0.97	1.2	1.1	1.5	1.3	1.3	1.2

observations and further quantifies the relationship between the BI, lateralization and binaural disparities in dI and dT.

Our psychoacoustic data indicate that complete lateralization (90°) occurs when there is a 16 dB or greater interaural intensity difference or when there is an interaural temporal difference of 1.6 ms or greater. These values are similar to those of Yost (1981). Mills (1960), Yost (1981) and the present study found that a 2 dB interaural intensity differences was needed for an observer to detect the movement of an image from midline (Table 3 and Fig. 1). When the interaural temporal differences is equal to or exceeds 2 ms the percept is no longer fused but becomes two distinct images (Yost and Nielsen, 1985). Irrespective as to which ear (i.e., left or right) was the leading ear, no differences were seen in the amount of disparity differences for either interaural intensity or interaural time differences. However, we did see a reduction in the variability of the amplitude of the potentials (Tables 5 and 6) when intensity differences were greater than 8 dB and when timing differences were greater than 0.8 ms.

The BI component for the auditory brainstem potentials shows a sharp well defined peak for interaural intensity differences from 0 to 2 dB. For differences greater than 2 dB there is a broadening of the component and a reduction in amplitude such that the component cannot be defined when $\Delta I = 16$ dB. In contrast, the middle latency BI component which also show a graded decrease in amplitude are still clear for interaural differences even as large as 32 dB. The difference in amplitude functions of the BI

Table 6
Means and standard deviations in the binaural interaction component for amplitude (μV) and latency (ms) changes for dT

	dT (ms)							
	0	0.05	0.10	0.20	0.40	0.80	1.6	3.2
ABR								
Amplitude (μV)								
Mean	0.92	0.80	0.73	0.55	0.47	0.39	-	-
SD	0.29	0.27	0.29	0.32	0.39	0.41	-	-
Latency (ms)								
Mean	6.9	6.9	6.9	7.0	7.0	8.8	-	-
SD	0.58	0.63	0.61	0.66	0.71	0.69	-	-
Width (ms)								
Mean	3.5	4.3	6.2	6.5	6.7	7.6	-	-
SD	0.44	0.49	0.53	0.51	0.55	0.83	-	-
N20								
Amplitude (μV)								
Mean	1.1	0.95	0.89	0.82	0.69	0.65	0.61	0.38
SD	0.17	0.23	0.27	0.31	2.8	0.33	0.39	0.37
Latency (ms)								
Mean	18.3	18.3	18.3	18.3	18.3	18.3	18.3	18.3
SD	2.91	2.7	3.0	2.9	3.1	3.3	3.5	3.3
Width (ms)								
Mean	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	0.49	0.53	0.51	0.60	0.58	0.67	0.66	0.71
P30								
Amplitude (μV)								
Mean	1.2	1.1	1.1	1.1	1.0	9.2	4.9	3.7
SD	0.62	0.66	0.67	0.71	0.69	0.74	0.71	0.73
Latency (ms)								
Mean	31.6	33.0	35.0	35.0	35.0	35.0	39.5	39.5
SD	3.4	3.3	3.5	3.8	3.5	4.1	4.0	4.3
Width (ms)								
Mean	2.0	2.0	2.3	2.3	2.5	2.5	2.7	2.7
SD	0.65	0.66	0.69	0.71	0.68	0.73	0.75	0.78
N40								
Amplitude (μV)								
Mean	11.0	11.0	11.3	11.0	10.0	9.0	5.0	4.0
SD	0.37	0.39	0.44	0.42	0.48	0.49	0.51	0.51
Latency (ms)								
Mean	47.0	47.0	47.0	47.0	47.0	47.0	47.0	47.0
SD	4.2	4.1	4.2	4.5	4.8	5.0	4.9	5.4
Width (ms)								
Mean	2.2	2.2	2.2	2.3	2.3	2.3	2.5	2.5
SD	1.1	1.1	0.99	1.3	1.2	1.5	1.3	1.5

components between the brainstem and middle latency potentials may reflect the presence of additional binaural processes rostral to the brainstem with characteristics different than those of the brainstem. Alternatively, the increase in amplitude of the middle latency components may be a passive reflection of the increase in number of neurons in the auditory pathway in the mesencephalic and diencephalic auditory nuclei amplifying the brainstem outputs.

