

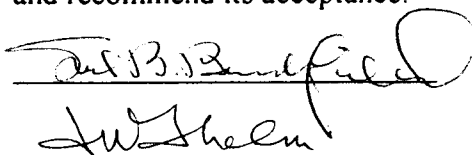
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I am submitting herewith a thesis written by Carol A. Boyd entitled "Human Frequency Following Response Correlates of Interaural Time Differences and Time-Intensity Trade". I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Audiology.



Dr. A. Krishnan, Major Professor

We have read this thesis
and recommend its acceptance:



Accepted for the Council:



Associate Vice Chancellor and
Dean of The Graduate School

Human Frequency Following Response Correlates of Interaural Time Difference and Time-Intensity Trade

A Thesis Presented for the Master of Arts Degree
The University of Tennessee, Knoxville

Carol A. Boyd
May 1997

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DEDICATION

This thesis is dedicated with much love to my mother, David R., Marshay, Jeanice, John, Candice, Lerkenda, and Marcia.

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To all, I greatly appreciate whatever contribution you have made to the completion of this project.

ABSTRACT

The binaural interaction component (BIC) is the difference between the sum of two independent monaural responses and a binaural response. The presence of an electrophysiologic correlate of lateralization as exhibited in the BIC of the human frequency following response (FFR) evoked by 500 Hz tone bursts and the alterations of the BIC as a function of interaural time differences (ITD) and time-intensity trading was investigated. The FFR-BIC was demonstrated. The FFR changes correlated with our psychoacoustic results suggesting that the FFR-BIC and the binaural (BIN) FFR may be related to lateralization of the fused image of a low frequency sound. The results related specifically to the reduction in the amplitude of the BIN FFR and the FFR-BIC components as the ITD increased and also to the increase in the amplitude of the BIN FFR and the FFR-BIC components as increasing IID imposed on an ITD lateralized sound. The nature of these alterations in the BIN FFR and the FFR-BIC components are consistent with the idea of non-linear neural interactive processes within the rostral brainstem mediating these changes.

TABLE OF CONTENTS		
CHAPTER		PAGE
I. REVIEW OF LITERATURE		1
Introduction		1
The Binaural Interaction Component		2
Neural Bases of Binaural Interaction		3
Neuroanatomy		3
Neuronal types		3
Neural Mechanisms for the BIC		4
Single-unit studies		4
Evoked potential studies		5
Neural Generators of the Evoked Potential BIC		7
The Frequency Following Response		9
Origin of the FFR		9
Response characteristics		9
BIC in the FFR		10
Time-Intensity Trading		11
II. RATIONALE		12
III. METHOD		14
Subjects		14
Stimulus		14
Recording System		15

Response Evaluation	15
Experimental Protocol	16
Experiment 1: Psychoacoustics	17
Experiment 2: Effects of ITD on the FFR-BIC	17
Experiment 3: Effects of trading intensity for time on the FFR-BIC	18
IV. RESULTS	20
Sound Lateralization: Psychoacoustic Data	20
Effects of ITD	20
Effects of Trading ITD with IID	20
Sound Lateralization: Human FFR Data	23
Effects of ITD on the FFR	23
Effects of IID (trading for ITD) on the FFR	28
Comparison of the Effects of ITD and IID on the FFR	32
Summary of Results	34
Effects of ITD on the FFR	34
Effects of IID (trading for ITD) on the FFR	34
Comparison of the Effects of the ITD and the IID on the FFR	35
V. DISCUSSION	36
Effects of ITD on the Binaural Response	36
The FFR Binaural Interaction Component	40

Effects of Trading ITD with IID on the Binaural Frequency Following Response	41
Anatomical Locus of Binaural Interaction	42
Correlation of FFR Results with Psychoacoustic Results	43
Potential Limitations of this Study	44
Implications	46
Conclusion	47
REFERENCES	48
APPENDICES	54
Appendix A: Subject Consent Form	55
Appendix B: Interaural Time Difference Individual Amplitude Data (in dB volts) for Summed Monaural Responses Evoked By 500 Hz Tone Bursts	58
Appendix C: Time-Intensity Trade Individual Amplitude Data (in dB volts) for Summed Monaural Responses Evoked By 500 Hz Tone Bursts	60
Appendix D: Interaural Time Difference Individual Amplitude Data (in dB volts) for Binaural Responses Evoked By 500 Hz Tone Bursts	62
Appendix E: Time-Intensity Trade Individual Amplitude Data (in dB volts) for Binaural Responses Evoked By 500 Hz Tone Bursts	64
Appendix F: Interaural Time Difference Individual Amplitude Data (in dB volts) for Binaural Interaction Component	66
Appendix G: Time-Intensity Trade Individual Amplitude Data (in dB volts) for Binaural Interaction Component	68
VITA	70

LIST OF FIGURES

FIGURE		PAGE
Figure 1.	Illustration of the lateralization paradigm used in this study.	19
Figure 2.	Psychoacoustic sound lateralization using the ITD cue.	21
Figure 3.	Psychoacoustic sound lateralization using the ITD cue for time-intensity trading.	22
Figure 4.	FFR waveform data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of ITD.	24
Figure 5.	FFR spectral data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of ITD.	25
Figure 6.	Median amplitude change across subjects representing MON(S) (top panel), BIN (middle panel), and BIC (bottom panel) as a function of ITD.	26
Figure 7.	FFR waveform data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of time-intensity trading.	29
Figure 8.	FFR spectral data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of time-intensity trading.	30
Figure 9.	Median amplitude change across subjects representing MON(S) (top panel), BIN (middle panel), and BIC (bottom panel) as a function of time-intensity trading.	31
Figure 10.	Comparison of changes in BIN and BIC amplitudes due to ITD and IID. IID changes are to the right and top of the figures. ITD changes are to the left and bottom of figures.	33
Figure 11.	A schematic diagram of the two-level decision making model for lateralization proposed by Furst et al.(1995).	39

CHAPTER I

REVIEW OF LITERATURE

Introduction

The auditory system is able to identify the location of a sound source in space by comparing the intensity, and the time of arrival of sounds at the two ears. The identification of the location of a fused sound image intracranially is termed lateralization, which involves binaural presentation of stimuli through earphones.

Psychoacoustic data indicate that complete lateralization (90 degrees) of the fused sound image occurs when there is about 15-20 dB or greater interaural intensity difference (IID) or when the interaural time difference (ITD) is 1.5 ms or greater (Mills, 1960; Yost, 1981; McPherson and Starr, 1995). When the ITD exceeds about 1.8 ms there is no longer a percept of a fused image (Yost and Nielsen, 1985, p. 156).

While the neurophysiological bases of sound lateralization are not completely known, it has been clearly demonstrated that auditory brainstem neurons are systematically influenced by either interaural time or interaural intensity differences presented in the binaural stimulation (Hall, 1965; Goldberg and Brown, 1969; Stillman, 1972; Moushegian et al., 1975). The superior olivary complex (SOC) is the first structure in the brainstem where this binaural interaction occurs (Rosenzweig and Wyers, 1955; Hall, 1965; Rose et al., 1966; Boudreau and Tsuchitani, 1967; Wernick and Starr, 1968; Guinan et al., 1972; Moushegian et al., 1975; and Dobie and Berlin, 1979). Binaural interaction has also been demonstrated at more rostral levels [including the lateral lemniscus (LL) and inferior colliculus (IC)] in the brainstem (Stillman, 1972; Rosenzweig and Sutton, 1957; Jeffress,

1948). On the basis of these results, it has been proposed that sound lateralization is mediated by a complex binaural interaction mechanism involving neural excitation and inhibition.

The Binaural Interaction Component (BIC)

The binaural interaction component (BIC) is the difference between the sum of two independent monaural responses and a binaural response. Dobie and Berlin (1979) describe this BIC as any deviation from a noninteractive model. This assumes that there are "generators" for two separate brainstem auditory-evoked responses (BSERs), one for each ear. Also, this assumes that binaurally evoked BSERs would be predicted exactly by summing the monaural evoked responses. Therefore, the presence of a binaural difference potential suggests that the binaural response is not a simple algebraic sum of two independent responses. Thus, the BIC is due to binaural processes mediating lateralization and binaural fusion. The BIC has been clearly demonstrated in the binaural neurons of the superior olivary complex (Wernick and Starr, 1968; Moushegian et al., 1975) and the inferior colliculus (Flammino and Clopton, 1975; Erulkar, 1959; Stillman, 1972). The BIC has also been demonstrated in the evoked potentials recorded in animals (Wernick and Starr, 1968; Dobie and Berlin, 1979; Gardi and Berlin, 1981; Ozdamar et al., 1986; Wada and Starr, 1989), and humans (Hosford et al., 1979; Dobie and Norton, 1980; Arslan, Prosser, and Michelini, 1981; Wrege and Starr, 1981; Dobie, 1982; Berlin et al., 1984; McPherson et al., 1993; McPherson and Starr, 1995).

Neural Bases of Binaural Interaction

Neuroanatomy

Wernick and Starr (1968), Moushegian, Rupert, and Gidda (1975), Flammino and Clopton (1975), and Erulkar (1975) have shown that binaurally innervated neurons are present in the nuclei of the SOC and the IC in mammals. The first structure in the brainstem to process binaural information is the SOC. Irvine (1992) reports the SOC to be the first site at which afferent fibers from both cochlea nuclei's converge, and the point wherein the majority of neurons at that level and at all higher levels of the auditory pathway are binaurally innervated. The medial superior olive (MSO) receives direct fibers from the cochlea nuclei (CN) of both sides and predominately represents low frequency stimuli. The lateral superior olive (LSO) receives direct inputs from the ipsilateral cochlear nucleus and indirect inputs from the contralateral cochlear nucleus via the medial nucleus of the trapezoid body (MNTB). The cue available in low frequency stimuli, the ITD, is used by the MSO in detecting the direction of a sound source. The LSO predominately processes high frequency stimuli.

Neuronal types

Goldberg and Brown (1969) originally proposed two major types of binaural neurons in the SOC of the dog. With the first type of neurons, stimulation from either ear can be excitatory, therefore called excitatory-excitatory (EE) neurons. These types of neurons are not sensitive to IID, as long as the average intensity is constant between the ears. The second type of neurons are excited by stimulation from one ear and inhibited by

stimulation of the other ear, therefore called excitatory-inhibitory (EI) responses. Irvine (1992) suggests that the inhibitory input in most EI or IE neurons is revealed because the net effect of the response to binaural stimulation is smaller than the response to monaural stimulation of the ear providing excitatory input. In other words, the effects of inhibition causes reduced binaural responses in comparison to the monaural response receiving only excitatory input.

McPherson and Starr (1995) using far-field electrophysiologic recordings described the same two types of binaural neurons in the SOC, the EE and EI neurons. They report the EE neurons are found primarily in the MSO nucleus, excited by stimuli's received from both ears and the EI neurons primarily in the LSO nucleus, excited by input from one ear and inhibited by input received at the other ear.

Neural Mechanisms for the BIC

Single-unit studies

Yin and Chan (1990) studied the neural mechanisms underlying interaural time sensitivity in the MSO. The cells in the MSO respond in accordance with the "coincidence model" proposed by Jeffress (1948). This model suggests that axons, caused by acoustic stimulation, carry impulses from the respective ear and the cells at which the two impulses converge simultaneously will indicate the position of the sound source. Thus, one can determine which acoustical signal occurred first or last. For example, if an acoustic signal is delayed at the left ear, the propagational velocity of neural transmission will cause impulses generated at each ear to converge simultaneously at the left side. Thus, this

model places emphasis on excitatory inputs.

Single unit studies of the MSO have been difficult because of the thinness of the nucleus and the large field potentials in that area due to the convergence of many fibers. Yin and Chan (1990) report that much of the binaural phenomenon seen in the inferior colliculus of the cat (Kuwada and Yin, 1983; Yin et al., 1986, 1987) are reflections of binaural processing originating in the MSO. They found an overrepresentation of low frequency neural fibers in the MSO. This finding is consistent with the idea that the MSO is important for the processing of ITDs and stresses the usefulness of ITDs for low frequency stimuli. They report that most cells in the MSO are binaural, responsive to stimulation of either ear, and sensitive to changes in time. Fine structure processing has phase locking characteristics with periodic discharge rates. This processing mimics the frequency of the stimulus throughout the duration of the signal. Cells in the MSO receive inputs carrying temporal information about the fine structure of the acoustic stimulus, which enables them to achieve sensitivity to ITDs.

Rosenweig and Sutton (1957) attempted to determine whether a BIC was present at the LL of the cat. They found a statistically significant interaction when the time interval between clicks, one delivered to each ear, was less than 2 ms.

Evoked potential studies

The BIC has been clearly identified in the auditory brainstem response (ABR) of animals and humans and in the human frequency following response (FFR). Dobie and Berlin (1979) found the BIC to be robust in guinea pigs at peak IV (3.5 to 4.0 ms)

regions: IID of +20 dB and ITD of +1 ms. Wave IV in animals is the same as wave V in humans. Though much reduced in magnitude this BIC was also present at extreme levels for IID of +40 dB and ITD of +3 ms. Dobie and Norton (1979) observed a strong BIC in the guinea pig maximally at approximately 4 ms in latency with IID's and ITD's equal to 0.

Ornitz and Walter (1975) and Stockard et al. (1979) reported that the latency and morphology of the human ABR was affected by the initial phase of the stimulus primarily in components IV through VI, which are those that exhibit binaural interaction in humans. Wrege and Starr (1981) discovered that the BIC was reduced in amplitude for components IV through VI when the stimulus was 70 dBSL and as the signal intensity decreased, the amplitude of the IV-V complex of the BIC also decreased. The shift in the latency of the BIC and the shift in the IV-V complex were proportional from 0 to 500 microseconds but was absent at an ITD of 900 microseconds. Amplitude and latencies did in fact depend on click polarity in that, condensation clicks produced BICs of larger amplitudes and longer latencies than did rarefaction clicks.

