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NAME OF AUTHOR/*NOM DE L'AUTEUR* DAVID HALLMAN

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THE EFFECTS OF BINAURAL CLICK INTEGRATION AS REPRESENTED IN THE AUDITORY BRAINSTEM EVOKED RESPONSE (BER).

by

David William Hallman

B.Sc. (Hons.), University of Victoria, 1975

M.A., Simon Fraser University, 1977

A THESIS SUBMITTED IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

in the Department

of

Psychology

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THE EFFECTS OF BINAURAL CLICK INTEGRATION AS REPRESENTED IN THE AUDITORY BRAINSTEM EVOKED RESPONSE (BER).

Author:

(signature)

DAVID HALLMAN

(name)

Sept 27/79

(date)

APPROVAL

Name: David William Hallman

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Examining Committee:

Chairperson: Vito Modigliani

Barry Beyerstein
Senior Supervisor

A. Leonard Diamond

Ray Koopman

Hal Weinberg

Patrick Gannon
External Examiner
Assistant Director
Medical Services Department
Occupational Health/Hearing
Workers' Compensation Board of B.C.

Date Approved: Sept. 24, 1979

Abstract

This study was carried out to investigate changes in the Auditory Brainstem Evoked Response (BER) resulting from the interaction of binaural click stimuli (sound lateralization) in the Classical Auditory System (CAS). Twenty normal hearing male and female university students served as subjects. Scalp evoked responses were collected from vertex/mastoid, vertex/non-cephalic and mastoid/mastoid derivations. Click parameters included phase, time and intensity. Both the amplitude and latency of six positive and five negative BER peaks were determined and analyses of variance carried out. Comparisons included binaural vs algebraic summed equivalent monaural responses; binaural rarefaction vs binaural condensation evoked responses; mastoid 'reference' vs non-cephalic 'reference' derivations; and, monaural right vs monaural left latencies for BER positive peak five.

Clear evidence for binaural coding at an early and a later BER generator source was shown. In addition, support was provided for the following:

a) a reiteration of the BER active nature of mastoid "reference" sites;

b) the mimicking of dipole configurations by Peak 2;

c) a larger overall vertex/non-cephalic waveform, as compared with a vertex/mastoid waveform, suggesting that all BER waves are homophasic at the vertex and mastoid;

d) active CAS processing represented in some BER troughs;

e) BER representation of a dual CAS information processing system -- one involving peripheral coding, and a second involving central decoding and encoding for localization;

f) phase-coding as an end-organ process;

g) a right/left difference in the coding and transmission of information concerning phase;

h) possibility of bilaterally equal BER intensity/latency functions; and,

i) possibility that the BER "slow wave" is a monaural phenomena.

The implications of the above are discussed in terms of expected changes based on past BER research, other lines of auditory research and hypothesized BER generator sources.

Table of Contents

List of Tables

List of Figures

List of Appendices

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CHAPTER ONE

The brainstem evoked response (BER), recorded from a vertex/mastoid derivation, has been studied extensively in the last ten years. Based on animal and human studies, it has been widely accepted that the wave peaks of this response represent the successive activations of the classical auditory system (CAS). Much time and effort has been expended on the delineation of the specific generator sources for each BER wave peak, and a general consensus has emerged. This study attempts to integrate past BER research with other lines of auditory research in an effort a) to determine if auditory binaural interactions (sound lateralization) are represented in the BER; and, b) to correlate BER changes (if they occur) with expected changes based on hypothesized generator sources.

