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Binaural Interaction in Human Auditory Brainstem Evoked Potentials

Kathy S. Wrege, MD, Arnold Starr, MD

• **Binaural interaction was examined by recording human auditory brainstem responses to clicks from scalp electrodes. Deviations of binaurally evoked responses from the sum of monaurally evoked potentials were observed during waves IV through VI. Amplitude and latency of the interactions depended on click polarity: condensation clicks produced interactions of larger magnitude and longer latency than did rarefaction clicks. Latency differences cannot be accounted for by small latency shifts of the components of monaurally or binaurally evoked potentials resulting from changes in click polarity. Binaural interaction amplitude decreased as click intensity decreased and interaural delay increased. Attenuation of binaural interaction with interaural time differences was maximal at an interaural delay of 900 μs. Latency of interaction was prolonged in one subject with low- and high-frequency hearing loss; latency of binaural interaction in subjects with only high-frequency hearing loss was normal. These results suggest that binaural interaction in these potentials reflects binaural processing of low-frequency acoustic stimulation.**

(*Arch Neurol* 1981;38:572-580)

Auditory brainstem responses (ABRs) are far-field reflections of activity originating in brainstem portions of the auditory pathway. Several variables that affect this response have been studied in human beings: (1) subject factors, such as age, sex, and body temperature; (2) recording factors, such as electrode location and response filtering; and (3) stimulus factors, such as polarity, rate, and acoustic spectrum.[1-7]

In addition, recent studies with human beings,[8,9] guinea pigs,[10] and other species[11] showed that certain components of the ABR evoked by binaural stimuli differ from the sum of the monaurally evoked responses. Jewett[12] first described this phenomenon in cats and defined the difference between the binaural ABR and the sum of the monaural ABRs as the binaural interaction component. Binaural interaction may be important as an electrophysiological index of binaural neural processes.

In the present study, we investigated binaural interaction in human ABRs and defined some of the stimulus and recording factors necessary for its detection. Emphasis was placed on examining sensitivity of the binaural interaction components to stimulus polarity. Ornitz and Walter[4] and Stockard et al[7] reported that the latency and morphologic features of the human ABR components vary with the initial phase of the acoustic stimulus. They observed phase sensitivity in components IV through VI; which are precisely those that exhibit binaural interaction in the human ABR.[8,9]

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METHODS

Eleven subjects aged 24 to 35 years participated in the experiment. Eight subjects had normal pure tone audiograms, two had high-frequency hearing loss, and one had both a high- and low-frequency hearing loss with normal thresholds in the midfrequency range. Metal disk electrodes were attached to the scalp at vertex (active), neck (CII; reference), and right mastoid (ground). Interelectrode resistance was below 4 kΩ. Subjects reclined on a bed in a double-wall sound-attenuating room throughout the testing session.

The acoustic stimuli were generated by delivering 100-μs pulses to earphones (TDH-39). The acoustic signal measured in a 6-cc coupler is shown in Fig 1. Click polarity was reversed by inverting the electrical pulse to the attenuators. Summation of the condensation and rarefaction acoustic waveforms resulted in a flat line, indicating that the output of the earphones was symmetrical in the intensity regions examined. Clicks were presented at intervals of 39 ms and at an intensity of 70 dB sensation level (SL) determined for each normal subject. This SL differed by less than 10 dB across normal-hearing subjects. For each subject, thresholds to condensation and rarefaction clicks were found to differ by less than 3 dB. The two subjects with high-frequency hearing loss had thresholds 8 to 10 dB higher than normal subjects. However, clicks were presented at 70 dB normal hearing level for these two subjects. Thresholds for the subject with both high- and low-frequency hearing loss were within the normal range.

The scalp-derived potentials were filtered (100 Hz to 3 kHz) and amplified by a factor of 100,000. A digital computer was used to average the evoked activity at a sample rate of 16.67 kHz for 20.48 ms after stimulus onset (40 μs per point, 512 points). An artifact rejection routine was used to reduce the inclusion of high-amplitude muscle potentials in the averaged responses. The responses to 2,000 click presentations were averaged for each of three conditions: right monaural, left monaural,

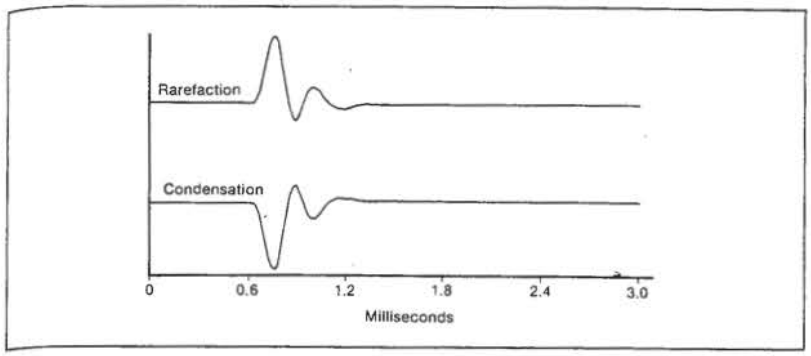


Fig 1.—Acoustic waveform of click stimulus. Clicks were generated by delivering 100-μs pulses to earphones (TDH-39). Acoustic waveforms were obtained from earphones measured in 6-cc coupler (B&K 4152) with microphone (B&K 4144) coupled to sound-level meter (B&K 2209).