Furst et al. (1985) report that the stimuli must be perceived as a single fusion intracranially in order for a BIC to be present. The first major peak of the BIC to click stimulation occurred at about 7.5 ms and was observable only when the binaural signals were perceptually fused and intracranially localized. Fusion and localization did not occur when ITDs were greater than 1.2 ms or when IIDs were greater than about 30 dB, therefore, the BIC was absent. McPherson and Starr (1995) report a BIC peak at approximately 7 ms and it is of maximal amplitude for an ITD of 0 ms and an IID of 0 ms. As these cue values increase the BIC illustrates a gradual broadening in duration, decrease

in amplitude, and prolongation in peak latency. These researchers support the findings of Furst et al. (1985) in that the BIC only occurs when the binaural stimuli are perceptually fused or intracranially localized. They report an absence of the BIC at or above an IID of 16 dB, the point where there is complete lateralization, or an ITD of 1.6 ms, the point when binaural fusion is lost. This research also indicates the effects on the BIC latency is greater for the IID than for the ITD.

Neural Generators of the Evoked Potential BIC

Melcher (1996) aimed to determine the cellular generators of the BIC by injecting kainic acid into the SOC or the cochlea nucleus (CN) in cats. A normal evoked potential BIC was described as possessing two prominent positive responses (one labeled as "b" and the other as "d") and a negative trough (labeled as "c"). Results from this study were compatible with the conclusion that MSO principal cells generate "b" and the cells act maximally when inputs from the two ears arrive within the same time window. The effects of the lesions on the BIC revealed that spherical cells in the anteroventral cochlear nucleus (AVCN) are essential for the production of a BIC. Lesions that were confined to this portion of the CN reduced the BIC substantially. This researcher further indicates that although the LSO is a clear site of binaural interaction, perhaps it may play a rather insignificant role in the BIC. The LSO, where EI neurons are present, receives inputs from spherical cells in the ipsilateral AVCN and indirect inputs from globular cells in the contralateral AVCN and posteroventral cochlear nucleus (PVCN). However, a lesion of the AVCN/PVCN had no effect on the BIC. Lesions in this area were expected to elicit a

BIC change.

Gardi and Berlin (1981) reported the BIC waveform to be typically biphasic (negative and positive), with peak latencies for click signals at 80 dBpeSPL of 4.0 and 4.75 ms, respectively. To clarify the possible sites of origin of the BIC, they used a two-step ablation to search for neural structures that might be responsible for its' generation. They conclude that the BICs generator site has a more caudal source other than the IC and it "occurs in time after wave III in the ABR for the reason that no physiologically synchronous temporal integration occurred at or before the level of the generator of wave III in the ABR" because synchronous integration/binaural representation occurs at the SOC. The SOC is thought (along with other structures) to generate wave IV (Jacobson, 1994). This conclusion was drawn because results indicated that the BIC did not occur until approximately 4.0 ms after the stimulus onset, as measured by the initial negative peak. There was approximately 40% reduction in the amplitude of the BIC waveform and about .2 to .3 ms increase in latencies after ablation. Ablation of the inferior colliculus (IC) caused little change to occur to the morphology, amplitude, or latency of the BIC waveform. After complete mid-sagittal sectioning of the brainstem, to eliminate crossed connections, wave III was completely eliminated. This finding indicates that the BIC was due to crossed fibers. Dobie and Berlin (1979) suggest BIC latencies of 3.5 to 4.0 ms to be most consistent with third order neurons, many of which arise from the SOC.

Gardi and Berlin (1979) found the BIC was more likely to be generated by higher frequency tone-pips rather than lower frequency tone-pips. They report "the BIC waveform can be accounted for by the presence of high frequency neurons firing in

response to equal intensity signals presented binaurally to each ear". Based on this reasoning, the only primarily high frequency sensitive region in the proposed generator area is the LSO.

The Frequency Following Response (FFR)

Origin of the FFR

The frequency following response (FFR) is a sustained response with a waveform that mimics the frequency of the stimulus, reflecting neural activity synchronized to individual cycles of low frequency stimuli (Parthasarathy and Moushegian, 1993). The first report of the FFR recorded from human scalp was published by Moushegian et al. in 1973. Evidence suggests an upper brainstem source for the human FFR.

Response characteristics

The FFR resembles the cochlear microphonic and it reproduces the electrical waveform of the sound input with an onset latency of approximately 6 milliseconds (ms). The FFR response latency is comparable to the wave V of the click evoked ABR (Glaser, Suter, Dasheiff, and Goldberg, 1976). The FFR has been described (Gerken, Moushegian, Stillman, and Rupert, 1975) as consisting of several waves at 2 ms intervals phase locked to the period of the tonal stimulus. The FFR was obtained over a range of frequencies, but was most apparent at lower frequencies.

Hink, Kadera, Yamada, Kaga, and Suzuki (1980) report FFRs arise from the brainstem because neural information could not travel much beyond this level during the 6 ms latency period of the FFR. In addition, near field recordings from the IC also show a

response at 6 ms. At this level of the auditory pathway, information from the two ears have had several opportunities for integration, in the SOC, the dorsal nucleus of the LL, and IC.

BIC in the FFR

Researchers believe that the BIC is a consequence primarily of high frequencies (Gardi and Berlin, 1981), a combination of high and mid frequencies (Wilson et al., 1985), or essentially low frequencies (Wrege and Starr, 1981). Gerken et al. (1975) compared binaural to summed monaural FFR's in humans and found no significant differences using a 500 Hz stimulus. This was not true for other studies (Parthasarthy and Moushegian, 1993; Wernick and Starr, 1968). These researchers found that the BIC can be evoked by low-to-high frequencies, however, morphologic characteristics (i.e., latency and amplitude) of the BIC to low frequencies resemble the FFR and do not resemble BIC's evoked by high and mid frequencies.

Parthasarthy and Moushegian (1993) reported a study of the FFR BIC to tonal stimuli in normal hearing adults. The consequences of increasing signal repetition rate minimally affected the FFR and BIC latencies to low frequency tone bursts of 500 and 1000 Hz attributed to the presence of a large population of phase locking neurons in the SOC. However, the amplitudes of the responses were modified. The effects of frequency on the FFR and BIC showed significantly shorter latencies to a 1000 Hz versus 500 Hz tone burst. Thus, as frequencies are lowered, longer waveform latencies are produced, although, not in a predictable or uniform fashion. The effect of intensity on the FFR and BIC amplitude was insignificant.

Time-Intensity Trading

Time-intensity trading is a process whereby the interaural cues are independently manipulated and placed in opposition with each other. If a signal in one ear is made to lead the signal in the other ear by a certain amount of time, the image will move to the lead ear. However, if sound arrives earlier at one ear while at the same time the signal intensity is increased at the other ear, the sound source will lateralize from the lead ear back to the midline because of the increase in intensity. The trading ratio describes the amount of time needed to offset a given intensity difference.

Harris (1960) used a filtered click to restrict the signal energy to a relatively narrow frequency region to measure the time-intensity trading ratio at various center frequencies. The results indicated that the trading ratio depended on the frequency region of the signal. At low frequencies (below approximately 1500 Hz) the trading ratio was much less than at higher frequencies. The value of the trading ratio in the low frequencies was typically 25 $\mu\text{sec/dB}$ to 90 $\mu\text{sec/dB}$ at the higher frequencies.

CHAPTER II

RATIONALE

Sound localization is thought to be mediated by a complex neural interaction mechanism involving excitation and inhibition of neural inputs presented to the binaurally sensitive neurons in the auditory brainstem. Physiological studies have not only demonstrated the existence of this binaural interaction in the auditory brainstem neurons (Wernick and Starr, 1968; Moushegian, Rupert and Gidda, 1975; Stillman, 1972; Flammino and Clopton, 1975) but also have shown that it is systematically influenced by localization cues of IID and ITD presented in binaural stimulation (Hall, 1965; Goldberg and Brown, 1969; Stillman, 1972; Moushegian, Rupert, and Gidda, 1975).

A BIC reflecting binaural processes mediating localization and binaural fusion has also been demonstrated in the scalp recorded ABR from animals (Dobie and Berlin, 1979; Wada and Starr, 1989) and humans (Furst et al, 1985; McPherson et al, 1993; McPherson and Starr, 1995). Recently, a BIC and its dependence on an IID has been described for the human FFR (Parthasarathy and Moushegian, 1993; McDaniel and Ananthanarayan, 1994). Unlike the onset ABR, the human FFR measures sustained neural activity within the rostral brainstem (Smith et al., 1975; Gardi et al., 1979) that is phase-locked to the individual cycles of low frequency tonal stimuli. Also, the human FFR is robust for stimulus frequencies below 1000 Hz where the ITD is the prominent cue for localization. Given these characteristics of the FFR it seems reasonable that the FFR-BIC would be influenced by ITD in a predictable manner. Therefore, one objective of this

study is to characterize the FFR-BIC alterations as a function of ITD.

Electrophysiological and psychoacoustic data indicate that the ITD and IID cues can be traded one for another. To the extent that the FFR-BIC reflects neural processes involved in lateralization, systematic variations or trading time with intensity should also produce a predictable change in the FFR-BIC. Although the ITD can be traded for an IID, the two may not be traded in an equivalent manner. Harris (1960) suggests that time differences and intensity differences may not be equivalent because there is variability in localization judgments when sounds are centered that are equal in intensity versus when an image is centered by offsetting an intensity difference with a time difference. Therefore, the second objective of this study is to examine this issue of trading the two cues. Specifically the alterations in the FFR-BIC during the course of trading the lateralized image, due to ITD, by an IID (which brings the image back to midline) will be evaluated.

CHAPTER III

METHOD

Subjects

Ten human subjects ranging in age from 18 to 30 years participated in this study. The criteria for inclusion in the study included: (i) hearing sensitivity of 20 dB HL or better for octave frequencies from 500 Hz to 8000 Hz, bilaterally; (ii) normal appearance of the ear canal; and (iii) normal tympanograms. All subjects were unpaid volunteers.

Stimulus

Frequency following responses (FFR) were recorded using two independently generated 500 Hz tone bursts. The overall duration of each tone burst stimulus was 40 ms, including 5 cycles (10 ms) of linear rise and fall times and thirty cycles (60 ms) of plateau duration. The tone burst stimuli was digitally synthesized and controlled by a signal generation and data acquisition system (Tucker-Davis, System II) interfaced to a microcomputer (Zenith, 486DX, 66MHz). The dual-channel stimulus generation module (TDT-DA1) was triggered by pulse(s) delivered by the timing generator module (TDT-TG6). Each output of the dual channel stimulus generators was then routed to a separate programmable attenuator (TDT-PA4). The outputs of the attenuators was routed to the headphone buffer module (TDT-HB) before being delivered to the earphones. The timing generator module also controlled the timing of the stimuli and provided the synchronous trigger pulse for the onset of digitization. Digital signal generation including

control of parameters, such as overall intensity, interaural time difference, interaural intensity difference and programming of the research protocol was accomplished by an interactive signal generation and control software (TDT-Siggen). The tone burst stimuli was presented at a rate of 4.8/second using rarefaction polarity (unless otherwise specified) either monaurally or binaurally through magnetically shielded TDH-49 earphones encased in Mu-metal.

Recording System

Subjects reclined comfortably in an acoustically shielded room after being instructed to sit quietly and remain still during the recording session. The FFRs were recorded differentially between scalp electrodes placed on the midline of the forehead at the hairline and the 7th cervical vertebra (C7 location). Another electrode placed on the left mastoid served as the common ground. The interelectrode impedances was maintained below 3000 ohm. The EEG inputs was amplified by 200,000 and band-pass filtered from 100 to 3000 Hz (6 dB/octave roll-off, RC response characteristics). Each response waveform was an average of 2000 stimulus presentations over a 100 ms analysis window using a sampling rate of 25 kHz. Data acquisition was accomplished by the signal averaging and data analysis software (BioSig) of the TDT, System II.

Response Evaluation

Evaluation of the FFRs to monaural and binaural stimulation included: (i) algebraic summation of the monaural FFRs; (ii) derivation of the FFR-BIC by subtracting the

binaural FFR from the summed monaural responses; (iii) Fast Fourier Transforms (FFT) of summed monaural FFRs, binaural FFRs and the FFR-BIC. The FFT yielded both the magnitude and the phase spectrum of the FFR. However, only the FFT magnitude spectral peak at 500 Hz was utilized to express FFR amplitude for each experimental condition. Using this measure of amplitude, the change in the FFR-BIC as a function of interaural time difference (ITD) and time-intensity trade was evaluated. Finally, the FFRs obtained under the binaural condition which produced a midline fused image of the tone burst stimuli served as reference for comparing with FFRs obtained under binaural that lateralized the fused image. The data analysis portion of the averaging program was used to accomplish response addition and subtraction, spectral analyses, and printing of waveforms and spectral data.

Experimental Protocol

Consistent with the objectives of the study there were three experiments. Initially, in order to determine the alteration of the stimulus as a function of ITD and time-intensity trading, psychoacoustic experiments were performed. The subjects identified on a polar chart the location of the sound image, presented through the earphones, in the head. The responses were evaluated with 0 degrees representing the midline and 90 degrees representing complete lateralization to one side and averages were taken across subjects. In the second experiment the derived FFR-BIC was evaluated as a function of ITD. In the third experiment the derived FFR-BIC was evaluated under experimental conditions where in the lateralized image due to ITD (as determined by the psychoacoustic experiment) was

gradually shifted to the midline using IID favoring the lag ear. The experimental protocol for each experiment is described below.