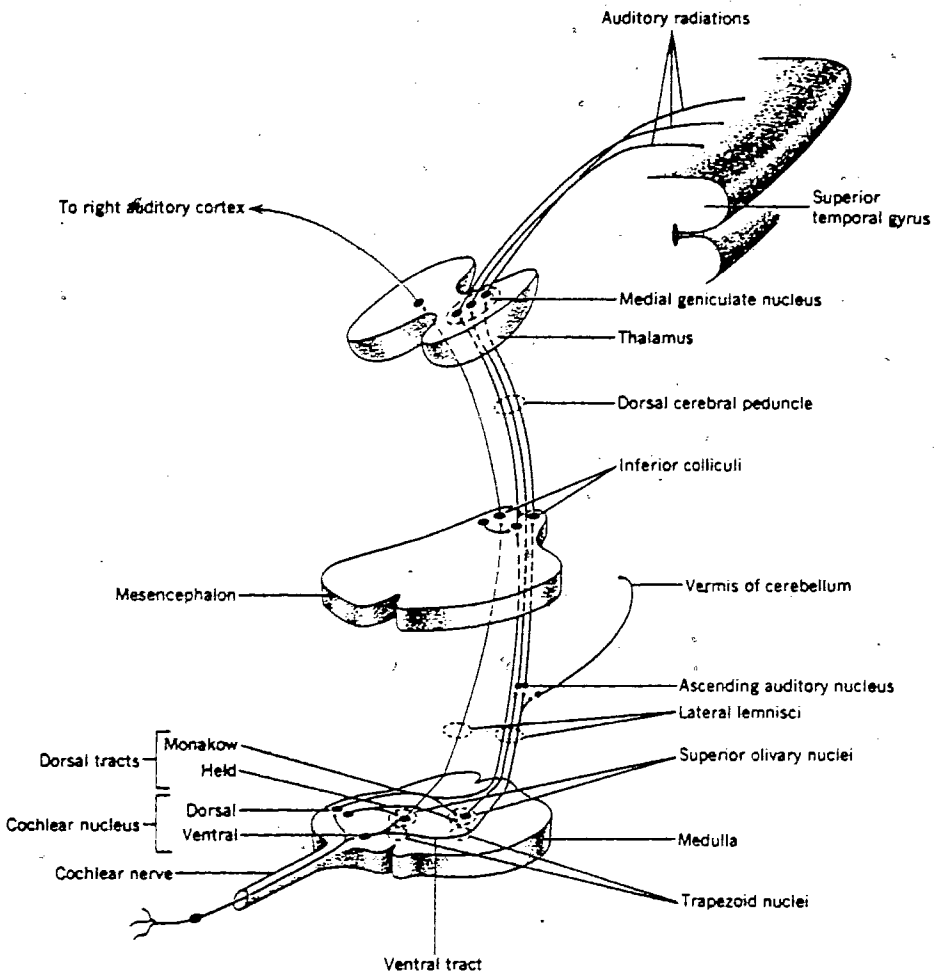
THE CLASSICAL AUDITORY SYSTEM (CAS)

The Centripetal System (Figures 1 and 2)

The anatomical organization of the CAS has been determined mainly from animal studies. While it has been conjectured that human structure resembles that of other species, the observed interspecies variation makes this extrapolation somewhat hazardous. Nevertheless, this hypothesis, modified by limited human data, constitutes our best estimation of the structure of the human CAS. The majority of the following has been condenced from Gulick (1971) and Gacek (1972).