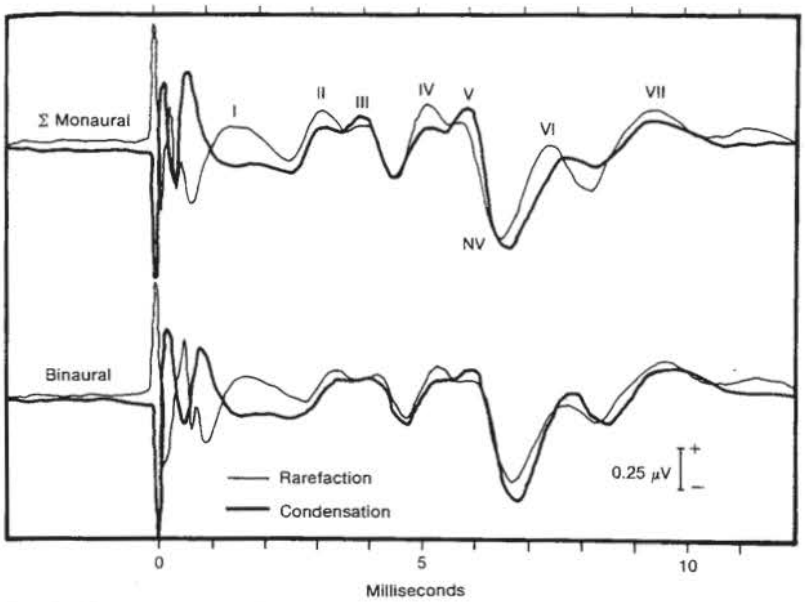
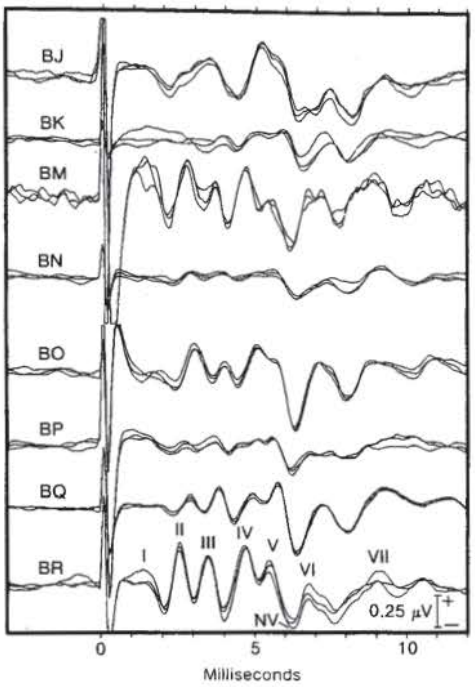


Fig 3.—Composite waveforms of sum of left and right monaurally evoked responses (Σ monaural) and binaurally evoked responses to clicks presented at 70 dB sensation level. Waveforms were constructed by averaging together evoked responses of eight normal-hearing subjects. Note that evoked potential waveform is dependent on click polarity. See text for further description.

Fig 2.—Left monaurally evoked responses from subjects with normal pure-tone audiograms. Each trace is average of responses to 2,000 rarefaction clicks presented at 70 dB sensation level. Note that wave I is of low amplitude in recording montage used (Cz to CII).



and binaural. The sequence was then repeated five more times to present 12,000 click trials for each condition. The polarity of both clicks was then reversed, and the entire sequence was repeated. To evaluate the reliability of the evoked potentials, five of the 11 subjects were retested after several weeks.

For each condition the six replications of 2,000 stimulus trials were averaged. Binaural interaction was then determined by subtracting the binaurally evoked potentials from the sum of the monaurally evoked potentials ([left monaural + right monaural] − binaural). Measurements of latency and peak-to-trough amplitudes of waves IV through VI of the evoked potentials and of the binaural interaction waveform were made. As will be shown later, the two dominant peaks in binaural interaction always occurred in association with the IV-V complex and wave VI. Therefore, the amplitude of binaural interaction was calculated as a percentage of the peak-to-trough amplitude of the IV-V complex or wave VI of the summed monaural waveforms. This relative measure of binaural interaction amplitude was used to adjust for individual differences in the amplitude of evoked activity. A repeated-measures analysis of variance was used for all statistical tests.

The effect of click intensity was evaluated in four of the subjects with normal hearing. The procedures were repeated at click intensities of 60 and 50 dB SL. Finally, the influence of interaural delay was evaluated in two subjects. Rarefaction clicks were presented at an intensity of 70 dB SL and the onset of clicks presented to the right ear was systematically delayed relative to those presented to the left ear. Binaural interaction was determined by the following procedure: (1) the right monaural response was delayed by the same amount as the interaural time difference; (2) the artificially delayed right monaural response was added to the left monaural response, producing the summed monaural response, and (3) the binaural response was subtracted from the summed monaural response, producing the binaural interaction waveform. Binaural interaction was determined for interaural delays of 0, 50, 200, 500, 900, 1,400 and 2,000 μs.

Informed consent was obtained from all subjects after the experimental procedures had been fully explained.

RESULTS

Subjects With Normal Audiograms

Both the morphologic features and amplitude of auditory brainstem potentials varied between individuals. Figure 2 illustrates the averaged left monaurally evoked response to rarefaction clicks presented at 70 dB SL for each of the subjects with a normal audiogram. The individual components of the brainstem potentials have been labeled above the responses of subject BR. For each subject, three replications have been superimposed. The variability in the latency of wave V across subjects (SD = 0.15 ms) may be due to differences in their ability to determine hearing threshold, since the latency of wave V was well correlated (r = .61) with the absolute settings of the attenuators.

To examine the influence of stimulus polarity on the evoked potentials, the responses of each subject were adjusted so that the latency of peak V was the same. A composite waveform

was then constructed by averaging the potentials across individuals. Composite waveforms were constructed for left monaural, right monaural, and binaural potentials.

The morphologic features of the composite auditory brainstem potentials varied with click polarity (Fig 3). In both the monaural and binaural conditions, the peak-to-trough amplitude of wave IV was larger to rarefaction than condensation clicks (7/8 subjects), whereas wave V was larger to condensation than to rarefaction clicks (6/8 subjects). In the monaural condition wave VI (peak-to-trough) was larger to rarefaction clicks (6/8 subjects), whereas in the binaural condition wave VI was larger to condensation clicks (6/8 subjects). The latency of both the negative trough following wave V (designated NV) and wave VI was greater to condensation than rarefaction clicks. The difference in latency was greater in the monaural condition (0.23 ± 0.10 ms) than in the binaural condition (0.09 ± 0.08 ms) for each subject. Thus, the dependence of the amplitude of waves IV and V on stimulus polarity was relatively independent of whether a monaural or binaural stimulus was presented, whereas the amplitude and latency of wave VI was dependent on both polarity and the mode of stimulus presentation (monaural vs binaural condition). The changes described for the composite auditory brainstem potentials were evident in the individual evoked potentials, as shown in the example in Fig 4.

Binaural interaction was assessed by subtracting the binaurally evoked potentials from the sum of the monaurally evoked potentials (Fig 5). An upward deflection occurred when the sum of the monaurally evoked potentials was greater than the binaurally evoked potential. The waveform of binaural interaction was bimodal; the first peak (peak A) was invariably associated with the IV-V complex and NV, and the second (peak B) occurred about 2 ms later, associated with wave VI or the trough following wave VI. Across subjects, the amplitude of the interaction at 70 dB SL was independent of absolute stimulus intensity. Short-latency binaural interactions were observed in six of eight subjects, but these interactions were (1) small and variable both within and across subjects, (2) occurred sometimes in response to only one click polarity, and (3) at 70 dB SL the amplitude across subjects was correlated with absolute stimulus intensity. Further analysis was therefore restricted to the interaction associated with the IV-V complex and wave VI. An example of short-latency interaction can be seen between 3 and 4 ms in Fig 5 (to condensation clicks) and in Fig 6 (to rarefaction clicks).