Experiment 1: Psychoacoustics

Subjects were asked to identify on a polar chart the location of a fused image within the head under the following experimental conditions:

1. Binaural stimulation with ITD set at 0, 0.4, 0.8, and 1.0ms.
2. Binaural stimulation with the ITD set at 1.0 ms, while presenting IID values of +3, +6, +9, and +12 dB favoring the lag ear (i.e., the ITD and IID cues were presented in opposition to gradually move the fused image to the midline).

Experiment 2: Effects of ITD on the FFR-BIC

FFRs were obtained under the following experimental conditions:

1. FFR to monaural stimulation of the right ear at 65 dB HL.
2. FFR to monaural stimulation of the left ear at 65 dB HL.
3. FFR to binaural stimulation with ITD values of 0, 0.2, 0.4, 0.6, 0.8, 0.9, 1.0, and 1.2 ms.

The 0-1.2 ms ITD range was chosen because lateralization of the tone burst from midline to one ear is complete within this range of ITD values as determined behaviorally from the first experiment. It should be noted that although lateralization is complete by a ITD value of about 1.0 ms, a longer ITD was included in the protocol to examine the FFR-BIC under conditions where the fused image of the stimulus begins to breakdown.

Experiment 3: Effects of trading intensity for time on the FFR-BIC

FFRs were obtained under the following experimental conditions:

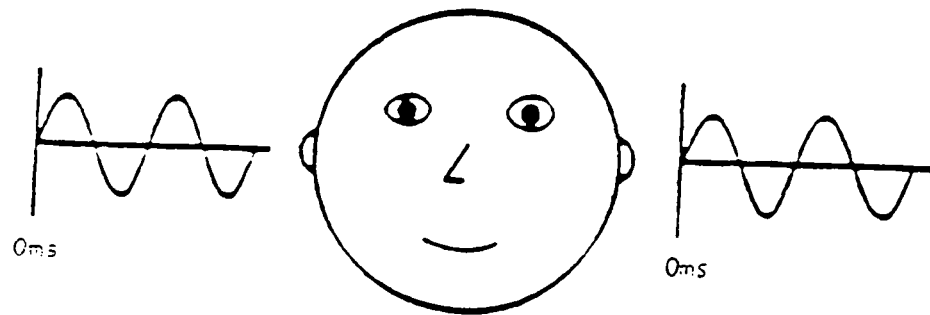
1. With the ITD value of 1.0 ms (the point of complete lateralization), FFR to binaural stimulation while presenting IID values of +3, +6, +9, and +12 dB favoring the lag ear (i.e., the ITD and IID cues were presented in opposition to gradually move the fused image to the midline).
2. FFR to monaural stimulation of the right ear with IID values of +3, +6, +9, and +12 dB.

The 9 dB range of IID, presented in 3 dB steps, was chosen because the preliminary experiment revealed that this range of IID values was sufficient to move the lateralized image (due to the ITD) back to the midline.

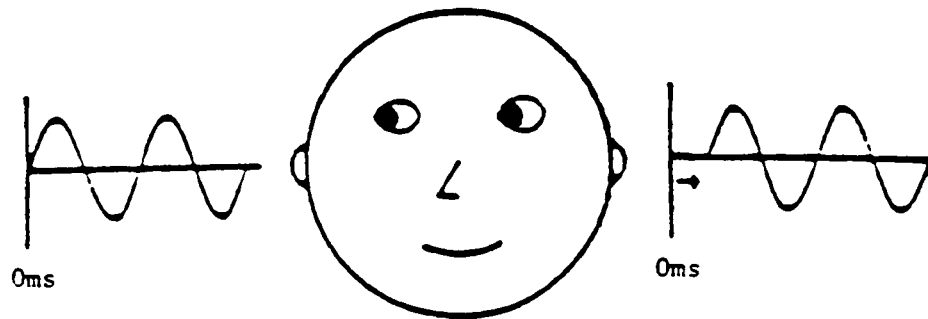
Figure 1 shows these lateralization paradigms.

A. The interaural time and intensity cues are equal at each ear

19



B. Interaural time difference cue



C. Time-intensity trading

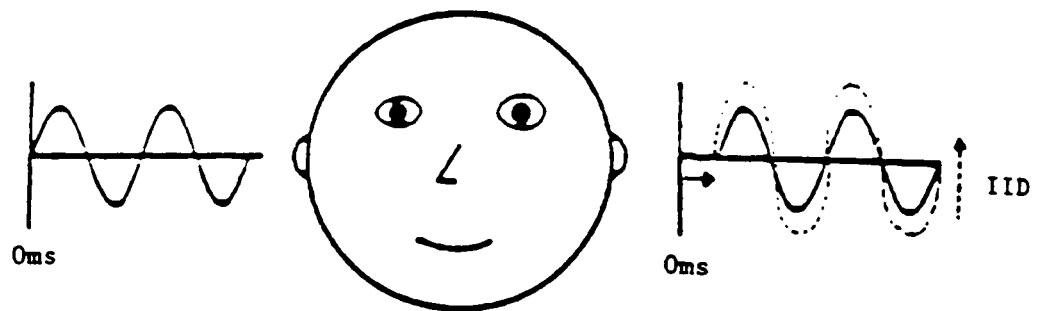


Figure 1. Illustration of the lateralization paradigm used in this study.

CHAPTER IV

RESULTS

Sound Lateralization: Psychoacoustic Data

Effects of ITD

The intracranial location of the fused image of the binaurally presented 500 Hz tone burst stimuli is plotted as a function of interaural time difference (ITD) in Figure 2. It is clear from this figure that as ITD increases the fused image lateralizes from the midline towards the lead ear in a predictable manner. Also, greater shifts in location of the image occurs for ITD values less than 0.8 ms with little or no further lateralization thereafter. It should be noted that at complete lateralization the azimuth of the fused image is about 83 degrees re midline suggesting that the image does not completely lateralize to the lead ear.

Effects of trading ITD with IID

The location of the sound image is plotted as a function of interaural intensity difference (IID) in Figure 3. Recall that in this experiment IID values are presented for a binaural stimuli that is already lateralized to one ear using an ITD of 1.0 ms. Thus, increasing the IID will shift the fused image back to the midline. As can be seen in Figure 3, the lateralized image does indeed shift back towards the midline as IID is increased and is very close to midline at an IID of 12 dB. Steeper shifts towards the midline occurs for IID values between 6 and 12 dB with smaller shifts for IIDs below 6 dB.

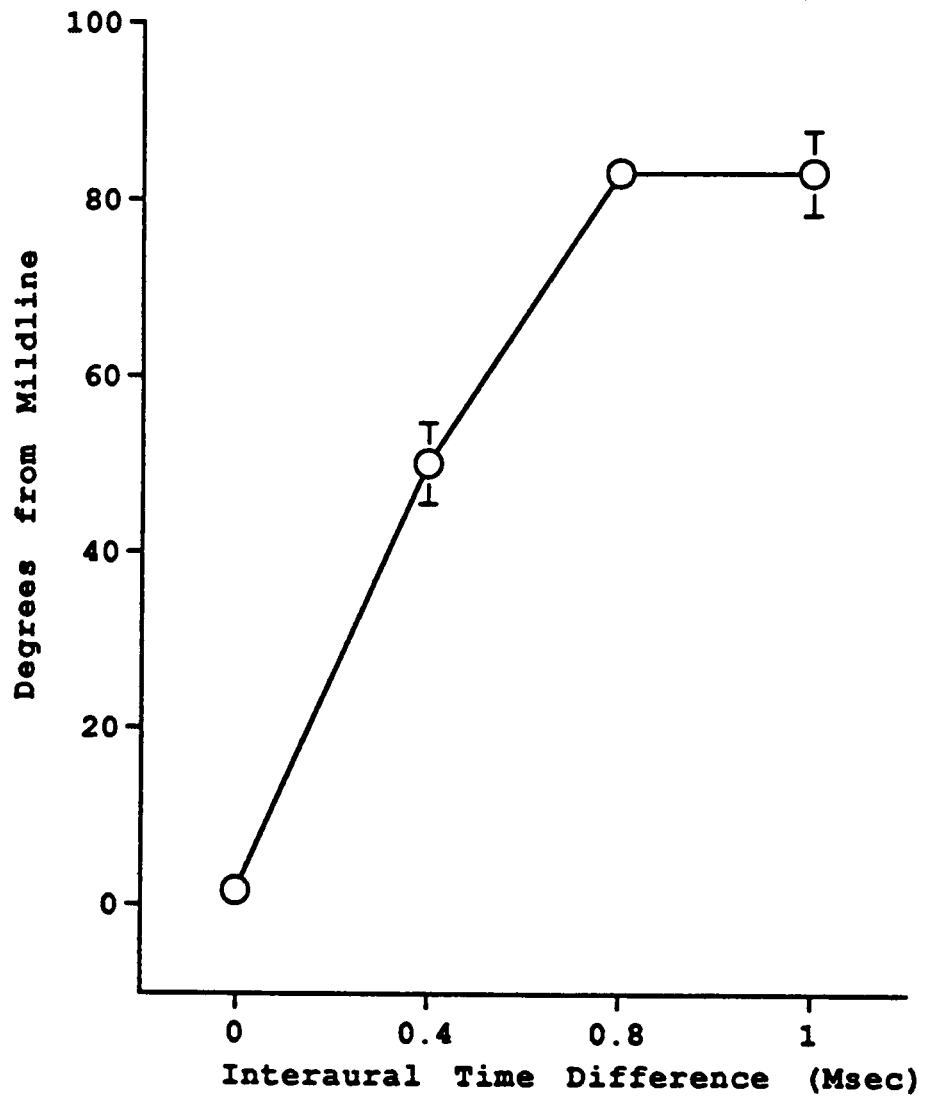


Figure 2. Psychoacoustic sound lateralization using the ITD cue.

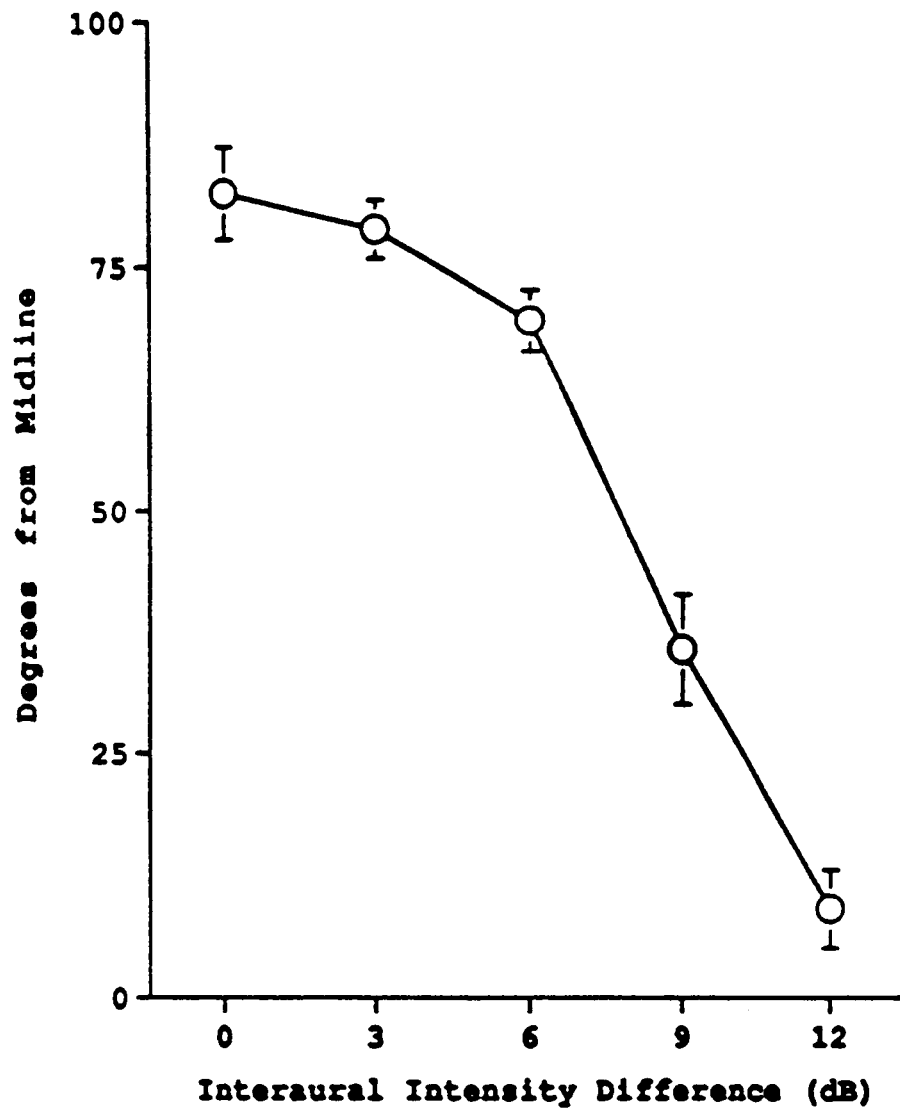


Figure 3. Psychoacoustic sound lateralization using the ITD cue for time-intensity trading.