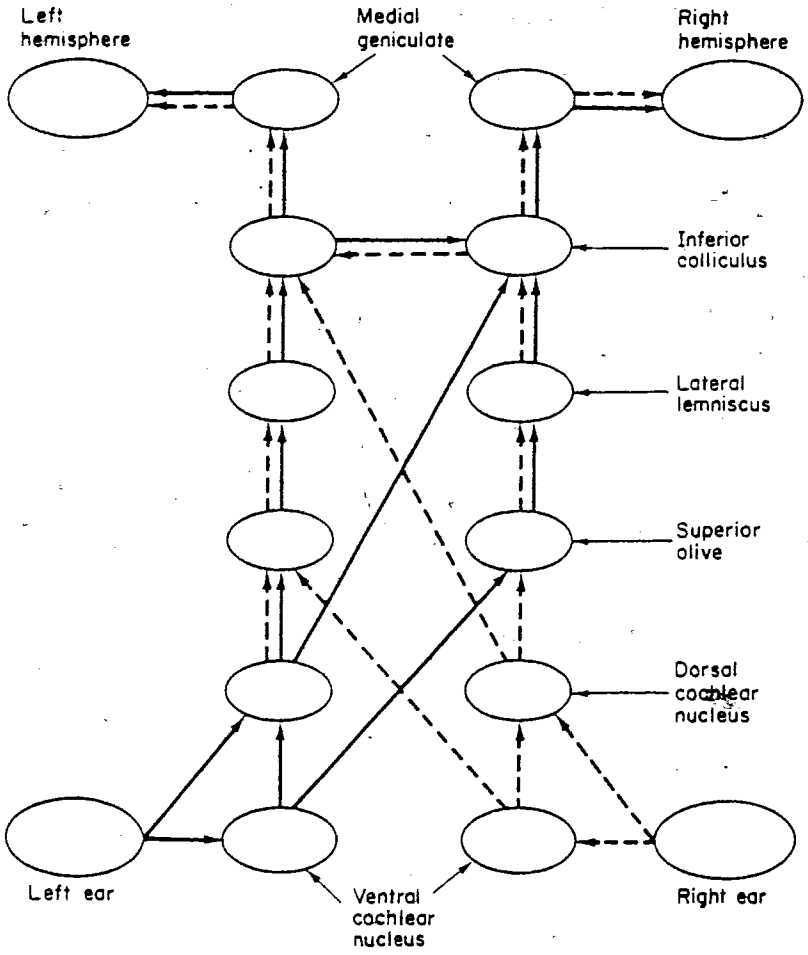
The cochlea transduces mechanical energy in the form of sound waves into tonotopically organized neural codes through its accessory structures and orderly arrangement of receptor elements. These codes are transmitted to the auditory cortex via a number of nuclear stations, within which further processing probably occurs, as indicated by the progressively greater number of parallel pathways and diverse interconnections.

The afferent innervation of the Organ of Corti is accomplished by the bipolar cochlear neurons. These cell bodies, numbering in excess of 31,000 (Rasmussen, 1940),



Schematic arrangement of the major ascending auditory pathways.

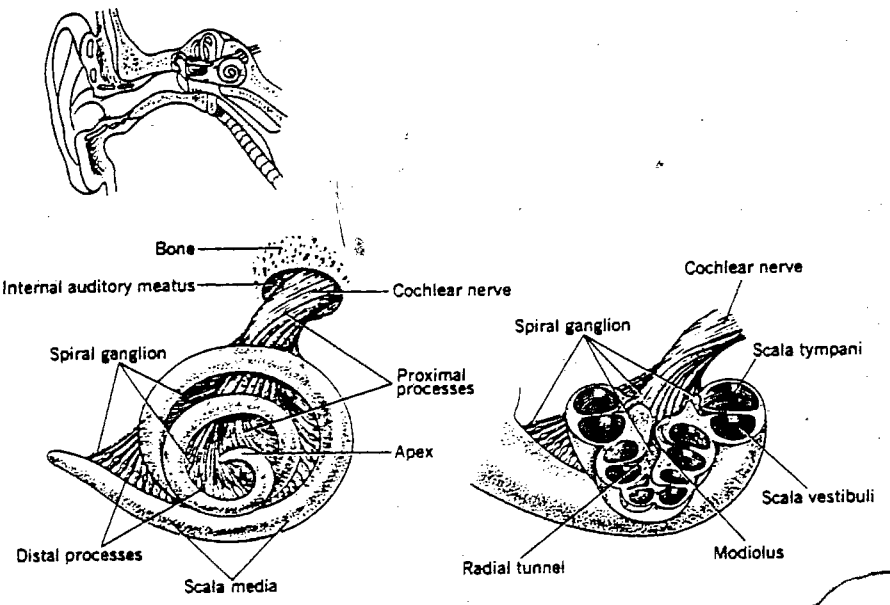
FIGURE 1
(GULICK, 1971)