The peak latency of approximately 7 ms of the major BI component in the brainstem potentials is consistent with the interpretation that binaural processing is initiated in the brainstem (Moushegian et al., 1972; Starr and Hamilton, 1976; Møller et al., 1981; Møller et al., 1981; Wrege and Starr, 1981; McPherson et al., 1989,and Starr, 1993). We would argue that the loss of definition in the BI component at $dI = 16$ dB and $dT = 1.6$ ms, values associated with complete lateralization (dI) and with the loss of fusion (dT) is compelling evidence for linking neural processes generating the brainstem BI component with binaural perceptions.

The medial superior olivary complex is the first nucleus on the ascending auditory pathway to receive bilateral inputs and thus represent binaural auditory processing (Moushegian et al., 1975). It has been shown that when the crossing fibers of the trapezoid body are lesioned in the brainstem there is a loss of BI in the brainstem potentials (Buchwald and Huang, 1975; Fullerton and Hosford, 1979; Gardi and Berlin, 1981; Levine et al., 1993). Moreover, lesion studies in animals have shown such lesions to be accompanied by the inability of the animals to use binaural cues, particularly interaural time disparities, for behavioral judgments (Diamond and Neff, 1957; Diamond et al., 1962).

Wada and Starr (1989) found that when the trapezoid body was sectioned in the guinea pig that the later P4 and N4 (proposed to have similar generators as waves V–VI in human brainstem potentials components) were lost and that the decrease in the BI component was related to the number of fibers sectioned during the experiment. Likewise complete sectioning of the medial superior olivary produced no binaural interaction at P4 and N4. Little or no effect in the BI component was seen after unilateral destruction of the lateral lemniscus or the inferior colliculus. However a complete loss of the BI component for N4 occurred for bilateral destruction of the lateral lemniscus.

Two types of binaural neurons in the superior olivary nucleus have been identified: excitatory-excitatory neurons (EE), primarily found in the medial superior olivary nucleus, excited by stimulus received from both ears, and excitatory-inhibitory neurons (EI), primarily found in the lateral superior olivary nucleus, that are excited by input from one ear and inhibited by input from the other ear (Galambos et al., 1959; Irving and Harrison, 1967; Moushegian et al., 1985). According to Caird and Klinke (1983) the medial superior olive cells are sensitive to interaural time differences while the lateral superior olive cells are sensitive to both interaural time differences and interaural intensity differences. Levine et al. (1993) suggests that dT is abnormal in lesions affecting the medial superior olivary bodies, and dI is abnormal in lesions affecting the ventral acoustic stria. The graded decrease in the amplitude of the BI component of the brainstem potentials with increasing dI and dT may reflect the well-known changes in activity of the EI cells to changes in stimulus features of binaural signals.

Jones and Van der Poel (1990) observed that as dT was increased from 0 to 0.8 ms, the latency of the BI component of the brainstem potentials increased by approximately half the interaural time difference, without an effect on the duration of the component. They concluded that the brainstem binaural component may reflect (1) sound localization mechanisms sensitive to dT; and (2) the output of binaurally responsive neurons, probably in the superior olivary complex, which are responsive to a particular dT according to the relative length of presynaptic axons relaying inputs from either ear. The present observation that the amplitude of the BI component is graded with changes in dI and dT is consistent with the Gaumond and Psaltikidou (1991) model of BI, suggesting that the binaural difference is generated by differing levels of output of EI cells.

The middle latency portion of the BI component showed a graded decrease in amplitude throughout the range of interaural intensity and timing differences tested, persisting at values when the brainstem interaction components were lost ($dI = 16$ dB and $dT = 3.2$ ms). It would appear that the generators for the BI components of the middle latency potentials represent either additional processing of the brainstem binaural outputs or independent mesencephalic/diencephalic binaural processes.

It appears evident then that the presence of an auditory brainstem BI component correlates with perceptual fusion of binaural signals and features of the interaction component can be significantly related to lateralization of the fused image. We recently reported a patient with bilateral auditory nerve timing dysfunction (Starr et al., 1991) who was unable to fuse binaural signals. Van der Poel et al. (1989) reported two patients with multiple sclerosis who were similarly unable to perform an interaural timing difference task because of a failure of binaural fusion. Thus, bilateral lesion of the auditory pathway at the level of the VIIIth nerve and/or brainstem can be accompanied by a disorder of both fusion and lateralization.

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