Sound Lateralization: Human FFR Data

Effects of ITD on the FFR

The nature of the alterations in the amplitude of the FFRs obtained with monaural and binaural stimulation were compared to evaluate the effects of ITD on these responses. The FFRs from one subject representing the summed monaural and the binaural responses are plotted as a function of ITD in Figure 4. Upon comparison of the monaural response (Left Panel) and the binaural response (Center Panel), it can be seen that the FFR amplitudes for both of these responses decrease with increasing ITD. While the amplitude of the monaural responses seem to decrease monotonically, the binaural response appears to show appreciable amplitude decrement starting at only about 0.4 ms. Also, it should be noted that the amplitude of the summed monaural response tended to increase for ITD values 0.8 ms and greater. In addition, the monaural responses tend to show multiple peaks for ITD values greater than about 0.6 ms. The FFR-BIC component (Right Panel) also showed amplitude decrement with increasing ITD (although relatively smaller amplitude change with ITD in comparison to the summed monaural and the binaural FFRs) up to about 0.8 ms. For ITD values 0.8 ms and greater the FFR-BIC appears to show an unexpected amplitude increment. These findings are more clearly represented in spectral data displayed in the three panels of Figure 5.

The median amplitude change as a function of increasing ITD for the summed monaural, binaural, and the FFR-BIC responses are plotted in Figure 6. The summed monaural response (Top Panel) shows a clear amplitude reduction as ITD is increased up to an ITD value of 0.8 ms. For ITD values greater than 0.8 ms, the summed monaural

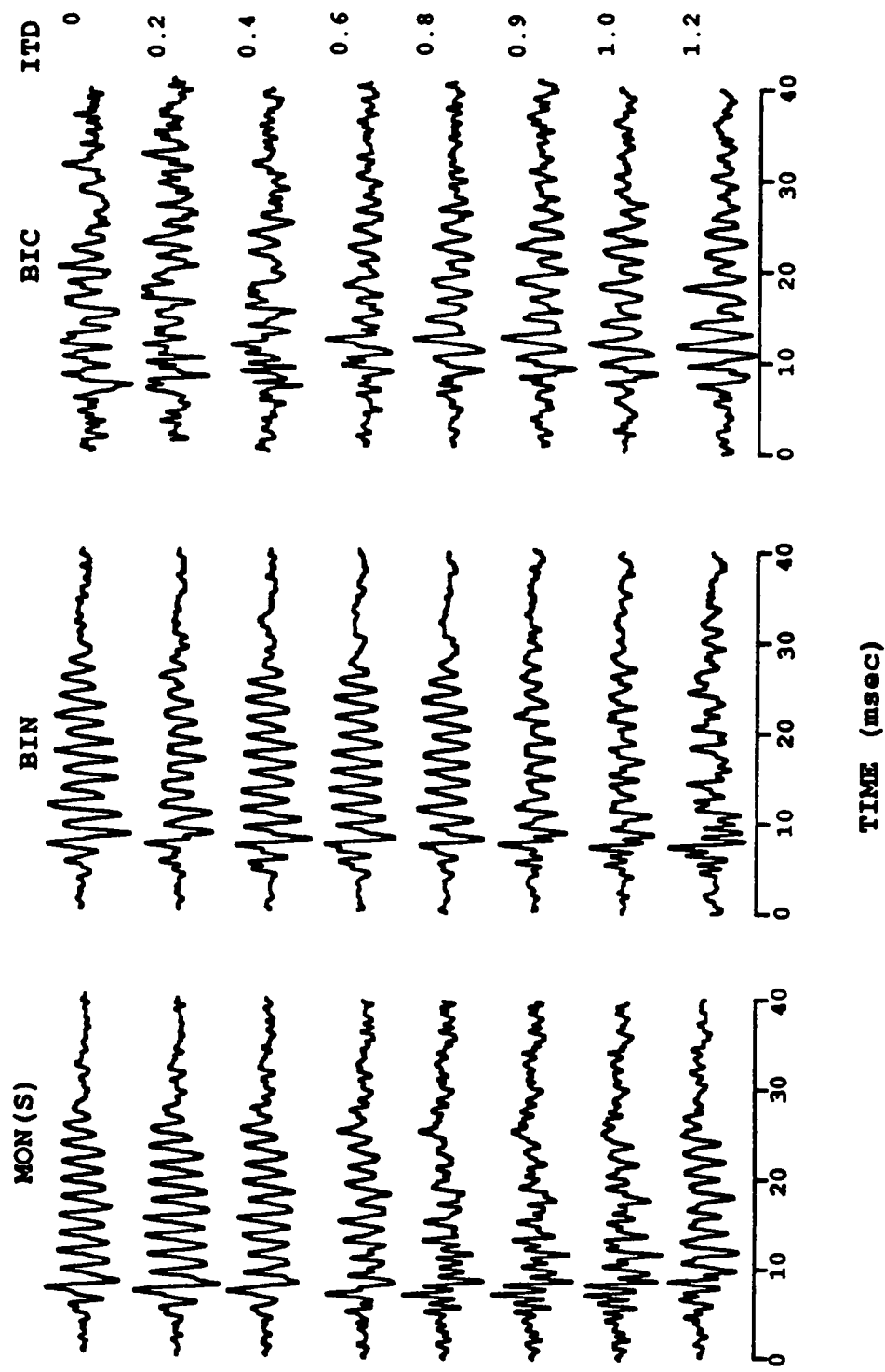


Figure 4. FFR waveform data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of ITD.

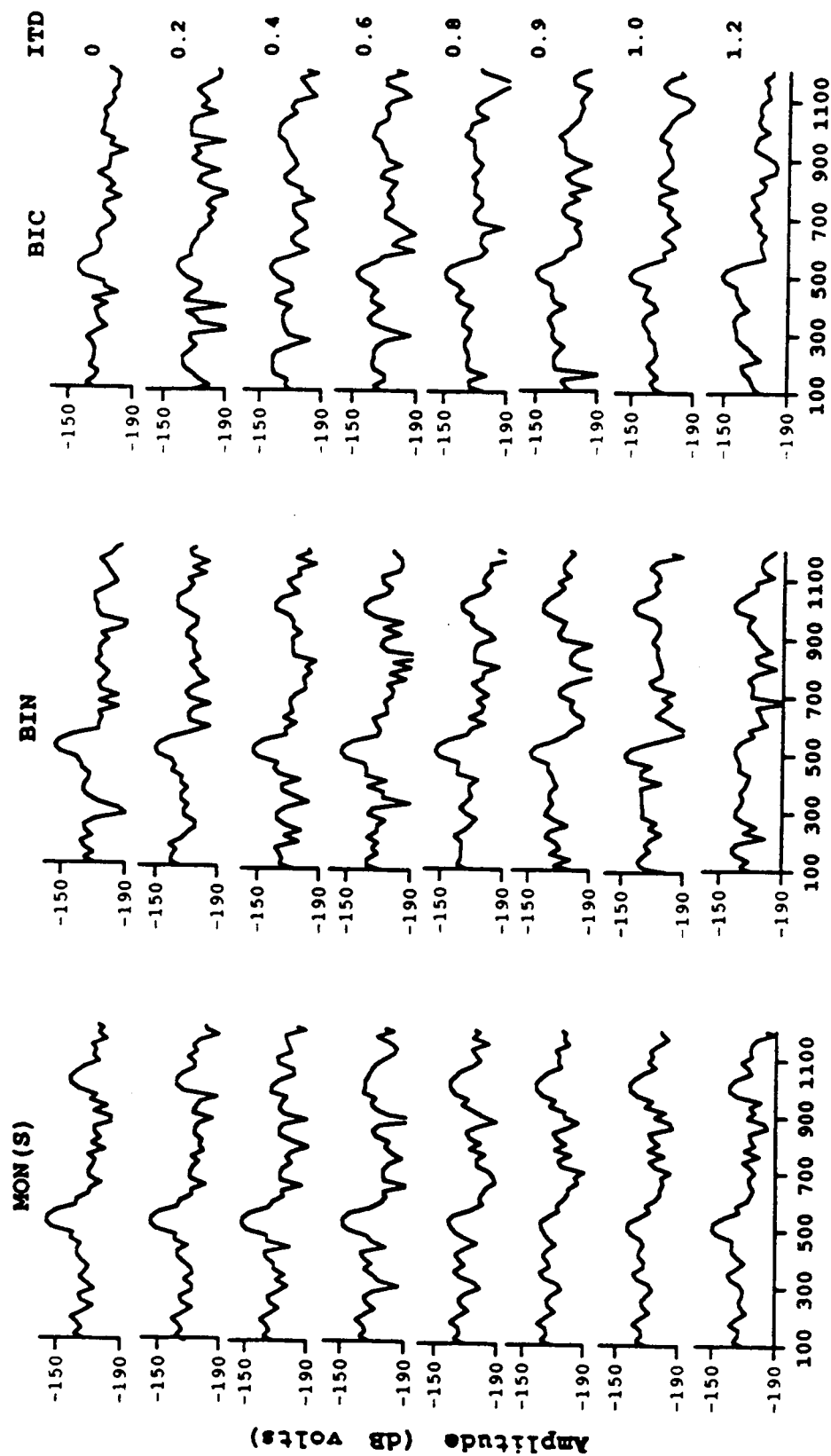


Figure 5. FFR spectral data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of ITD.

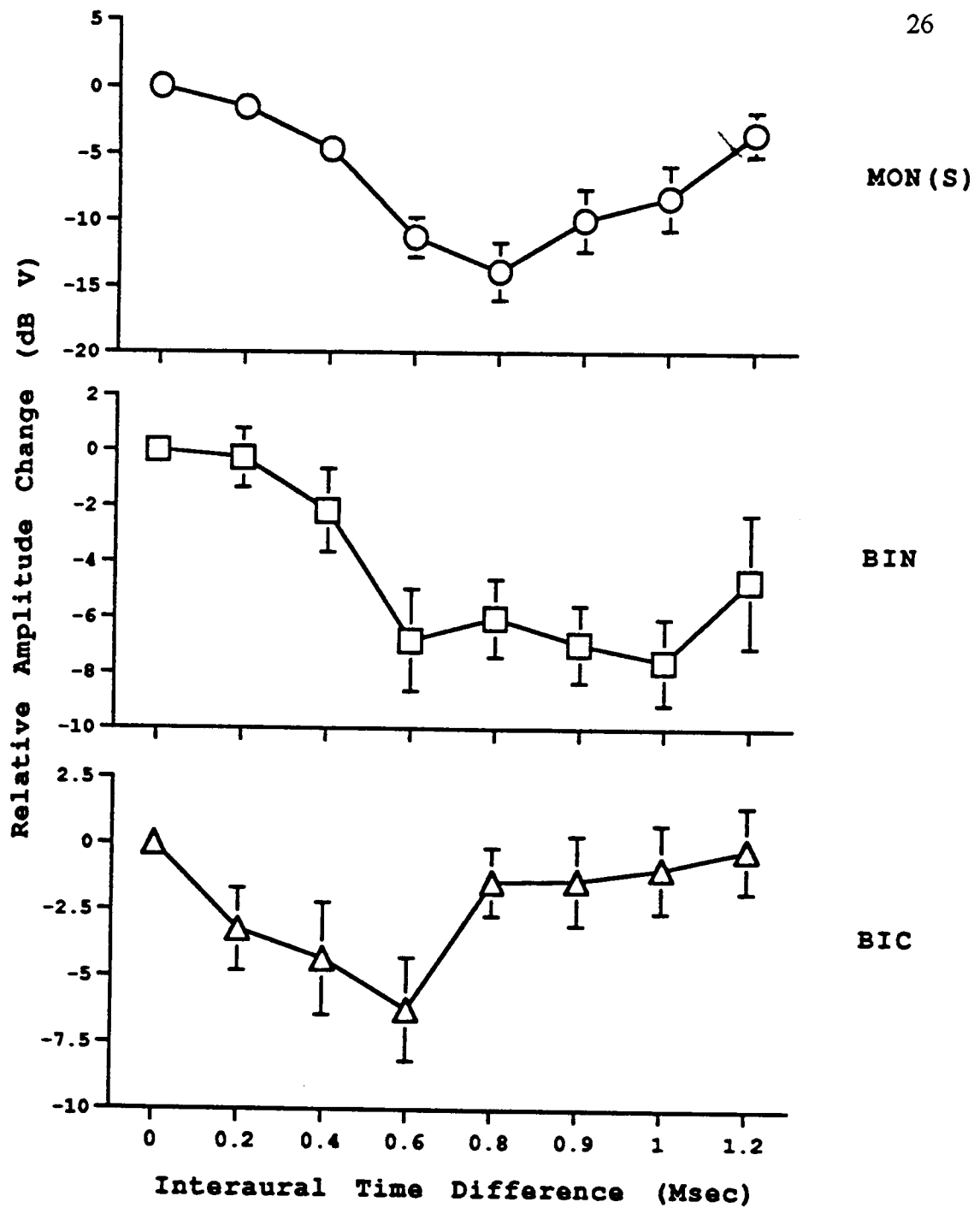


Figure 6. Median amplitude change across subjects representing MON(S) (top panel), BIN (middle panel), and BIC (bottom panel) as a function of ITD.

response tends to increase in amplitude relative to the amplitude at an ITD equal to 0.8 ms. Viewed differently, the magnitude of amplitude reduction for the summed monaural response gets smaller as ITD is increased above 0.8 ms. However, it should be noted that the amplitude of the summed monaural response never exceeds the amplitude of the summed monaural response at an ITD of 0 ms.

The middle panel of Figure 6 shows median amplitude change as a function of ITD for the binaural response. It can be seen that the binaural response decreases in a sigmoidal fashion for ITD values up to 0.6 ms. Unlike the summed monaural response, the binaural response amplitude tends to decrease, albeit very gradually, for ITD values above 0.6 ms. Note that the overall magnitude of amplitude change is smaller for the binaural response in comparison to the summed monaural response.