The amplitude and latency of binaural interaction varied with click polarity (Fig 5). The largest peak-to-trough amplitude of the interaction waveform (peak A) was measured and compared to the peak-to-trough amplitude of the IV-V complex in the sum of the monaurally evoked potentials. The amplitude of the interaction reflects both amplitude and latency differences between the sum of the monaurally and binaurally evoked potentials. For example, in Fig 5 peak A in response to rarefaction clicks was primarily due to amplitude differences, whereas in response to condensation clicks the interaction was primarily due to latency differences. However, the contribution of the evoked potential latency or amplitude differences to binaural interaction did not vary systematically with click polarity across individuals. Whether produced by differences in latency or amplitude, peak A was significantly larger ($P < .05$) in response to condensation clicks (37% ± 10% of the sum of the monaural IV-V complex) than to rarefaction clicks (25% ± 8%).

The latency of peak A of the interaction was 0.8 ms greater in response to condensation clicks (6.10 ± 0.40 ms) than in response to rarefaction clicks (5.30 ± 0.62 ms; $P < .001$). This latency difference cannot be attributed to the observed latency shift in the components of the evoked potentials accompanying change in click polarity, which amounted to only 0.15 to 0.35 ms. Thus, the interaction in response to rarefaction clicks preceded wave V by 0.47 ± 0.46 ms, whereas the interaction in response to condensation clicks followed wave V by 0.27 ± 0.47 ms ($P < .001$).

The peak-to-trough amplitude of the second component (peak B) of the binaural interaction waveform was also measured and compared to the peak-to-trough amplitude of wave VI. Although the amplitude of this component was highly variable across subjects, the polarity sensitivity was qualitatively similar to that observed for the first component. Relative to wave VI, the amplitude in the rarefaction condition was 119% ± 143%. In the condensation condition, the amplitude was 258% ± 300% of wave VI amplitude. The latency of this second component in response to rarefaction clicks was 7.0 ± 0.4 ms (wave VI latency: 7.3 ± 0.2 ms) and 8.4 ± 0.6 ms in response to condensation clicks (VI latency: 7.8 ± 0.7 ms). Thus, although the amplitude of peak B was more variable across subjects than the amplitude of peak A, the latency differences between rarefaction and condensation conditions were comparable.

Although the overall waveform of both the evoked potentials and the binaural interaction differed in amplitude and latency across individuals, for each subject the waveforms were stable within and across recording sessions. Six replications of binaural interaction from one recording session are shown in Fig 6, along with the interaction obtained from the same subject during a recording session three weeks later.

Changing click polarity produced a latency shift of 0.24 ms in the occurrence of the first upward deflection of the acoustic waveform (Fig 1). The change in the stimulus could account for the 0.15- to 0.35-ms latency shift of the auditory brainstem components that accompanied reversal of click polarity. However, the latency shift of binaural interaction as a function of click polarity was roughly three times larger (ie, 0.83 ms) than the latency shift for either the acoustic stimulus or the evoked potential components.

Effect of Signal Intensity

The amplitude of the binaural interaction decreased when click intensity was reduced from 70 to 60 dB SL. Moreover, the attenuation of binaural interaction was greater than the attenuation of the evoked potentials. For example, the amplitude of binaural interaction to rarefaction clicks presented at 70 dB SL was 23% ± 4% of the amplitude of the IV-V complex, whereas at 60 dB SL the interaction was only 15% ± 7% of the IV-V complex amplitude (Table). However, at both intensities, the interaction was larger in response to condensation clicks than in response to rarefaction clicks. When click intensity was decreased to 50 dB SL, binaural interaction to rarefaction clicks was attenuated further in one subject, augmented in one subject, and could not be distinguished from background noise in a third subject. Binaural interaction to condensation clicks presented at 50 dB SL was further attenuated (relative to higher stimulus levels) in all subjects. The latency of binaural interaction relative to wave V in the monaural summed waveform remained constant independent of signal intensity. Thus, the latency of

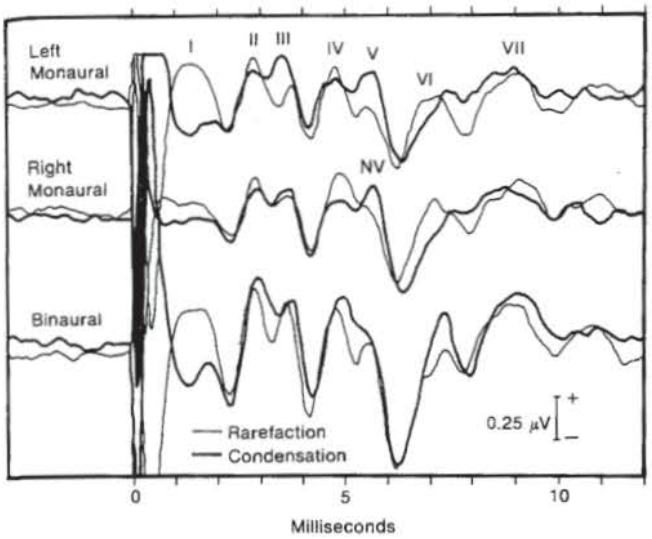


Fig 4.—Evoked potentials of subject BM to rarefaction and condensation clicks presented at 70 dB sensation level. Note that sensitivity of components IV, V, NV, and VI to click polarity described in composite waveforms is also evident in individual evoked potentials.