SCHEMATIC ARRANGEMENT OF CAS
FIGURE 2
(MOORE, 1977)

comprise the spiral ganglion of Rosenthal's canal. The lateral processes reach directly to the end organ, while the medial processes project to the medulla through the internal auditory meatus. The medial processes of the cochlear nerve, are twisted tightly together resulting from the developmental coiling of the cochlea. In terms of the BER, this twisting raises some question as to the source of wave one (see Figure 3). Since the dipoles of each individual action potential will be oriented at all meridians of a 360 degree circle, and therefore tend to cancel, it may not be logical to assume that wave one originates from the eighth nerve. Fibers from the apical-turn of the cochlea occupy a central route through this nerve, while basal-turn fibers follow a route near the peripheral and inferior margins. Of interest is that the density of fibers originating in the middle cochlear turns (locus of maximal activation by middle frequencies) is greater than the density from either pole, consistent with the known greater sensitivity in humans to middle frequency tones.

The auditory portion of the eighth cranial nerve enters the medulla near the border with the pons; the full length of this nerve averages only 5mm, while the myelin-sheath diameters vary between 3u and 10u. The terminus of all these first-order auditory fibers is the cochlear nucleus (CN), a nuclear station comprised of two major divisions: a) the dorsal cochlear nucleus (DCN); and, b) the ventral cochlear nucleus (VCN) which is



The neurology of the right cochlea. The scala vestibuli, scala tympani, and all surrounding bone are removed, thus leaving only the membranous scala media. The distal neural endings connect with the spiral ganglion from which the proximal endings twist upon each other and run medially through the internal auditory meatus to synapse in the medulla.

FIGURE 3
(GULICK, 1971)

further divided into the posteroventral cochlear nucleus (PVCN) and the anteroventral cochlear nucleus (AVCN). Upon entering the anteroventral complex, all fibers bifurcate, sending axons into the DCN and PVCN divisions (Rose et al., 1960). Degeneration studies (Sando, 1965) and other experiments concerning the location of best fiber frequency within the CN (Rose et al., 1965) suggest that the topographical ordering of the cochlea is reproduced in the CN. Fibers originating in the upper basal and middle regions of the cochlea terminate in the dorsal portion of both the DCN and PVCN, while those from the apical regions terminate in the ventral areas of both CN divisions. Fibers from the lower basal turns, however, show a much more general termination within the CN (Lorente de No, 1933; Rasmussen, 1960; and, Sando, 1965). Although the afferent input is to both the DCN and VCN, by far the majority project to the VCN. All evidence so far indicates that no first-order axons bypass the CN. The greatest number of axons synapse with second-order neurons. However, a certain percentage are internuncial, thus providing the necessary structure for complex cellular interactions to occur at this level.

Second-order acoustic fibers project from the CN to the mesencephalon and thalamus via the lateral lemniscal acoustic pathways. These fibers ascend by way of three main tracts within the medulla; the tracts of Monakow and Held in the

dorsal position, and the Trapezoid body in the ventral position.

The tract of Monakow (dorsal acoustic stria) is the larger of the two dorsal tracts (see Figure 2). Originating in the dorsal portion of the DCN, these fibers pass through the reticular formation, decussate via the posterior tegmentum, send collaterals into the contralateral superior olivary complex (SOC) and terminate in either the dorsal nucleus of the lateral lemniscus (LL) or in the inferior colliculus (IC). No axons of this tract terminate higher than the IC level. The tract of Held (intermediate acoustic stria) originates in the VDCN and terminates on cells in both the ipsilateral SOC and contralateral SOC (after decussating via the intermediate tegmentum). The Trapezoid body (ventral acoustic stria) originates in the VCN, passes through the anterior tegmentum and sends fibers to both the ipsilateral and contralateral trapezoid nuclei. Some of the axons synapse at this level with third-order acoustic neurons before ascending into the LL, while others ascend directly into the LL, sending collaterals into the Trapezoid bodies and reticular formation.

The terminations of the intermediate and ventral acoustic striae in the SOC result in a pattern that is probably important for the location of sounds in space, as discussed below. There are diffuse terminations with the main superior olivary segment, lateral terminations with the homolateral

accessory olivary region, and medial termination with the contralateral accessory olivary nucleus. Another possibly important pattern has been described by Rasmussen (1967); the trapezoid bodies send axons into the lateral superior-olivary nuclei, which have direct efferent connections with the contralateral VCN. This results in a direct connection between the two VCN via a chain encompassing only two synapses.