The median amplitude change of the FFR-BIC response is shown in the bottom panel of Figure 6. Like the summed monaural and the binaural response, the FFR-BIC decreases in amplitude for ITD values ranging from 0 to 0.6 ms. However, unlike the binaural response and more like the summed monaural response, the FFR-BIC shows a smaller magnitude of amplitude decrement for ITD values greater than 0.6 ms. In fact, the FFR-BIC amplitude change for ITD values greater than 0.6 ms may be characterized as a gradual amplitude increment. Again, it should be noted that the overall reduction in amplitude for the FFR-BIC, over the ITD values ranging from 0 to 0.8 ms, is smaller than that seen for the summed monaural response but more similar to the amplitude change observed for the binaural response.

Effects of IID (trading for ITD) on the FFR

In this section the results relating amplitude changes in the monaural and the binaural FFR while shifting the ITD lateralized image back to the midline using IID are presented. The summed monaural, binaural, and the FFR-BIC from one subject is plotted as a function of IID in Figure 7. The summed monaural response (Left Panel) appears to grow in amplitude gradually as the IID is increased. The amplitude growth with IID is more apparent for the binaural (Center Panel) and the FFR-BIC (Right Panel) waveforms. The spectral data shown in Figure 8 more clearly depicts the trends described above for the three sets of responses.

The median amplitude change in the FFR spectral peak at 500 Hz is plotted as a function of increasing IID in Figure 9. The amplitude change for the summed monaural response (Top Panel) can best be described as non-monotonic characterized by an initial rapid growth in amplitude followed by a gradual increase in amplitude and as the IID is increased above about +6 dB the amplitude of the response actually decreases. However, note that the amplitude of the response is always greater than the reference condition of 0 dB IID. The relatively prominent error bars, depicting one standard error of the mean, indicates appreciable variability in the data. The amplitude data for the FFR-BIC (Bottom Panel) appears to mimic the summed monaural amplitude function. In contrast, the binaural amplitude function (Middle Panel) shows a systematic increase in amplitude as the IID is increased, although the magnitude of the amplitude change is smaller than the summed monaural response.

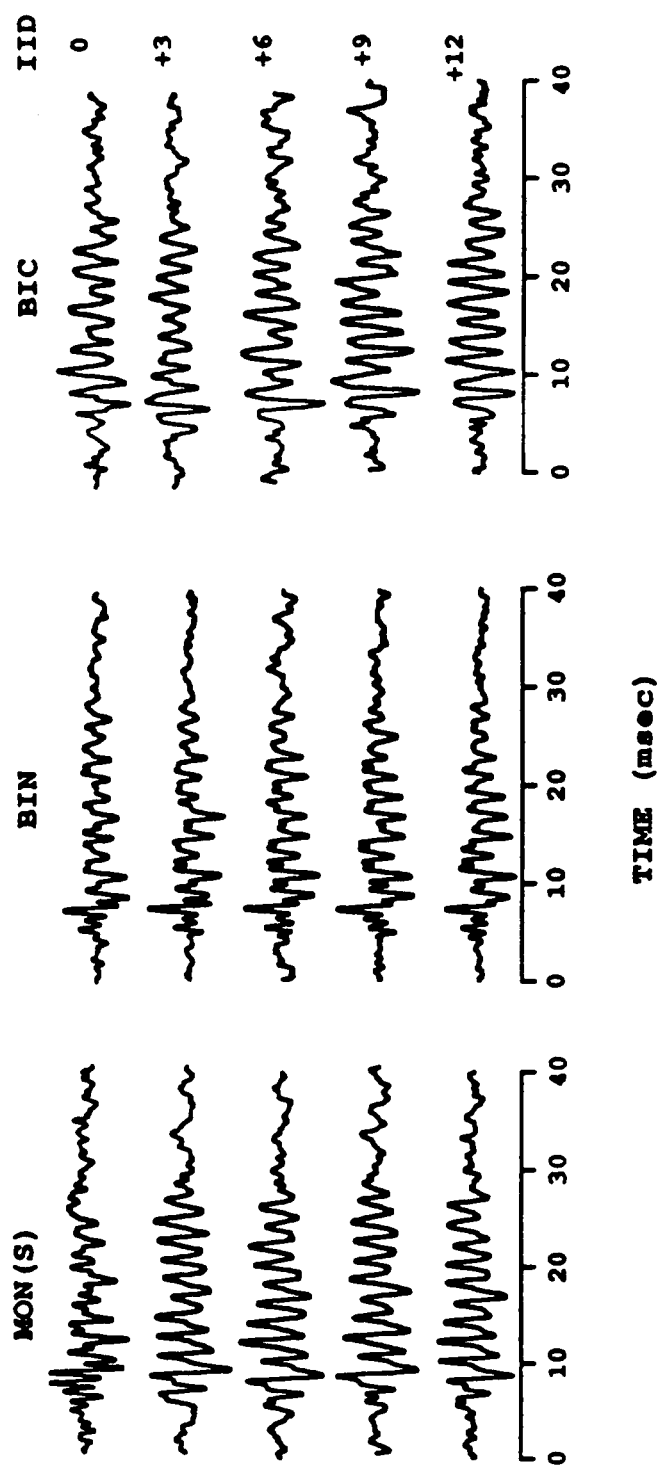


Figure 7. FFR waveform data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of time-intensity trading.

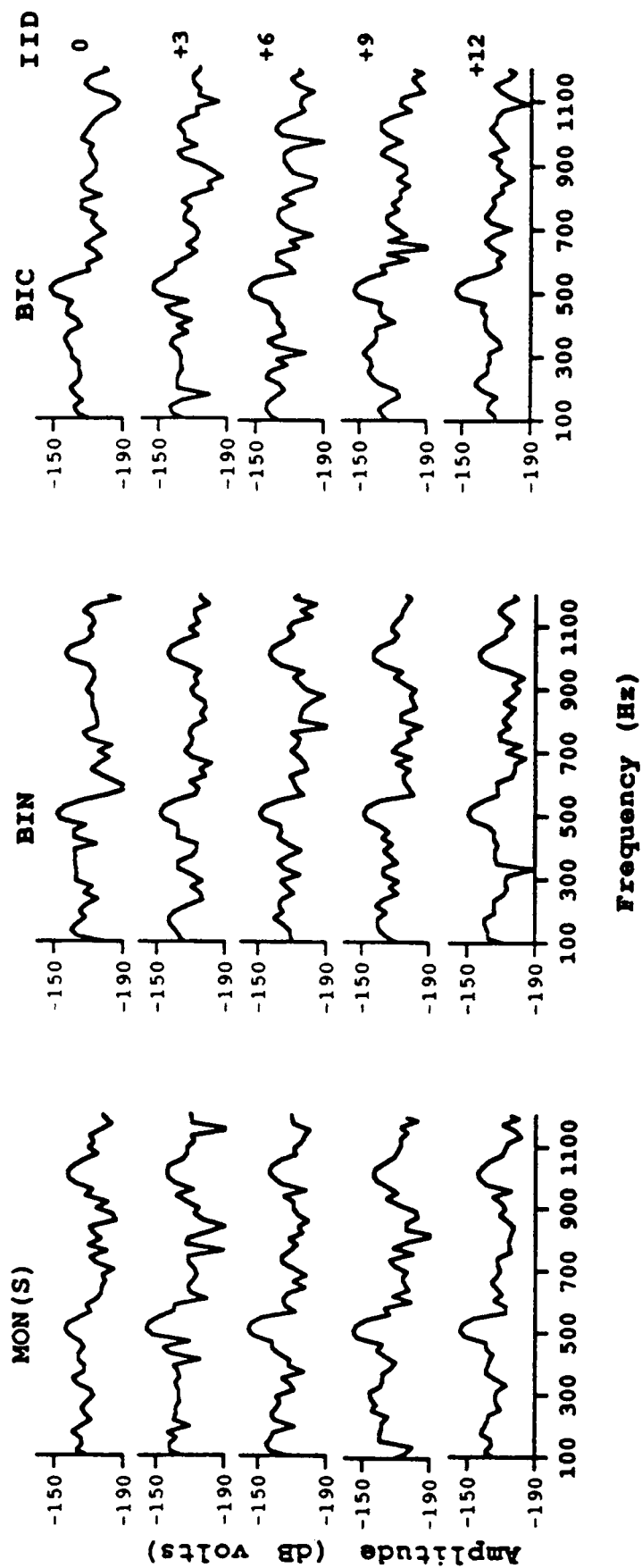


Figure 8. FFR spectral data from one subject representing MON(S) (left panel), BIN (center panel), and BIC (right panel) as a function of time-intensity trading.

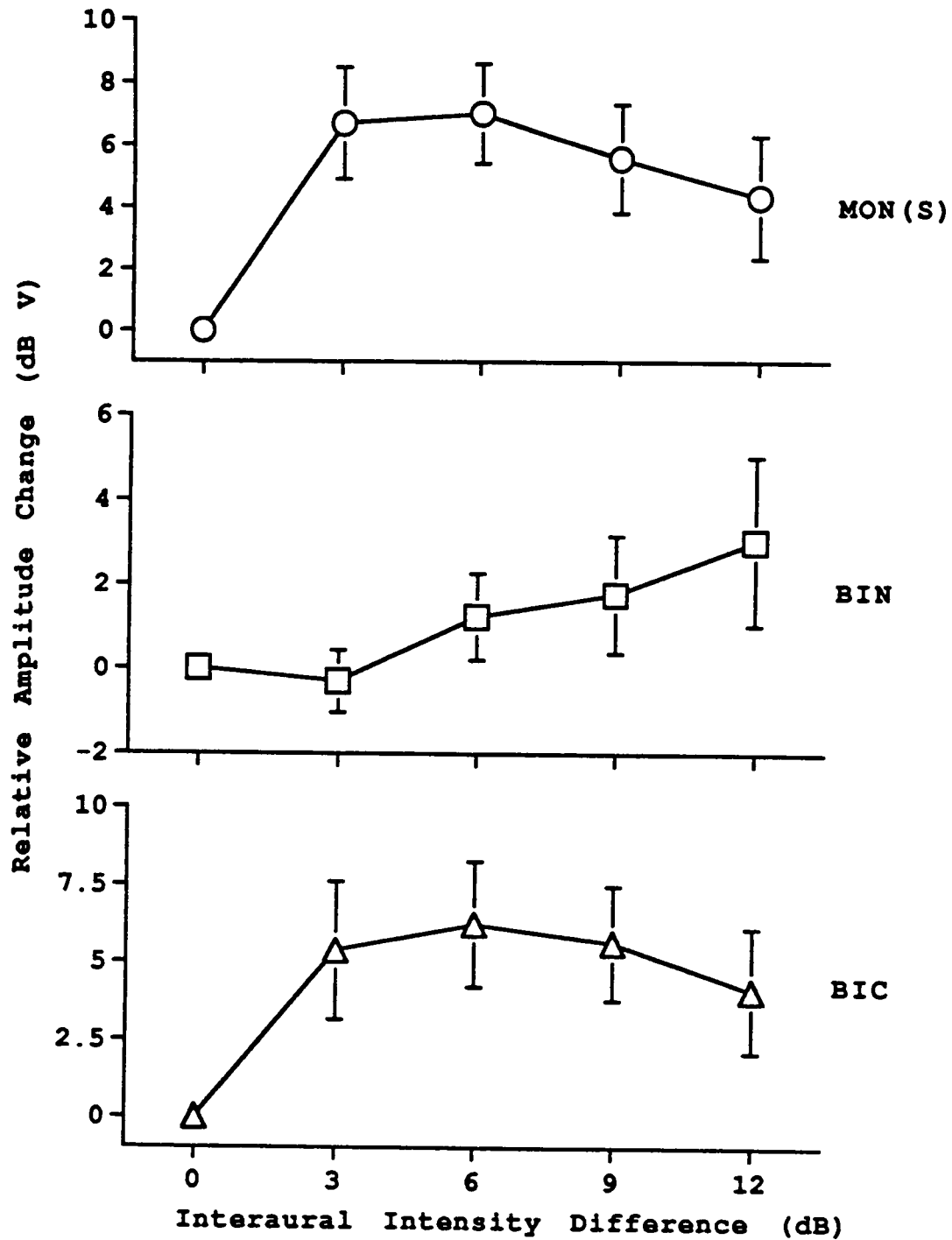


Figure 9. Median amplitude change across subjects representing MON(S) (top panel), BIN (middle panel), and BIC (bottom panel) as a function of time-intensity trading.

Comparison of the Effects of ITD and IID on the FFR

In order to determine if changes in ITD and IID have similar or differential effects on the FFR we compared the amplitude functions for the binaural, and the FFR-BIC components obtained under ITD and IID manipulations. The left panel in Figure 10 compares the median amplitude change in the binaural response when ITD is changed (open circles) with the binaural response when IID is changed (open squares). Note the reference axes for the IID change are to the right and top of the figure. It is clear from this figure that the amplitude changes in the binaural response are different for ITD and IID alterations. Specifically, the overall magnitude of the amplitude change is appreciably greater for the ITD manipulation than the IID. This suggests that the slope of the amplitude change as ITD is increased (when the fused image moves from midline to the lead ear) is greater than the slope of the amplitude change when IID is increased (when the lateralized image shifts back to the midline).

The amplitude change in the FFR-BIC for the ITD (open circles) and the IID (open squares) changes are plotted in the right panel of Figure 10. Again, the amplitude change functions for the FFR-BIC under ITD manipulation is very different from that resulting from IID manipulation. While the ITD amplitude function shows a systematic decrease in the FFR-BIC with increasing ITD the IID function exhibits a non-monotonic amplitude behavior as the IID is decreased.