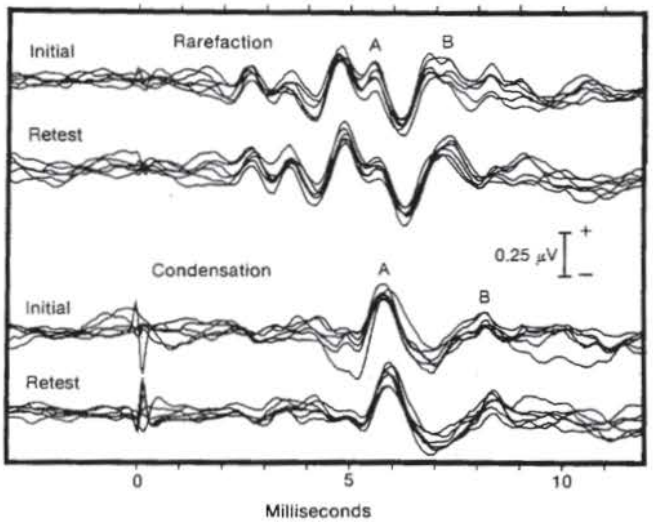
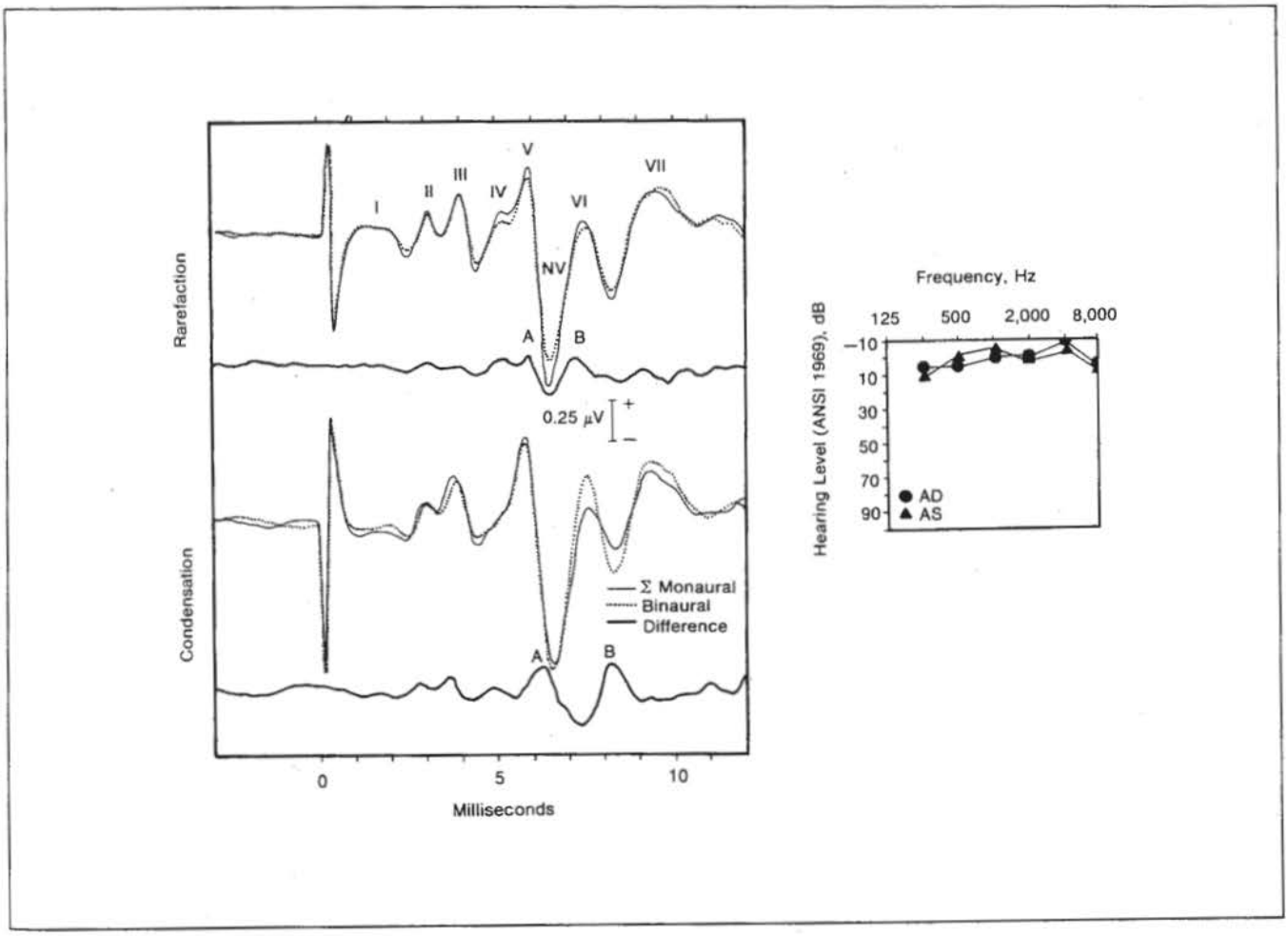


Fig 6.—Binaural interaction obtained during initial recording session and after a three-week interval (subject BR). Note reliable differences of waveforms derived from rarefaction and condensation conditions.

Fig 5.—Auditory brainstem responses (upper traces of each set) and binaural interaction (lower traces of each set) to rarefaction clicks and condensation clicks in normal-hearing subject (BQ). First and second binaural interaction components are labeled A and B, respectively. Note that amplitude and latency of binaural interaction are functions of click polarity. Pure tone audiogram (right ear, AD; left ear, AS) is presented on right.



the interaction varied with the latency of wave V.

Effect of Interaural Delay

As interaural delay was increased from 0 to 500 μs, the latency of the binaural interaction increased. This latency increase was proportional to the interaural delay, such that the latency of binaural interaction remained constant relative to the latency of the IV-V complex evoked by the trailing stimulus. At delays greater than 500 μs, many of the components of the monaurally evoked response were no longer recognizable in the binaural or summed monaural waveforms (Fig 7, top). For this reason, latency and amplitude measurements of the binaural interaction relative to the IV-V complex, or wave VI, could not be made. To make quantitative measurements, we identified the binaural interaction components on the basis of two criteria: (1) the latency of binaural interaction was assumed to stay constant relative to the latency of the IV-V complex evoked by the trailing monaural stimulus and (2) peak A was identified as the positive peak immediately preceding the large negative trough in the binaural interaction waveform; peak B was assumed to have a constant temporal relation to peak A. These criteria were adopted on the basis of observations made for delays equal to or less than 500 μs, where the relationship between binaural interaction and the IV-V complex in the binaural and summed monaural responses could still be observed.