The SOC appears to play three important roles: a) as the first level for the integration of binaural inputs (as discussed below); b) as a relay center for the CAS; and, c) as a center for the mediation of certain eye and hand movements in response to sound as well as the reflex contractions of the middle-ear muscles. In addition, there appear to be reflex connections that involve the somatic musculature via the spinal nerves. Another important consideration, in terms of the source of the BER, is that the SOC has been described as a closed field pool by Lorente de No (1947). That is, most of the dendrites are located in the center of the nuclear mass, while the somata lie in the periphery. Since the currents will flow radially between the periphery and the center of the pool, theoretically, no neural activity can be detected outside the volume of the nucleus.

Second- and third-order fibers pass through the LL while third- and forth-order fibers make synaptic contact at this level before ascending to higher auditory nuclei. In addition,

the LL pathways are the origin of two important collateral routes; one projecting to the vermis of the cerebellum, and a second, more diffuse route involving the reticular formation.

Almost all axonal processes synapse at the level of the IC; those that do not, send collaterals into the IC and synapse in the parvocellular portion of the medial geniculate bodies (MGB), after passing through the brachium of the IC. No neurons, however, from the CN or SOC reach the nucleus of the MGB directly. Decussations of the CAS above the level of the SOC occur at the level of the dorsal nucleus of the LL (small commissure) and the IC (strong commissure). The IC is composed of three divisions: a) a main spherical nucleus; b) a gray nuclear mass medial to the main nucleus; and, c) a smaller lateral nucleus. These divisions are richly interconnected with each other as well as the divisions of the contralateral side. In addition, there are some connections with the visual centers of the superior colliculi.

No ascending neurons decussate or bypass at the level of the MGB. Therefore, these final neurons which send axons via the auditory radiations to the superior temporal gyrus of the cortex (transverse gyri of Heschl) by way of the sublenticular portion of the posterior limb of the internal capsule, are at least of the fourth order.

The MGB are composed of a superior and an inferior lobe. While the inferior lobe's role is chiefly with the ascending

projections, both lobes receive descending projections. The inferior lobe has been cytologically divided into the pars principalis (PP) and the pars magnocellularis. The majority of, if not all, ascending projections are by way of the PP, and terminate in an orderly fashion in the primary auditory center (AI) of the cortex. Projections from the anterior portion end rostrally on AI, while those of the posterior portion end caudally on AI.

The Centrifugal System

Rasmussen (1946, 1953) described two separate efferent neural systems that originate at cortical levels and end in either the SOC or DCN.

The first system originates in auditory areas AI or AII (secondary cortical auditory areas in the inferior temporal lobe) and descends to the IC through the MGB. Second-order fibers synapse at this level and descend to the SOC where they synapse with multipolar cells comprising the olivocochlear nucleus.

The second system originates in both auditory areas AI and AII as well as associated areas. Similar to the first system, most of these fibers make synaptic contact at the IC, although collaterals project into the reticular formation and the MGB. Second-order fibers descend as a compact bundle running along

side the ascending tracts, and terminate mainly at the level of the ipsilateral preolivary nucleus, although a number of fibers do terminate at the level of the contralateral nucleus. Numerous other third-order efferents synapse at various levels in this tract and terminate in the DCN.