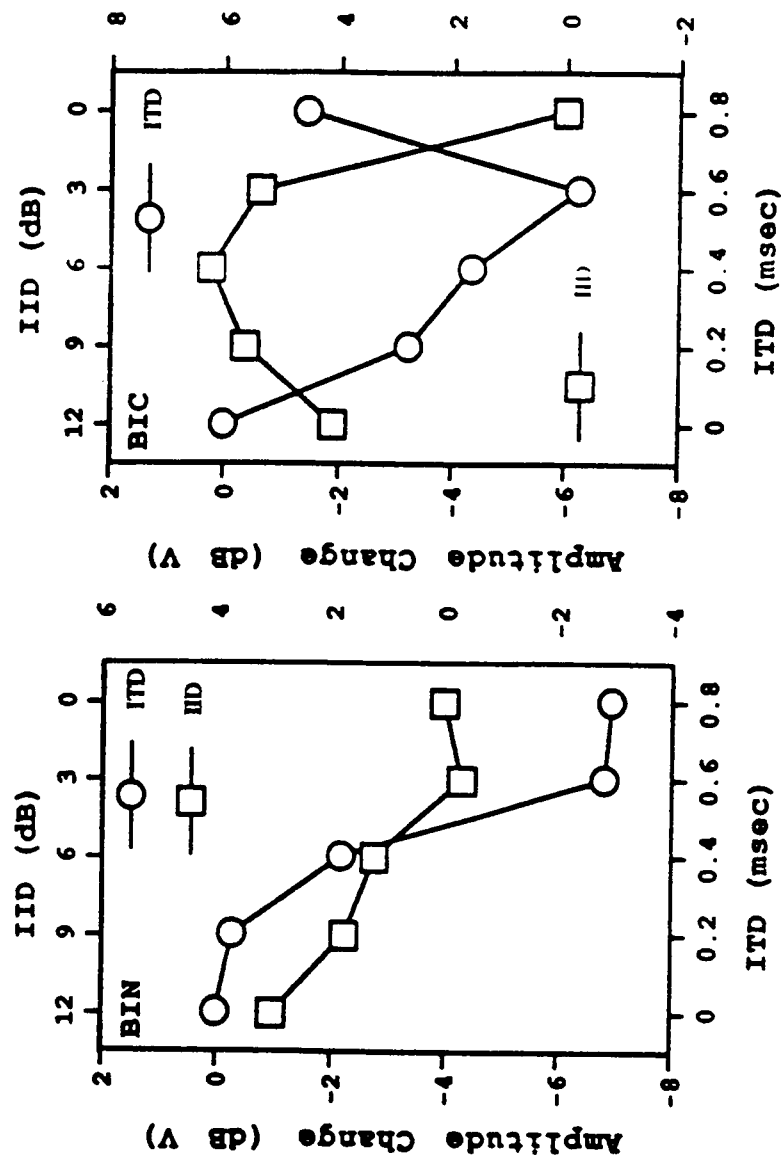


Figure 10. Comparison of changes in BIN and BIC amplitudes due to ITD and IID. IID changes are to the right and top of the figures. ITD changes are to the left and bottom of figures.

Summary of Results

Effects of ITD on the FFR

1. MON(S) responses decrease with increasing ITD up to 0.8 ms but increase for ITD values of 0.8 ms and greater.
2. MON(S) responses possess multiple peaks for ITD values greater than 0.6 ms.
3. With increasing ITD, BIN responses decrease appreciably after 0.4 ms and continues with gradual decrements after 0.6 ms.
4. Like the MON(S) and BIN, the FFR-BIC decreases in amplitude for ITD values ranging from 0 to 0.6 ms.
5. At ITD values greater than 0.6 ms, there is an unexpected increase in amplitude for the FFR-BIC. Like the MON(S), the FFR-BIC shows a smaller amount of amplitude decrement for ITD values greater than 0.6 ms.
6. The overall reduction in amplitude for the FFR-BIC from 0 to 0.8 ms is smaller than that seen for the MON(S) responses and more similar to the amplitude change for the BIN responses.

Effects of IID (trading for ITD) on the FFR

7. The MON(S) response increases in amplitude gradually as the IID is increased (image shifting back towards the midline).
8. The MON(S) has an initial rapid growth in amplitude followed by a gradual increase in amplitude. As the IID is increased above +6 dB the amplitude decreases.

9. The amplitude of the FFR-BIC tends to mimic that of the MON(S). However, the BIN responses show a systematic increase in amplitude as the IID is increased.

Comparison of the effects of the ITD and the IID on the FFR

10. In the BIN responses, although there is a decrease in amplitude, the decrements are different for ITD and IID alterations. The overall amount of amplitude change is appreciably greater for ITD rather than IID.
11. For the FFR-BIC conditions, the ITD amplitude shows a systematic decrease with increasing ITD. The IID exhibits an increase in amplitude as the IID is decreased (moving the image towards the lead ear) to about +6 dB then the amplitude begins to decrease.

CHAPTER V

DISCUSSION

The results of this study relate specifically to the reduction in the amplitude of the binaural and the FFR-BIC components as the ITD is increased and to the increase in the amplitude of binaural FFR and the FFR-BIC components as an increasing IID is imposed on a ITD lateralized sound. The nature of these alterations in the binaural FFR and FFR-BIC components are consistent with the idea of non-linear neural interactive processes within the rostral brainstem mediating these changes. In addition these FFR changes can be correlated with our psychoacoustic results to suggest that the FFR-BIC and the binaural FFR may be related to lateralization of the fused image of a low frequency sound. In the subsequent sections, the ITD data and then the IID data.

Effects of ITD on the Binaural Response.

Results of this study showed that the amplitude of the FFR-BIC and the binaural response decreased as the ITD was increased. While the binaural response, in particular, remaining unchanged for ITD values larger than 0.8 msec, the FFR-BIC showed a reversal in amplitude for ITDs larger than 0.6 msec. The reduction in the magnitude of the binaural and the BIC with increasing ITD have been reported for the click evoked ABR (McPherson and Starr, 1995; Jones and Van der Poel, 1990; Furst et al., 1985; Prasher et al., 1981; Dobie and Berlin, 1979;).

Evoked potential binaural interaction, like neural interaction in single units, are

commonly explained using models of binaural interaction. Models of binaural interaction based on psychoacoustics and physiology (Colburn and Durlach, 1978) assume that sounds are presented to a central binaural interaction processor following processing of monaural inputs separately at the periphery. The processor acts either as a correlation device or as a coincident counter.

In order to explain the decrease in the BIC as function of increasing ITD, Furst et al. (1985) used a binaural interaction processor model with the following specifications: (1) the processor produces an output only when the inputs from the two sides arrive within about 1.0 ms; and (2) the output level of this processor is proportional to its average input level from the two ears. The reduction in the binaural response in particular and to an extent the FFR-BIC over an ITD range of about 0.8 msec is consistent with this model which would predict reduced output as ITD is increased because the average input level to this processor is altered by the interaural time disparity. However, the reversal in the amplitude for the FFR-BIC at about 0.6 to 0.8 msec is not consistent with the predicted output of this model. We believe that the FFR-BIC amplitude reversal for ITD values 0.6 msec and above reflects primarily the characteristics of the monaural summed response. That is, the algebraic resultant FFR produced by adding two monaural sustained periodic responses that have multiple components (e.g. FFR1 and FFR2) that are not time aligned (due to the artificial time shift applied to response from one ear before summation of the monaural responses) could show greater amplitude at ITDs where the components of the two monaural responses are in phase. Since the binaural interaction is derived by subtracting the binaural response from the summed monaural response it is not

surprising that the amplitude of the derived FFR-BIC actually shows a reversal at ITDs where the summed monaural response is larger. The fact that the binaural response in our data is reduced in amplitude and does not change appreciably for ITDs greater than about 0.8 msec lends further support to this possibility.

More recently a two-level decision making model has been proposed by Furst et al. (1995). This model is illustrated in Figure 11. The model suggests that in the first level, each superior olivary complex (SOC) receives inputs from both cochlear nuclei (a schematic diagram of the model is shown in Figure 11). Independent level and time comparators exist in each SOC whose outputs in turn project independently to the next level (for our discussion we will assume that the higher level is the rostral brainstem). The rostral brainstem then uses the four possible inputs from the two SOC's to determine the location of the fused image of the sound.

In the case of a dichotic signal with no interaural disparities the outputs from both ITD and IID channels from each SOC will project to the rostral brainstem. Since there is symmetrical outputs from the two SOC's, the rostral brainstem will place the source to be located in the center by virtue of neural interaction among neurons that are in a specific spatial location. As ITD is increased, (such that the left ear leads-as was the case in our study) the output of the time comparator from the left SOC is reduced and is absent for large ITDs. Thus only the time comparator output from right SOC and the level comparator output from both SOC's actively project to the rostral brainstem (because IID=0). The change in both the number of channels and the nature of the outputs from the two SOC's (as a result of the ITD) will produce an asymmetrical input to the rostral

TWO-LEVEL DECISION-MAKING MODEL for CLICK LATERALIZATION

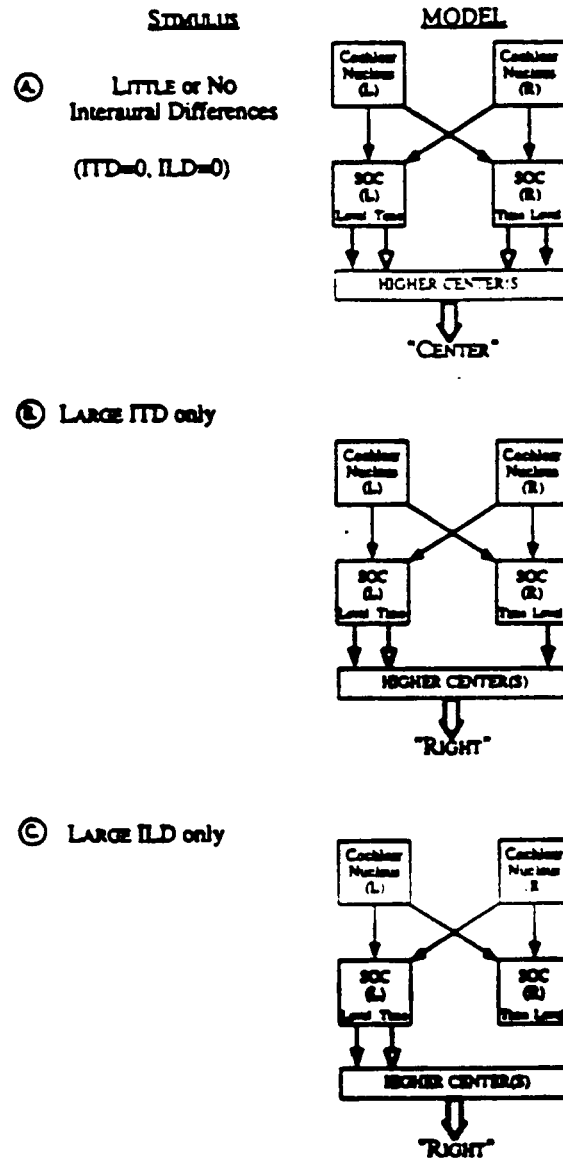


Figure 11. A schematic diagram of the two-level decision making model for lateralization proposed by Furst et al. (1995).

brainstem. The nature of the asymmetry will place the source to the left side by virtue of a spatial shift in the place of neuronal interaction in the rostral brainstem.

According to this model then the decrease in the binaural FFR and the FFR-BIC with increasing ITD reflects both an increase in the asymmetry and a decrease in the number of outputs from the two SOC's. Thus, reduction in amplitude can be interpreted to mean (i) increased inhibition due to increased asymmetrical inputs to the rostral brainstem; or (ii) decreased excitatory inputs without change in inhibition.

The FFR Binaural Interaction Component

Binaural interaction in the brainstem has been documented in the auditory evoked response (Dobie and Berlin, 1979; Dobie and Norton, 1980; Parthasarathy and Moushegian, 1993; Wrege and Starr, 1981) and the 500 Hz tone burst (Parthasarathy and Moushegian, 1993; Hink et al., 1980)). Consistent with the presence of a BIC demonstrated in this study and previous studies a binaural response is not a simple algebraic sum of two independent responses. The BIC was derived by subtracting the binaural response from the sum of two independent monaural responses. Our results show a reduction in amplitude of the binaural response and is attributed to complex neural interaction among binaural neurons within the brainstem. If this neural interaction did not occur then the sum of two monaural responses added together would equal a binaural response. Specifically these binaural neurons are classified as EE neurons, excitation occurring from stimulation of either ear, or EI neurons, excitation occurring from stimulation of one ear and inhibition resulting from stimulation of the other ear. The

effects of inhibition, due to a decrease in the neural firing rate, causes reduced binaural responses in comparison to monaural responses which receive only excitatory inputs. The binaural interaction demonstrated in this study was altered by ITDs and IIDs.

Effects of Trading ITD with IID on the Binaural Frequency Following Response

The results of this experiment showed that the binaural FFRs and the FFR-BIC showed an increase in amplitude with increasing IID. However, it should be noted that the amplitude change for the FFR-BIC was minimal for IID values greater than 6 dB. Similar results have been reported for both, the ABR-BIC component (McPherson and Starr, 1995; Jones and Van der Poel, 1990; Furst et al., 1985; Prasher et al., 1981; Dobie and Berlin, 1979) and the FFR-BIC component (McDaniel, 1994).

It should be pointed out that this IID experiment is somewhat different in that IID values are used to move an image back to midline that has already lateralized to one ear by an ITD of 1 msec. Thus, unlike the other experiments where the BIC would decrease (as the fused image would move from the midline towards one ear) as IID is increased, Furst's binaural processor model would predict the behavior observed for our binaural FFR and the FFR-BIC. The second specification of the processor in particular (i.e. the output level of the processor is proportional to its average input level from the two ears) will account for our data since increasing IID would increase the average input level. Our results showing complete lateralization by an IID of 12 dB satisfies the first specification and would ensure that the two inputs to the processor is within 1.0 msec (A 30 dB IID would be equivalent to a 1 msec difference).