As interaural delay was increased from 0 to 900 μs, the amplitude of peak A gradually decreased (Fig 7, top and bottom). With an interaural delay of 900 μs, the amplitude of peak A was at a minimum. A similar pattern was observed for peak B of the interaction waveform, although this component was more variable across the two subjects. For subject BQ (Fig 7, top, and closed circles in Fig 7, bottom), as interaural delay was increased from 0 to 900 μs, the amplitude of peak B first increased to a maximum at a 200-μs interaural delay and then decreased to 53% (of interaction at 0 delay) at 900 μs. Peak B amplitude for this subject then increased to 78% at 1,400-μs interaural delay and decreased to a minimum value of 20% with a delay of 2,000 μs. For subject BM (open circles, Fig 7, bottom) the amplitude of peak B gradually decreased to a minimum of 19% at an interaural delay of 900 μs, then increased with longer interaural delays. Thus, with the exception of the small amplitude of peak B in subject BQ at 2,000-μs interaural delay, the amplitude of both peak A and peak B was at a minimum with an interaural delay of 900 μs.

Amplitude of Binaural Interaction, Peak A*

	Intensity, dB SL†		
	70	60	50
Rarefaction, %	23±4	15±7	19±9
Condensation, %	35±12	28±8	19±9

*Relative to IV-V complex of Σ monaural. Values are means ± SD (n = 4).
†SL indicates sensation level.

Binaural Interaction in Subjects With Hearing Loss

The evoked responses and binaural interaction obtained from the two subjects with high-frequency hearing loss did not differ significantly from subjects with normal audiograms. One subject had a loss in one ear of greater than 30 dB for frequencies above 4 kHz (see audiogram, Fig 8); the other subject had a loss in both ears greater than 20 dB for frequencies above 1.5 kHz. The latencies of wave V at 70 dB SL were within normal limits, probably reflecting recruitment in a sensorineural hearing loss.[14]

For both subjects with high-frequency hearing loss, wave IV was larger in the rarefaction condition and wave V was larger in the condensation condition. As in normal subjects, the latency shift of NV was larger in the monaural potentials than in the binaural potentials. However, in contrast with subjects with normal hearing, wave VI was not consistently larger to rarefaction clicks in the monaurally evoked potentials and it was not consistently larger to condensation clicks in the binaurally evoked potentials.

The effect of polarity reversal on binaural interaction in subjects with high-frequency hearing loss did not differ from normal-hearing subjects. In the example shown in Fig 8, the interaction associated with the IV-V complex occurred 0.08 ms later than wave V in the rarefaction condition and 0.52 ms later than V in the condensation condition. These values are within the range of those for subjects with normal audiograms. Likewise, the latency difference between rarefaction and condensation evoked interaction is also within the range of that observed in subjects with normal hearing. Finally, the magnitude of peak A (relative to the monaural summed IV-V complex) was greater in the condensation condition (30.5% ± 5%) than in the rarefaction condition (26.0% ± 4%) for both subjects, as occurs in normal subjects.

The evoked responses and binaural interaction in the subject with hearing loss affecting both low and high frequencies differed from that in subjects with normal audiograms (Fig 9). A distinct wave IV was not apparent; therefore, the dependence of wave IV amplitude on click polarity could not be assessed. Furthermore, click polarity reversal did not result in significant amplitude changes of the IV-V complex. The latencies of the peaks in the evoked response to 70 dB SL clicks were within normal limits. However, wave VI was not present in response to monaural stimulation with rarefaction clicks, although it was present in the binaurally evoked response.

There was no interaction associated with the IV-V complex. The earliest, reliably recorded interaction occurred 1.16 ms later than the IV-V complex in the rarefaction condition and 2.12 ms later than the IV-V complex in the condensation condition. These values fall outside of the range of subjects with normal audiograms. However, the absolute latency and amplitude difference between rarefaction and condensation evoked interaction do not differ from subjects with normal audiograms. The sensitivity of binaural interaction latency to click polarity is compared across subject groups in Fig 10. Binaural interaction in the subject with both high- and low-frequency hearing loss occurred later relative to wave V than either normal subjects or subjects with high-frequency loss (Fig 10, left). However, in all subjects the latency of the interaction produced by condensation clicks was greater than the latency of the interaction produced by rarefaction clicks. Moreover, this latency difference was similar across all subject groups (Fig 10, right).

COMMENT

The results of this study demonstrate that binaural interaction in the auditory system is apparent in the human ABR 4.5 to 7.0 ms after click onset, coincident with waves IV-VI. The binaural interaction reflected in the evoked potential indicates a nonlinear processing of binaurally presented click signals when compared with the sum of the evoked potentials to monaural signals. The nonlinearity is small, amounting to only 30% reduction in the amplitude of components IV-VI at 70 dB SL. Furthermore, the amplitude of binaural interaction relative to the IV-V complex decreases as signal intensity decreases.