The efferent cochlear bundle, which descends from the SOC to the hair cell receptors, is composed of two separate branches; the olivocochlear bundle, and the peduncle of the olive. The olivocochlear bundle, consisting entirely of crossed fibers, originates in the medial accessory superior olivary nucleus and passes near the abducent nucleus before joining the cochlear nerve to terminate on the outer hair cells. The peduncle of the olive, also originating from the accessory olive area, is only 1/4 the size of the olivocochlear bundle and is composed of totally uncrossed axons (Rasmussen, 1960) which terminate near the inner hair cells. Although the number of fibers entering the cochlea is rather small (approximately 500), they ramify extensively within the cochlear duct, resulting in excess of 40,000 endings on the outer hair cells. The distribution of the endings on the outer hair cells is not uniform, ranging from 6-8 endings per cell near the base and decreasing progressively toward the apex. Although the efferent fibers terminate directly on the outer hair cells, they do so only rarely on the inner hair cells. The consensus is that, rather than affecting the hair cells

directly, these endings have regulatory control over the activity of the afferent fibers. It is evident that the extensive ramification of these relatively few fibers results in an innervation pattern that is diffuse and undifferentiated. The centrifugal fibers are likely a means of feedback control of the auditory input, both processing and sharpening signals, and inhibiting noise.

That the auditory system is involved in a number of complex integrative functions is indicated by the converging and diverging nature of the neural connections, the descending as well as ascending pathways, the numerous crossed connections, and the increasing number of axons as the pathway ascends from one nucleus level to the next. As a graphic example of the increasing complexity of the CAS, Chow (1951) carried out a cell count of one half of the monkey's CAS:

CN	=	88,000
SOC	=	34,000
LL	=	38,000
IC	=	392,000
MGB (PP)	=	364,000
Cortex	=	10,000,000

CHAPTER TWO

BRAIN STEM EVOKED RESPONSE (BER)

The recording of the auditory brain stem response via scalp electrodes is a relatively new procedure. Although both the technical knowledge and the instrumentation were available in the early 60's, records were not obtained until the late 60's, probably as a result of misconceptions about the plausibility of neural volume conduction (Cooper et al., 1965). Physiological sensory research at this time primarily involved single unit recording via depth electrodes in animal preparations and/or averaged cortical evoked potentials. Clinical recordings of the cochlear microphonic and eighth nerve action potentials had become quite sophisticated (electrocochleography), and it was through efforts to improve existing techniques that the scalp recorded BER was developed.

Cochlear microphonic potentials from the round window were observed in humans as early as 1935 (Fromm et al.). During the next 15 years amplification and recording techniques were greatly improved (Perlman and Case, 1941; Lempert et al., 1947;

and, Lempert et al., 1950). In 1950 (Lempert et al.), promontory recording, accomplished by passing a needle electrode through the tympanic membrane during surgery, was suggested as a possible clinical technique. In 1961 a diagnostic method was proposed by which click evoked brain stem action potentials could be recorded from the round window, again during ear surgery (Ruben et al.). By 1967 electrocochleography (promontory recording) had become a routine diagnostic technique (Portmann et al., 1967), however, it failed to provide either a panoptical view of the auditory system, or to deal with the distructive effects of the electrode. In that same year, two important studies in the initial development of BER recording were also reported. In both cases the attempt was to find a way to measure cochlear action potentials (eighth nerve and cochlear nucleus APs) without resorting to surgical procedures. Using averaging techniques, Yoshie et al. (1967) recorded these potential fluctuations from a needle electrode placed in the skin of the external auditory meatus referred to the earlobe, while Sohmer and Feinmesser (1967) recorded them from the earlobe referred to the nose. Sohmer and Feinmesser were able to identify four successive negative peaks, approximately one msec apart, although the last two showed a great deal of variation in latency. These workers concluded that the first two peaks were the N1 and N2 components of the cochlear action potential,

while the following two were either the repetitive firing of the eighth nerve, or the activation of auditory nuclei in the brain stem. In a following study, Sohmer and Feinmesser (1970) recorded these same waves with an earlobe/vertex electrode derivation, although the published waveform clearly shows the first six BER waves. Implicit in these reports was the acceptance of volume conduction of neural potentials over relatively extensive distances.

In 1970 the first comprehensive study of these early auditory EP components was reported. Jewett (1970), using depth electrodes in cats, recorded five positive waves comparable both in latency and amplitude to those reported by Sohmer and Feinmesser. This study is important not only for being first but also because the results have been used extensively as the basis for further study.