Our results for the IID experiment can also be explained by Furst's two-level decision making model. When the image is lateralized to one ear (left ear in our case) with an ITD of 1.0 msec only one time output from the contralateral SOC (left) is actively projected to the rostral brainstem along with the two level outputs from each SOC. The decision maker places the image to be towards left side because of the asymmetrical outputs from the SOC. Now, when the IID is introduced on top of this to move the image to the midline, you introduce a gradually increasing time and level outputs from the right SOC such that when the image is centered the outputs of the two SOC's are essentially symmetrical. Thus, by virtue of the symmetrical outputs the decision maker would place the image at midline. In this case symmetrical outputs probably reflects both increased excitation and a reduction in inhibition.

Finally, comparison of the ITD and IID data indicated that the binaural FFR and the FFR-BIC behaved differently under these two conditions. Specifically, a relatively larger amplitude change was observed with ITD manipulation than with IID. Also, the FFR-BIC for the two manipulations showed different amplitude behavior. It is possible that these differences in the binaural FFR and the FFR-BIC for ITD and IID changes reflect independent and different underlying mechanisms mediating ITD and IID changes. The two-level decision model proposed by Furst (1995) would support this idea.

Anatomical Locus of Binaural Interaction

While the anatomical locus of this processor is not certain it would appear that this interaction phenomenon reflects events proximal to the neural elements generating the

human FFR-presumably the inferior colliculus. There is evidence to support not only the presence of binaurally sensitive neurons in the inferior colliculus (IC) but also the possibility that the human FFR is generated at this level.

The MSO is the first nucleus on the ascending auditory pathway to receive bilateral inputs and therefore, represents binaural auditory processing (Moushegian et al., 1975). Buchwald and Huang (1975), Fullerton and Hosford (1979), Gardi and Berlin (1981), and Levine et al. (1993) report that when the crossing fibers of the trapezoid body are lesioned in the brainstem there is a loss of binaural interaction in the brainstem potentials. Also, lesion studies in animals have shown these lesions to produce an inability in the animals to use the ITD binaural cue for behavioral judgements (Diamond and Neff, 1957; Diamond et al., 1962).

Wada and Starr (1989) report that when the trapezoid body was sectioned in the guinea pig that the later P4 and N4 (which have similar generators as waves V-VI in human brainstem potentials) were lost and the decrease in the BIC was related to the number of fibers sectioned. Complete sectioning of the MSO did not produce a BIC at P4 and N4. Complete loss of the BIC for N4 occurred for bilateral sectioning of the LL. However, very little effect if any was seen for unilateral sectioning of the LL or the IC.

Correlation of FFR Results with Psychoacoustic Results

Another aspect of interest to us was the relationship between the electrophysiologic FFR-BIC measure and the perceived location of the fused image as the ITD and IID are varied. Similar to our observation, Furst et al., (1985); and McPherson

and Starr (1995) observed that as ITD increased (the fused image moved toward the lead ear), the BIC amplitude decreased. Furthermore, they observed that whenever a BIC was present the binaural click stimuli was always perceived as a fused image and whenever the image was not fused, the BIC was not present. Based on these observations these authors concluded that the BIC and binaural fusion are related and that the BIC was sufficient to code for binaural fusion. Our psychoacoustic results appear to support this in that the FFR-BIC and the binaural response decreased in amplitude as the image was lateralized toward the lead ear. Smallest amplitude for the binaural response and the FFR-BIC were associated with ITD values of 0.8 and 0.6 msec, respectively suggesting a good inverse correlation between binaural response magnitude and complete lateralization.

Potential Limitations of this Study

Two limitations of this study that might account for the BIC is monaural asymmetry and acoustic crosstalk. It is assumed that if the two independent monaural responses are symmetrical and they are subtracted one response from the other, a flat line will result indicating no difference between the responses (Spivak and Seitz, 1988). However, differences between the two responses occur (Ballachanda, Rupert, and Moushegian, 1994) and these differences affect the BIC (Decker and Howe, 1981; Spivak and Seitz, 1988). If binaural stimulation is not processed symmetrically along the auditory pathway, it is questionable whether the interaction can be attributed to neural processing or the product of this asymmetrical processing of stimuli (Decker and Howe, 1981). In the present study and a previous study (McDaniels, 1994) performed in this laboratory,

asymmetry in amplitude and less often, morphology was noticed. In the previous study the left monaural response was subtracted from the right monaural response as described by Spivak and Seitz (1988). The result showed that the amplitude differed from that of the difference trace representing the monaural conditions. It was assumed that the BIC was not influenced by asymmetry because if this was the case, it would seem that the binaural response would have reflected these differences. The second limitation pertains to that of acoustic crosstalk. Acoustic crosstalk occurs when the monaural stimulation presented to the ipsilateral ear is intense enough to stimulate the contralateral ear. As a result, the monaural condition cannot be accurately predicted because the monaural response would actually be a sum of the ipsilateral ear and a small input from the contralateral ear. As a result, the interaction would be a product of an acoustic crosstalk response even if no binaural interaction existed (Ainslie and Boston, 1980). The effects of acoustic crosstalk was not investigated in the present study. Evidence has been established that rules out this possibility (Dobie and Berlin, 1979; Dobie and Norton, 1980; Dobie and Wilson, 1985). The use of masking may be used to eliminate acoustic crosstalk. If the BIC is a result of acoustic crosstalk, then the interaction component obtained with masking should differ from that obtained without masking. Dobie and Wilson (1985) recorded auditory brainstem potentials with and without masking and demonstrated that the effects of masking did not alter the ABR-BIC. They concluded that the BIC was not simply a product of acoustic crosstalk.

Implications

If the BIC and its behavior as a function of ITD reflects neural processes related to lateralization/localization, then this measure can be used clinically to assess the functional integrity of the brainstem. The midline condition of 0 ITD and the lateralized condition of 1.0 ms would be sufficient to use since lateralization (83 degrees) was achieved by this point. This measure could be added to the test battery of evaluating central auditory processing disorders (CAPD). The pathway of the central auditory processing mechanism extends from the pontomedullary junction of the brainstem to auditory projections throughout the brain (Musiek, 1985). Along this pathway are the structures which process binaural information (Rosenweig and Wyers, 1955; Wernick and Starr, 1968). Furthermore, since binaural fusion is related to the presence of the BIC, assessing of binaural fusion in the clinical setting may also be possible. Theoretically, binaural processing in the brainstem may be assessed by using this FFR paradigm.

The possibility of monaural asymmetry as a function of ITD requires further investigation. In a previous study, the midline condition was used to determine the presence of asymmetrical responses. In this study, the presence of monaural asymmetry was not evaluated. However, only at the midline conditions could this activity be evaluated because at other stimulus conditions asymmetry would exist as a result of different time and intensity level presented to each ear.

Conclusions

The general objective of this study was to determine if there was an electrophysiologic correlate of lateralization as reflected in the FFR. More specifically, this study examined whether there was a systematic alteration in the FFR-BIC as a function of ITD. Derivation of the FFR-BIC was achieved by summing two monaural responses (right and left ear) and subtracting the binaural response from the summed response. This difference was referred to as the BIC and is a result of overlapping neural populations. If neural interaction did not occur then the difference between the summed monaural and the binaural responses would have been a straight line, meaning no interaction would have been present. This was not demonstrated in our study. Rather, the BIC reflected a difference between the summed monaural and the binaural response as well as an electrophysiologic correlate of lateralization reflected in the human FFR.

REFERENCES

Ainslie, P. J., and J. R. Boston. 1980. Comparison of brainstem auditory evoked potentials for monaural and binaural stimuli. Electroencephalography and Clinical Neurophysiology 49: 291-302.

Arslan, E., S. Prosser, and S. Michelini. 1981. The auditory brainstem response to binaural delayed stimuli in man. Scandinavian Audiology 10: 151-55.

Ballachanda, B. B., A. Rupert, and G. Moushegian. 1994. Asymmetric frequency-following responses. Journal of the American Academy of Audiology 5: 133-37.

Berlin, C. I., L. J. Hood, and P. Allen. 1984. Asymmetries in evoked potentials. In: Hearing Science: Recent Advances, C. I. Berlin (Ed.). (pp. 461-77). San Diego: College-Hill Press.

Boudreau, J. C., and C. Tsuchitani. 1967. Binaural interaction in the cat superior olive S segment. Journal of Neurophysiology 31 (3): 442-54.

Buchwald, J. S., and C. M. Huang. 1975. Far-field acoustic response: origins in the cat. Science 189: 382-384.

Colburn, H. S., and N. I. Durlach. 1978. Models of binaural interaction. In Handbook of Perception, Vol. IV, Hearing, E. C. Cartarette and M. P. Friedman (Eds.) New York: Academic.

Decker, T. N., and S. W. Howe. 1981. Auditory tract asymmetry in brainstem electrical responses during binaural stimulation. The Journal of the Acoustical Society of America 69 (4): 1084-90.

Diamond, I. T., and W. D. Neff. 1957. Ablation of temporal cortex and discrimination of auditory patterns. Journal of Neurophysiology 20: 300-15.

Diamond, I. T., J. M. Goldberg, and W. D. Neff. 1962. Tonal discrimination after ablation of auditory cortex. Journal of Neurophysiology 25: 223-35.

Dobie, R. A. 1982. Binaural interaction in auditory brainstem responses: theoretical and methodological considerations. The Journal of the Acoustical Society of America 71: 1031-33.

Dobie, R. A., and C. I. Berlin. 1979. Binaural interaction in brainstem evoked responses. Archives of Otolaryngology 105 (July): 391-98.

Dobie, R. A., and S. J. Norton. 1980. Binaural interaction in human auditory

evoked potentials. Electroencephalography and Clinical Neurophysiology 49: 303-13.

Dobie, R. A., and M. J. Wilson. 1985. Binaural interaction in auditory brainstem responses: effects of masking. Electroencephalography and Clinical Neurophysiology 62 (1, January): 56-64.

Erulkar, S. C. 1959. The responses of single units of the inferior colliculus of the cat to acoustic stimuli. Proc. Royal Soc. (London) 150: 336-55.

Erulkar 1975. Physiological studies of the inferior colliculus and medial geniculate complex. In Handbook of Sensory Physiology, Vol. II, W. D. Keidel and W. D. Neff (Eds). New York: Springer.

Flammino, F., and B. M. Clopton. 1975. Neural responses in the inferior colliculus of albino rat to binaural stimuli. The Journal of the Acoustical Society of America 57: 692-695.

Fullerton, B. C., and H. L. Hosford. 1979. Effects of midline brainstem lesions on the short latency auditory evoked responses. Society of Neuroscience 5 (abstract): 20.

Furst, M., R. A. Levine, and P. M. McGaffin. 1985. Click lateralization is related to the B component of the dichotic brainstem auditory evoked potentials of human subjects. The Journal of the Acoustical Society of America 78 (5): 1644-51.

Furst, M., R. A. Levine, A. D. Korczyn, B. C. Fullerton, R. Tadmor, and D. Algom. 1995. Brainstem lesions and click lateralization in patients with multiple sclerosis. Hearing Research 82: 109-24.

Gardi, J. N., and C. I. Berlin. 1979. A preliminary report on the origin of the binaural interaction component of the BSER in the guinea pig. Society of Neuroscience 4: 20.

Gardi, J. N., and Berlin, C. I. 1981. Binaural interaction components: Their possible origins in guinea pig auditory brainstem response. Archives of Otolaryngology 107 (March): 164-168.

Gerken, G. M., G. Moushegian, R. D. Stillman, and A. L. Rupert. 1975. Human frequency following responses to monaural and binaural stimuli. Electroencephalography and Clinical Research 38: 379-86.

Glaser, E. M., C. M. Suter, R. Dasheiff, and A. Goldberg. 1976. The human frequency following response: its behavior during continuous tone and tone burst stimulation. Electroencephalography and Clinical Neurophysiology 40: 25-32.

Goldberg, J. M., and P. B. Brown. 1969. Response of binaural neurons of dog superior olivary complex to dichotic tonal stimuli: some physiological mechanism of sound localization. Journal of Neurophysiology 32: 613-36.

Guinan, J. J., Jr., S. S. Guinan, and B. E. Norris. 1972. Single auditory units in the superior olivary complex. I: Responses to sounds and classifications based on physiological properties. International Journal of Neuroscience 4: 101-20.

Hall, J. L., II. 1965. Binaural interaction in the accessory superior olivary nucleus of the cat. The Journal of the Acoustical Society of America 37: 814-23.

Harris, G. G. 1960. Binaural interactions of impulsive stimuli and puretones. The Journal of the Acoustical Society of America 32: 685-92.

Hink, R. F., K. Kodera, O. Yamada, K. Kaga, and J. Suzuki. 1980. Binaural interaction of a beating frequency following response. Audiology 19: 36-43.

Hosford, H. L., B. C. Fullerton, and R. A. Levine. 1979. Binaural interaction in human and cat brainstem evoked responses. The Journal of the Acoustical Society of America 65 (Suppl. 1): 86.

Irvine, D. R. F. 1992. Physiology of the auditory brainstem. In The Mammalian Auditory Pathway: Neurophysiology, A. N. Popper and R. R. Ray (Eds). (pp. 153-231). New York: Springer-Verlag.

Jacobson, J. T. 1994. Principles and Applications in Auditory Evoked Potentials. Needham Heights, Mass.: Allyn and Bacon.

Jeffress, L. A. 1948. A place theory of sound localization. Journal of Comparative and Physiological Psychology 41: 35-39.

Jones, S. J., and J. C. Van der Poel. 1990. Binaural interaction in the brainstem auditory evoked potential: evidence for a delay line coincidence detection mechanism. Electroencephalography and Clinical Neurophysiology 77: 214-24.