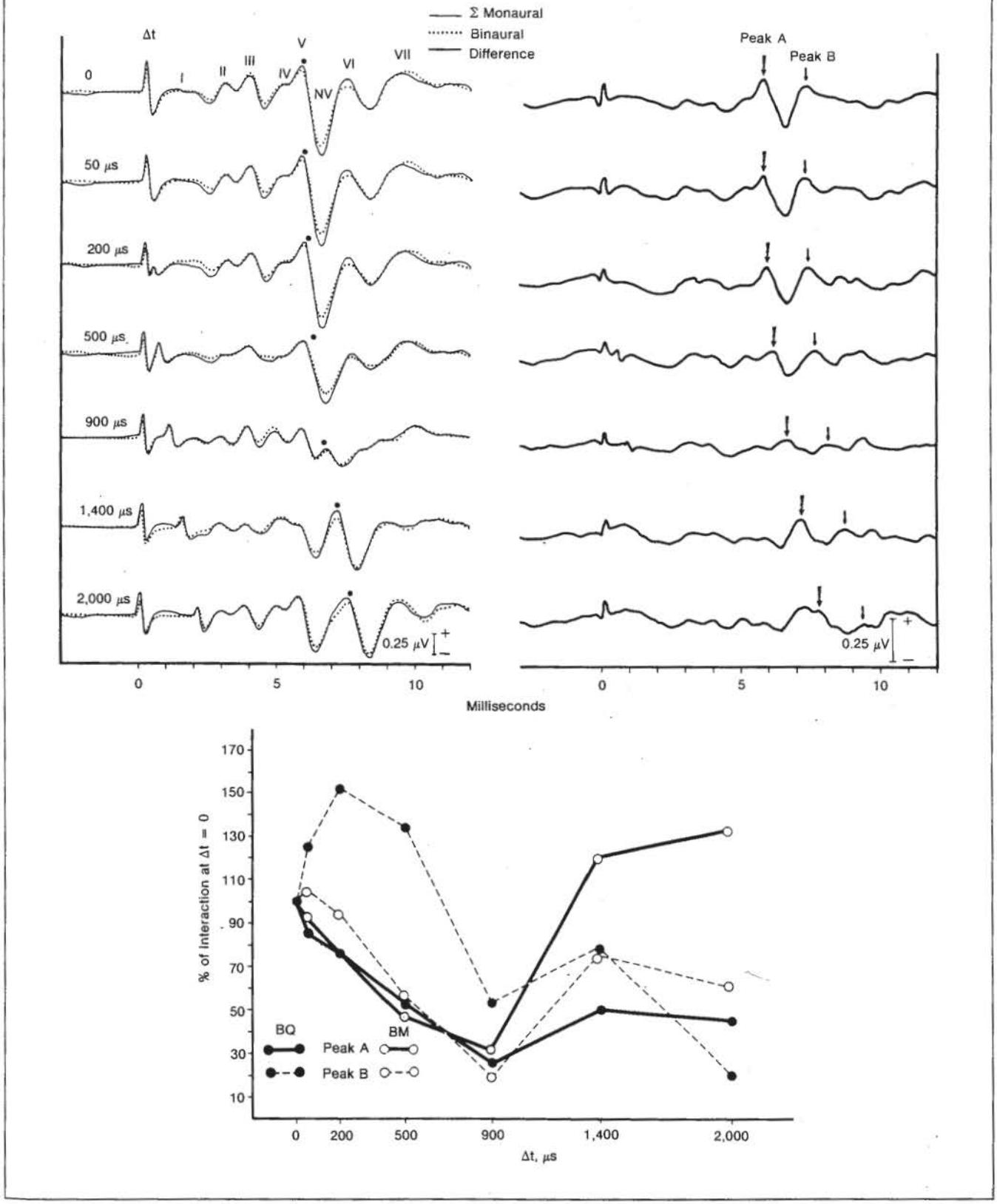


Fig 7.—Top, Effects of interaural delay on evoked responses (left) and binaural interaction (right) to rarefaction clicks (subject BQ). Filled circles above evoked response waveforms indicate estimated latency of wave V of trailing stimulus. Large and small arrows above interaction waveforms indicate peaks identified as first (peak A) and second (peak B) binaural interaction components, respectively. Bottom, Amplitude of binaural interaction components as function of delay. Ordinate is amplitude of binaural interaction expressed as percentage of amplitude at 0 delay. Functions for subject BQ (responses shown at top) indicated by filled circles, functions for subject BM are indicated by open circles. Note gradual attenuation of amplitude reaching its nadir at 900-μs interaural delay.

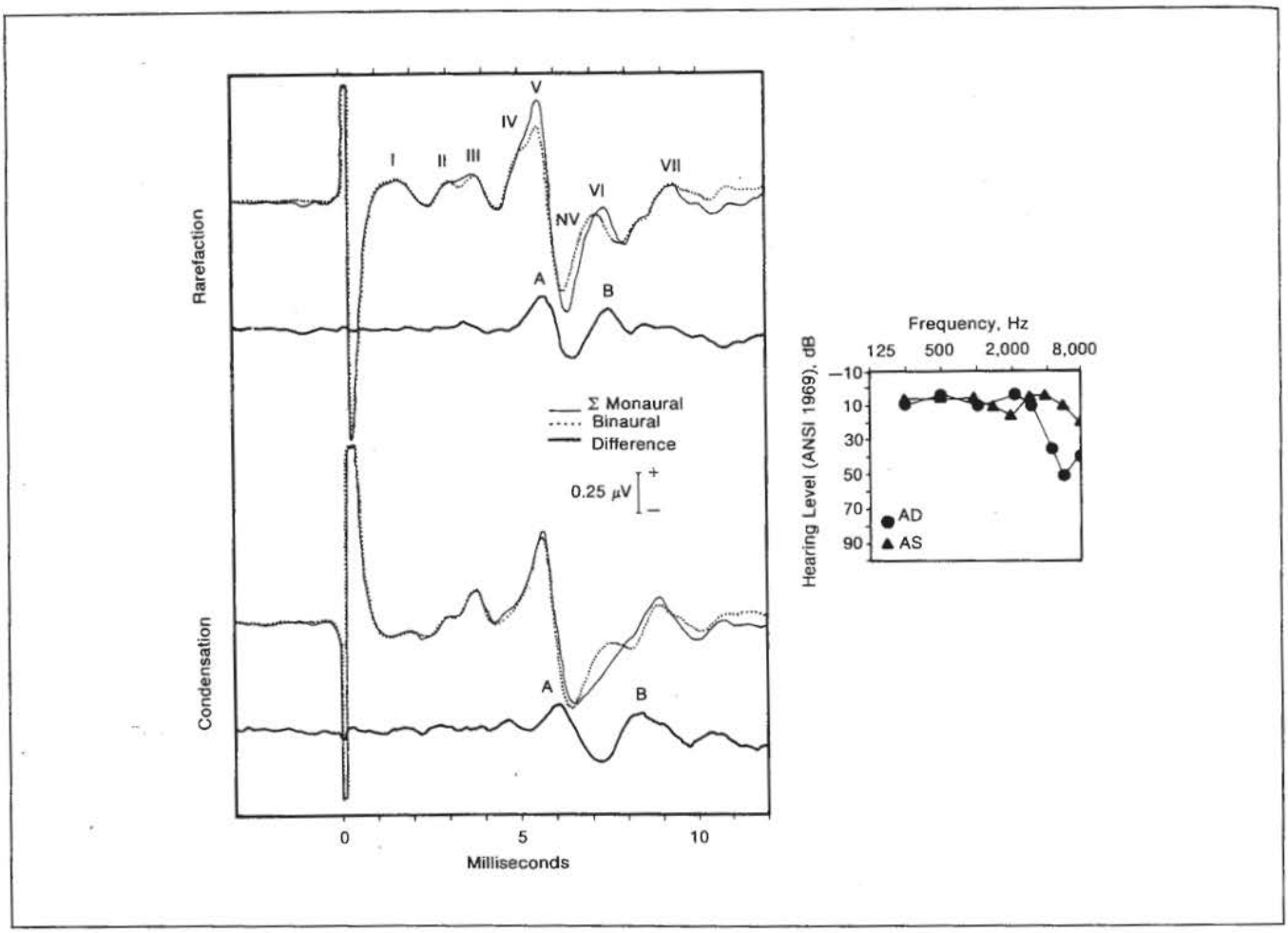


Fig 8.—Auditory brainstem responses and binaural interaction in subject with high-frequency hearing loss. Latency and amplitude of binaural interaction do not differ greatly from subjects with normal hearing. Pure-tone audiogram shown at right.