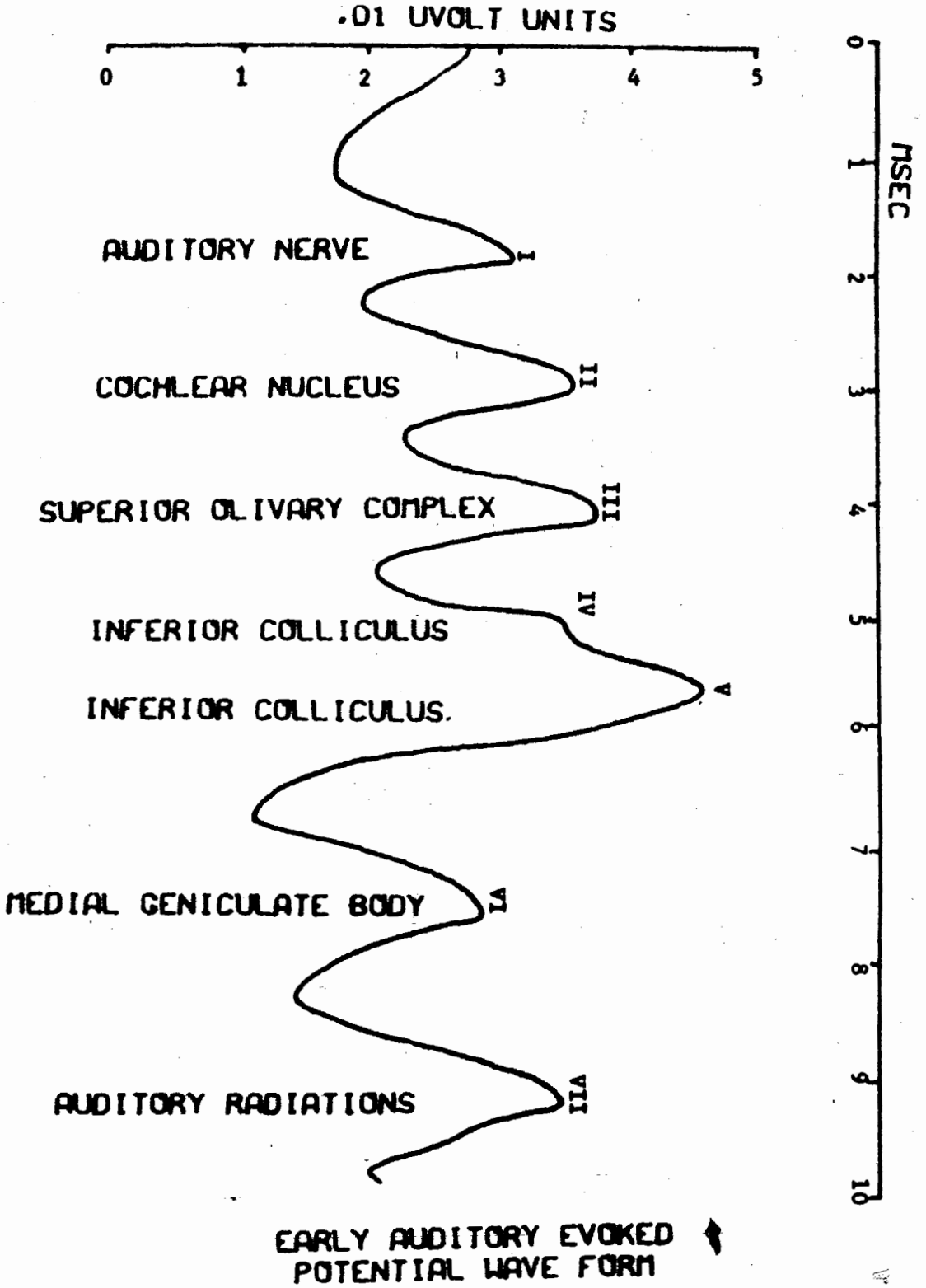
Jewett used two 'active' electrodes both referred to the cat's tongue. The first was placed in the caudate nucleus (a non-auditory nucleus) as a reference for any changes in waveform over time; the second was stereotaxically advanced through the brain stem auditory nuclei, with recordings taken at a number of sites. The intent was to correlate anatomical location with particular aspects of the waveform so that the contribution of any one auditory nucleus or tract could be determined. Jewett concluded that the first wave was generated by the action potentials of the eighth nerve, the second wave