Kuwada, S., and T. C. T. Yin. 1983. Binaural interaction in low frequency neurons in the inferior colliculus of the cat. I. Effects of long interaural delays, intensity, and repetition rate on interaural delay function. Journal of Neurophysiology 50: 981-99.

Levine, R. A., J. C. Gardner, S. M. Stufflebeam, B. C. Fullerton, E. W. Carlisle, M. Furst, B. R. Rosen, and N. Y. S. Kiang. 1993b. Effects of multiple sclerosis brainstem lesions on sound lateralization and brainstem auditory evoked potentials. Hearing Research 68: 73-88.

McDaniel, S., and A. Krishnan. 1994. Human frequency following response correlates of interaural intensity differences. Master's Thesis, University of Tennessee, Knoxville.

McPherson, D. L., and A. Starr. 1993. Binaural interaction in auditory evoked potentials: brainstem, middle, and long latency components. Hearing Research 6: 91-98.

McPherson, D. L., and A. Starr. 1995. Auditory time - intensity cues in the binaural interaction component of the auditory evoked potentials. Hearing Research 89: 162-71.

Melcher, J. R. 1996. Cellular generators of the binaural difference potential in the cat. Submitted for publication February 1996.

Mills, A. W. 1960. Lateralization of high frequency tones. The Journal of the Acoustical Society of America 32: 132-134.

Moushegian, G., A. L. Rupert, and J. S. Gidda. 1975. Functional characteristics of superior olivary neurons to binaural stimuli. Journal of Neurophysiology 38 (5): 1037-47.

Musiek, F. E. 1985. Application of central auditory tests: an overview. In Handbook of Clinical Audiology Third Edition, J. Katz (Ed.). Baltimore: Williams and Williams.

Ornitz, E. M., and D. O. Walter. 1975. The effect of sound pressure waveform on human brainstem auditory evoked responses. Brain Research 92: 490-98.

Ozdamar, O., N. Kraus, and J. Grossman. 1986. Binaural interaction in the auditory middle latency response of the guinea pig. Electroencephalography and Clinical Neurophysiology 63 (5, May): 476-83.

Parthasarathy, T. K., and G. Moushegian. 1993. Rate, frequency, and intensity effects on early auditory evoked potentials and binaural interaction component in humans. Journal of the American Academy of Audiology 4 (4): 229-37.

Prasher, D. K., M. Sainz, and W. P. R. Gibson. 1981. Effects of interaural intensity differences on binaural summation of brainstem auditory evoked potentials. British Journal Audiology 15: 189-94.

Rose, J. E., N. B. Gross, C. D. Geisler, and J. E. Hind. 1966. Some neural mechanisms in the inferior colliculus of the cat which may be relevant to localization of a sound source. Journal of Neurophysiology 29: 288-314.

Rosenzweig, M. R., and D. Sutton. 1958. Binaural interaction in lateral lemniscus of cat. Journal of Neurophysiology 21: 17-23.

Rosenzweig, M. R., and E. J. Wyers. 1955. Binaural interaction at the inferior colliculi. Journal of Comparative and Physiological Psychology 48: 426-31.

Smith, J. C., J. T. Marsh, and W. S. Brown. 1975. Far-field recorded frequency following responses: evidence for the locus of brainstem responses. Electroencephalography and Clinical Neurophysiology 39: 465-72.

Spivak, L. G. and M. R. Seitz. 1988. Response asymmetry and binaural interaction in the auditory brainstem evoked response. Ear and Hearing 9 (2): 57-64.

Stillman, R. D. 1972. Responses of high frequency inferior colliculus neurons to interaural intensity differences. Experimental Neurology 36: 118-26.

Stockard, J. E., J. J. Stockard, B. F. Westmoreland, and J. L. Corfits. 1979. Brainstem auditory-evoked responses: Normal variation as a function of stimulus and subject characteristics. Archives of Neurology 36: 823-31.

Wada, S. and A. Starr. 1989. Anatomical bases of binaural interaction in auditory brainstem responses from guinea pig. Electroencephalography and Clinical Neurophysiology 72: 535-44.

Wernick, J. S., and A. Starr. 1968. Binaural interaction in the superior olivary complex of the cat: an analysis of field potentials evoked by binaural-beat stimuli. Journal of Neurophysiology 31: 428-41.

Wilson, M., D. Kelly-Ballweber, and R. A. Dobie. 1985. Binaural interaction in auditory brainstem responses: parametric studies. Ear and Hearing 6: 80-88.

Wrege, K. S., and A. Starr. 1981. Binaural interaction in human auditory brainstem evoked potentials. Archives of Neurology 38 (September): 572-80.

Yin, T. C. T., and J. C. K. Chan. 1990. Interaural Time Sensitivity in medial superior olive of the cat. Journal of Neurophysiology 62 (2, August): 465-88.

Yost, W. A. 1981. Lateral position of sinusoids presented with interaural intensive and temporal differences. The Journal of the Acoustical Society of America 70 (2): 397-409.

Yost, W. A., and D. Nielsen. 1985. Fundamentals of Hearing. New York: Holt, Rhinehart, and Winston.

APPENDICES

APPENDIX A
SUBJECT CONSENT FORM

Informed Consent Form For Study

Title: Human Frequency Following Response Correlates of Interaural Time Difference and Time-Intensity Trade

I. Description

You are being asked to participate in an experimental study aimed at evaluating how neurons in the lower part of the brain, called the brainstem, of normal hearing individuals respond in relation to differences in time and intensity at each ear. The responses recorded from the brainstem in this project are called frequency following responses (FFRs). Should you agree to participate the test session will not exceed two non-stop hours in which you will be required to remain still for the entire session.

Your hearing sensitivity will be tested using a routine clinical test wherein tones of different pitches will be presented to you through headphones, and you will be instructed to respond, by pressing a button, when you hear each tone. Following completion of the above test, you will be asked to identify on a chart, the location of a sound image in the head, which will be presented through headphones at different times and intensities. The FFRs will then be recorded by placing electrodes, which are small metal discs, on the scalp. The electrodes are held in place by electrode paste and plastic tape after the surface of the skin has been gently cleaned. During the test session, you will be asked to simply remain still and relax in a recliner while you listen to a series of signals through headphones.

The above tests and procedures are routine clinical procedures which offer no known health risks. Regardless, every effort will be made to minimize any potential risk or discomfort. Although you may not benefit directly from this study, you will have contributed to our understanding of binaural processing in the brainstem.

II. Confidentiality

Any information obtained about you for this study will be kept strictly confidential. Confidentiality of your data as well as any identifying material will be protected by limiting access to only the Principal and Co-Principal Investigator's. Research records, just like hospital records, may be subpoenaed by court order. Your identity will not be revealed in any description or publication of this research. This consent form will be stored for 3 years following completion of the study at a secured location at the University of Tennessee.

III. Right To Refuse Or To End Participation

You are free to refuse to participate in this study or to withdraw at any time

without having it affect your care in any of the University clinics and without any loss of benefits to which you might otherwise be entitled.

IV. Voluntary Consent

I have read the above materials, and the Principal Investigator has explained the procedures and answered all my questions concerning this project. It has been explained to me that my identity will not be revealed in any description or publication of this research. Therefore, I consent to such publication for scientific purposes.

Any questions I have concerning my rights as a research subject will be answered by Carol A. Boyd, the principal investigator, or Dr. A.K. Ananthanarayan (Ravi), the co-principal investigator, at 570 S. Stadium Hall. Telephone (423) 974-1811.

A copy of this consent form will be given to me.

My signature below means that I have freely agreed to participate in this experimental study.

Signature of subject _____ Date _____

V. Investigator's Certification

I certify that I have explained to the above individual the nature and purpose, the potential benefits, and the possible risks associated with participating in this research study. I have answered any questions that have been raised, and have witnessed the above signature.

Signature of investigator _____ Date _____

Prior to this session, my professor signed stating that credit would be given to me at the completion of my participation in this experimental study. My signature below means that I understand the terms under which I will receive credit.

Signature of subject _____ Date _____

Signature of professor _____ Date _____

APPENDIX B
INTERAURAL TIME DIFFERENCE INDIVIDUAL AMPLITUDE DATA (IN dB
VOLTS) FOR SUMMED MONAURAL RESPONSES EVOKED BY 500 Hz TONE
BURSTS

INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS) FOR MONAURAL SUM

Subject	Interaural Time Difference (ms)							
	0	0.2	0.4	0.6	0.8	0.9	1.0	1.2
1	-146.02	-151.25	-153.66	-147.86	-144.03	-142.78	-141.86	-140.79
2	-145.02	-147.08	-150.70	-156.50	-155.50	-152.41	-149.93	-146.60
3	-140.32	-142.22	-145.79	-153.39	-155.34	-149.82	-146.39	-142.33
4	-147.85	-143.65	-146.93	-152.51	-153.26	-150.12	-147.48	-143.95
5	-139.81	-140.68	-142.64	-146.31	-153.63	-157.06	-153.17	-146.14
6	-147.68	-146.87	-146.86	-147.66	-149.39	-150.66	-152.22	-155.76
7	-139.51	-140.08	-141.58	-144.34	-148.92	-151.36	-151.46	-146.45

APPENDIX C
TIME-INTENSITY TRADE INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS)
FOR SUMMED MONAURAL RESPONSES EVOKED BY 500 Hz TONE
BURSTS

INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS) FOR MONAURAL SUM

Subject	Time - Intensity Trading (dB SPL)			
	83 dB	86 dB	89 dB	92 dB
1	-144.44	-142.18	-140.66	-139.18
2	-143.33	-145.81	-146.88	-149.42
3	-139.41	-139.99	-140.69	-142.16
4	-140.62	-140.57	-142.16	-143.02
5	-140.83	-141.03	-141.50	-142.75
6	-144.53	-142.81	-142.35	-142.58
7	-139.31	-138.99	-138.40	-138.78

APPENDIX D
INTERAURAL TIME DIFFERENCE INDIVIDUAL AMPLITUDE DATA (IN dB
VOLTS) FOR BINAURAL RESPONSES EVOKED BY 500 Hz TONE BURSTS

INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS) FOR BINAURAL

Subject	Interaural Time Difference (ms)							
	0	0.2	0.4	0.6	0.8	0.9	1.0	1.2
1	-146.46	-151.62	-149.93	-154.68	-153.91	-149.71	-146.48	-143.10
2	-146.55	-149.65	-157.56	-160.27	-148.33	-147.58	-147.34	-149.26
3	-143.15	-150.33	-149.98	-158.01	-151.53	-148.14	-147.97	-144.63
4	-146.35	-145.75	-148.52	-153.16	-146.58	-147.60	-146.55	-146.46
5	-142.73	-142.65	-142.95	-143.53	-146.40	-149.66	-150.47	-158.57
6	-147.61	-147.89	-146.20	-153.48	-159.21	-159.89	-155.17	-152.26
7	-141.87	-141.86	-143.25	-144.63	-147.87	-148.91	-153.14	-153.90

APPENDIX E
TIME-INTENSITY TRADE INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS)
FOR BINAURAL RESPONSES EVOKED BY 500 Hz TONE BURSTS

INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS) FOR BINAURAL

Subject	Time - Intensity Trading (dB SPL)		
	83 dB	86 dB	89 dB
1	-146.59	-148.96	-153.27
2	-146.94	-143.79	-140.92
3	-148.07	-151.12	-153.20
4	-147.51	-144.95	-143.47
5	-149.66	-149.00	-148.21
6	-150.56	-148.27	-147.18
7	-153.87	-149.75	-146.58

APPENDIX F
INTERAURAL TIME DIFFERENCE INDIVIDUAL AMPLITUDE DATA (IN dB
VOLTS) FOR BINAURAL INTERACTION COMPONENT

INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS) FOR BINAURAL INTERACTION COMPONENT

Subject	0	Interaural Time Difference (ms)						
		0.2	0.4	0.6	0.8	0.9	1.0	1.2
1	-152.15	-159.88	-145.64	-145.62	-144.84	-145.10	-146.73	-150.77
2	-151.15	-155.13	-155.49	-157.39	-153.12	-154.97	-155.79	-158.03
3	-151.16	-145.85	-147.99	-159.21	-154.40	-157.17	-156.32	-154.96
4	-149.49	-156.81	-161.69	-159.70	-150.92	-150.87	-150.42	-149.69
5	-152.86	-152.76	-155.44	-153.87	-149.29	-148.57	-148.16	-149.46
6	-151.41	-153.89	-156.46	-156.03	-151.43	-146.69	-146.26	-145.22
7	-150.28	-153.52	-152.95	-151.85	-148.09	-148.04	-145.76	-146.34

APPENDIX G
TIME-INTENSITY TRADE INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS)
FOR BINAURAL INTERACTION COMPONENT

INDIVIDUAL AMPLITUDE DATA (IN dB VOLTS) FOR BINAURAL INTERACTION COMPONENT

Subject	Time - Intensity Trading (dB SPL)			
	83 dB	86 dB	89 dB	92 dB
1	-143.35	-141.87	-140.32	-139.95
2	-139.39	-138.99	-138.21	-137.52
3	-139.13	-140.62	-141.92	-142.68
4	-138.90	-137.00	-137.00	-137.03
5	-146.57	-145.45	-146.84	-146.74
6	-139.36	-138.95	-138.06	-138.84
7	-142.71	-142.99	-143.51	-142.67

VITA

Carol A. Boyd was born in Arlington, Tennessee on October 6, 1973. She received her Bachelor of Science in Speech Pathology and Audiology from Tennessee State University in May of 1995. She is a candidate for the Master of Arts degree to be conferred May of 1997. Future plans include completing a Clinical Fellowship Year in Audiology and pursuing a Doctoral degree.