Other Reports

Binaural interaction has been recently described for a variety of animal species.[8, 10, 11] The interaction occurred at the time of the fourth scalp positive wave of the evoked response. Huang[11] noted that the interaction is actually bimodal, consisting in an initial brief-duration component associated with the fourth wave, which was larger in amplitude than a second broader component occurring approximately 1 ms later. Thus, it would appear that at least two components contribute to binaural interaction in these species. Levine[8] presented data that indicated that the second positive binaural interaction component in human beings may be due, in part, to acoustic crossover. We did not examine acoustic crossover effects.

The finding in the present study in human subjects of a bimodal interaction in the ABR, consisting in an initial component associated with the high-amplitude IV-V complex and a second component associated with wave VI, replicates the work of Dobie and Norton[8] and corresponds to the results of animal studies. The failure of Huang to demonstrate binaural interaction in the ABR in human subjects may result from the smaller number of stimuli averaged (2,000 vs 12,000 in the present study) and possibly the high frequency of his stimulus (10-kHz tone vs the broad-band click used in the present study).

The similarities of the interaction morphologic features and time of occurrence in relation to the evoked potentials across species are consistent with the hypothesis that homologous "generators" underlie the binaural interaction observed in these species. The site of these generators may reside in the rostral pons and/or midbrain; in man, the IV-V complex is dependent on the integrity of the midbrain.[13] Ben Clopton, PhD (written communication, April 1979), suggests that the interaction in the guinea pig may be generated from activity in the brachium of the inferior colliculus (BIC) or within the inferior colliculus itself. This hypothesis is based on the finding that binaural interaction recorded from an electrode in the BIC has similar latency, morphologic characteristics, interaural intensity, and interaural delay functions as binaural interactions derived from vertex recordings. Huang[11] also presented data pertinent to the generators of binaural interaction. Bilateral sectioning of the lateral lemniscus just ventral to the inferior colliculus abolished the broad, low-amplitude component of the brainstem evoked potential designated as peak V, which coincides temporally with the second broader component of the binaural interaction. Huang concluded that neurons in the inferior colliculus and those cells innervating inferior colliculus neurons are the primary generators of binaural interaction. This hypothesis must remain tentative, however, since direct lesion effects on the interaction itself were not reported, and the changes in the evoked potential occur

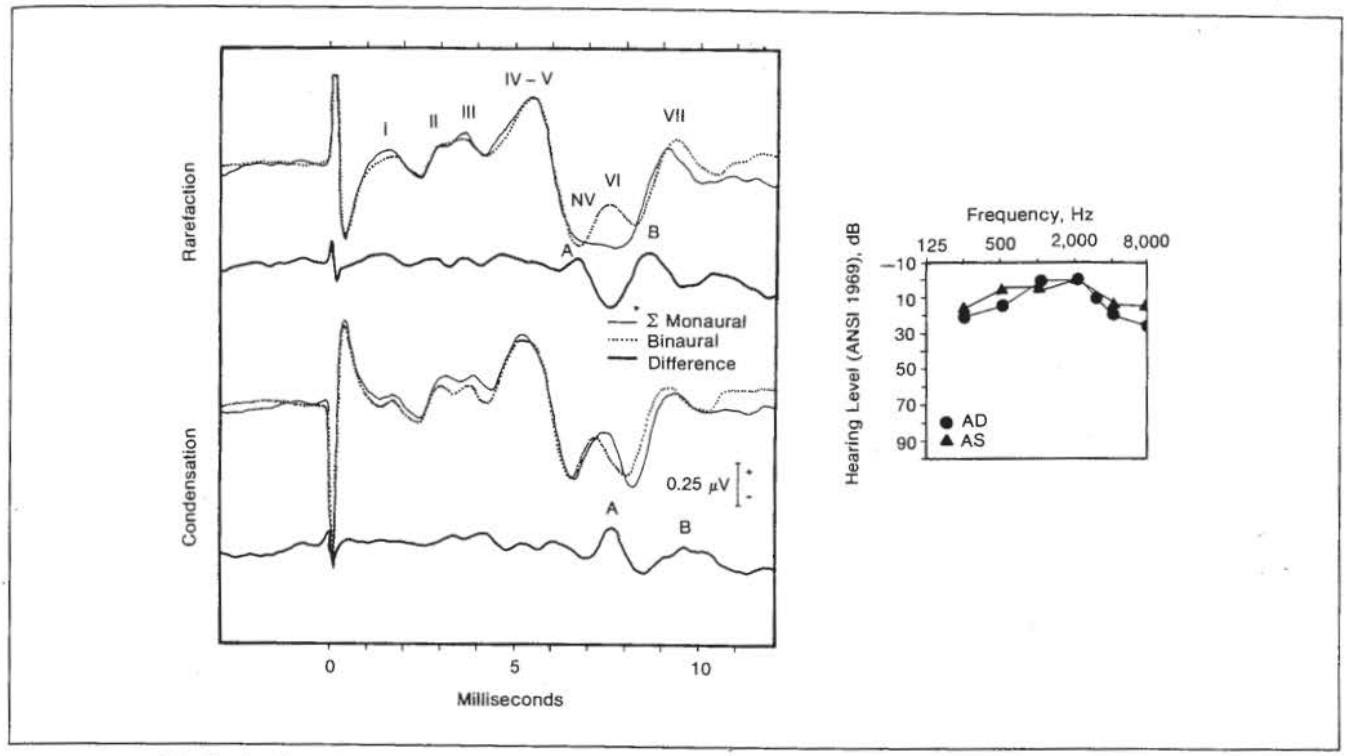


Fig 9.—Auditory brainstem responses and binaural interaction in subject with both high- and low-frequency hearing loss. Latency of binaural interaction relative to the IV-V complex is longer than observed in normal subjects or in subjects with high-frequency hearing loss. However, latency difference between binaural interaction in rarefaction as compared with condensation conditions is within normal limits.

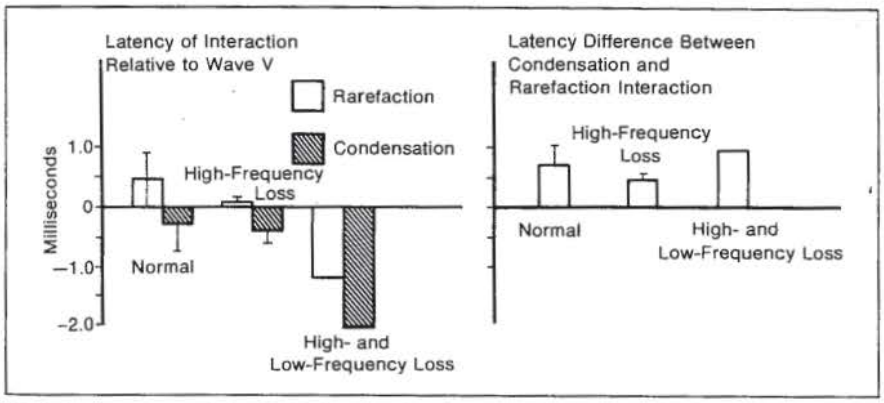


Fig 10.—Summary of effects of stimulus phase reversal on latency of binaural interaction across subjects. See text for description.

in a time domain that would be expected to affect only the second, not the first component of the binaural interaction.

Binaural interaction can be observed in the human brainstem evoked response as a deviation of the binaural response from the sum of the monaural responses. Since our measure of binaural interaction is essentially a difference score, it allows assessment of activity independent of such stimulus and recording variables as earphone placement, electrode impedance, or changes in physical measurements that would affect the monaural and binaural responses nondifferentially. Thus, binaural interaction may be a sensitive measure reflecting specific neural processes sensitive to the processing of monaural vs binaural stimuli.

Stimulus Polarity

The results of this study replicate and extend previous reports that stimulus polarity is an important variable in the amplitude and latency of the evoked ABR. The components most affected by polarity reversal were waves IV through VI. In agreement with Stockard et al,[7] wave IV is largest in response to rarefaction clicks and V is largest in response to condensation clicks. The evoked response to rarefaction clicks is typically of shorter latency than the response to condensation clicks (see also Ornitz and Walter[4] and Coats and Martin[14]). Previous studies have examined effects of stimulus polarity only under monaural stimulus conditions; we have found the effects described above also occur under binaural stimulus conditions. In addition, there are stimulus polarity effects that are dependent on whether a monaural or binaural stimulus is presented. These differential effects occur primarily on the later waves, NV and VI. These data suggest the possibility that two separate processes may underlie the effects of polarity reversal: one that is independent of the mode of stimulus presentation and a second process that causes differential effects dependent on whether a monaural or binaural stimulus is presented.

The morphologic characteristics, amplitude, and latency of the binaural interaction vary with click polarity. These effects reflect the differential effect of polarity inversion on the

monaural as compared with the binaurally evoked response. In particular, the changes in latency of the binaural interaction indicate that the differences between rarefaction and condensation evoked responses greatly exceed the small latency shift that results from click polarity reversal in the monaurally evoked response.

The effect of the polarity inversion on the latency of the binaural interaction is highly consistent across individuals. However, the large latency shift of 1 ms is not apparent in either the monaurally or binaurally evoked responses. This finding may be due to the fact that the interaction component of the binaural waveform is a minor percentage (20% to 30%) of the total evoked activity. Thus, a 1-ms shift of 25% of the evoked activity may be obscured by much smaller latency shifts of the remaining activity.

Inverting the polarity of the click stimulus will produce an effective latency change in the time of activation of cranial nerve VIII fibers owing to the fact that movement of the basilar membrane in only one direction produces neural activity.[15] Thus, in cranial nerve VIII fibers, stimulus polarity inversion will result in a latency difference that will correspond to one half the period of the stimulating frequency. The 180° phase shift will produce larger latency differences for low-frequency units than for high-frequency units.

Low-Frequency Component

The 1-ms latency shift observed in the binaural interaction as a function of click polarity would be consistent with the hypothesis that low-frequency components are involved in the production of the interaction recorded from surface electrodes. A 1-ms latency shift of the neural activity would occur for units stimulated by frequencies centering around 500 Hz. This hypothesis is also supported by the finding that high-frequency hearing loss (4 kHz and above) does not substantially alter the binaural interaction. In contrast, binaural interaction was delayed in the subject with both a low- and high-frequency hearing loss. This latency increase may be the result of the effective attenuation of those components of the response dependent on low stimulus frequencies. The results from the study of interaural delay also bear on this hypothesis. The attenuation of binaural interaction at an interaural delay of 900-μs would be expected if the effective stimulating frequencies were centered close to 550 Hz. However, this hypothesis must remain tentative until criteria independent of the binaural or summed monaural waveforms are developed to identify specific components of binaural interaction.

Future Experimentation

It is clear that additional experiments are needed to test the hypothesis that binaural interaction in the ABR reflects low-frequency stimulation. One experimental approach might be to employ the derived response technique using high-pass noise masking[16] to define the contribution of the various portions of the acoustic spectrum to binaural interaction. The major difficulty with this experiment is that binaural interaction involves a minor portion of the ABR, and the small amplitude of the derived brainstem responses in human beings may prohibit the detection of binaural interaction. It may prove more productive to analyze the acoustic aspects of binaural interaction in the ABR in experimental animals, where the evoked response is often of larger amplitude, and the number of trials necessary to define binaural interaction makes the experimental protocol more rapid.

The relationship of this electrophysiological measure of binaural interaction to binaural psychoacoustic phenomenon is unclear. The definition of this relationship could be important for analyzing binaural neural processes in man. Moreover, there may be clinical usefulness in measuring binaural interaction in the ABRs in man for defining brainstem lesions not apparent in monaural testing.

Subsequent to submission of this manuscript, another investigation of binaural interaction in auditory brainstem responses has been published.[17] Those authors reported that acoustic crossover may account for much of the observed binaural interaction. In order to assess the contribution of acoustic crossover in our system, we have retested two subjects as follows: In the monaural condition, 70 dB SL rarefaction clicks were presented to one ear while broad-band masking noise (55 dB SL) was delivered to the other ear. One of the subjects was also tested with the same monaural clicks, but with soft plastic clay inserted to occlude the opposite auditory canal. For the binaural condition, binaural clicks without masking noise or occluded ear canal were presented at 70 dB SL. Binaural interaction was still evident and did not differ significantly from the unmasked or unoccluded conditions. Thus, we conclude that acoustic crossover does not account for the binaural interaction described in this article. Nevertheless, we do agree with Ainslie and Boston that acoustic crossover can be a significant factor varying with differences in earphones, methods of acoustic presentation, and experimental procedures.

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