was generated near the cochlear nuclei, the third in the vicinity of the superior olivary complex, the fourth in an area ranging from the lateral lemniscus to the brachium of the inferior colliculi, and the fifth from an area including both the inferior colliculi and the medial geniculate bodies. A basic problem with Jewett's study arises from his interpretation of depth recordings based on the work of Woodbury (1965). Woodbury showed that an action potential moving either toward or away from an electrode in a three-dimensional infinite volume conductor will be recorded as a positivity, whereas, as it passes the electrode it will be recorded as a negativity. Jewett considered that both an increase in amplitude and an inversion in polarity in BER components as the electrode was advanced through the brain stem would indicate that component's generator source. However, considering both the complex intertwining of the auditory system's projections, and the anisotropic and inhomogenous finite neural volume conductor of the cat brain, it is questionable whether Woodbury's theory is applicable.

BER research has been carried out in a great many labs, each, more or less, supporting Jewett's historic findings. In addition, comparisons between humans, monkeys, cats and rats, based on the relative size and weight of the brainstem, have shown response similarity across species (Allen and Starr, 1978). In general, the first 10 msec of the stimulus-locked

auditory response shows a sequence of seven or more positive waves when summed at the vertex. The earliest wave appears in the 1.5 msec latency range and the seventh in the 7-8 msec range, with approximately one msec between wave peaks. Wave seven is the same as wave N0 of the middle auditory components (Mendel and Goldstein, 1969; Picton and Hillyard, 1974; and, Picton et al., 1974). Figure 4 (Hallman, 1977) shows a BER waveform with hypothesized neural generators. It should be noted that these 7 waves are the most consistently seen, both within and between subjects. Other waves occur at times, however, such as a wave between wave five and six reported by Don et al. (1977). Since the first BER wave (latency approximately 1.5 msec) is considered to be the first measurable component of the neural conduction of auditory signals as seen by a scalp electrode, it has been considered as a measure of peripheral conduction latency (PT), whereas, wave five minus wave one has been used as a measure of central conduction latency (CT) (Huang and Buchwald, 1978; Salamy and McKean, 1976; Salamy et al. 1975; and, Shah et al., 1978).

Jewett recognized that although the first wave may represent exclusively first order neurons, this is likely not to be the case for later waves. His reasons are based on a number of reports that show the activity in some of these nuclei extends into the period of the following waves (Ross et al., 1959; Moushegian, 1962; Katsuki et al., 1958; Galambos et



EARLY AUDITORY EVOKED POTENTIAL WAVE FORM

FIGURE 4

al., 1959; and, Thurlow et al., 1951). For example, the cochlear nucleus (BER wave at approximately 2.5 msec) shows activity that lasts up to 4.5 msec, and the superior olive (BER wave at approximately 3.5 msec) has single unit responses that vary between 2.5 and 10 msec.

BER PROPERTIES

The average latencies of BER wave peaks have normal distributions (Thornton, 1975) with little within or between subject variability (Amadeo and Shagass, 1973; Goldenberg and Derbyshire, 1975; Hecox and Galambos, 1974; Rosenhamer et al., 1978; Salamy and McKean, 1976; and, Weber and Fujikawa, 1977); although Amadeo and Shagass report that the latencies of wave two and four were found to be prolonged on the second day of recording. Myogenic contamination does not appear to be a factor. Potentials from the middle ear muscles have a ten msec latency, with intense stimulation (>75 dB) (Jewett et al., 1970; Picton et al., 1974; and, Simmons et al., 1966). In addition, Picton et al. (1974) indicate the following for auditory scalp muscle reflexes:

a) post-auricular muscle: variable from subject to subject and even within subjects. Large negative peak at 11.8 +/- 0.8 msec and positive at 16.4 +/- 0.7 msec;

b) temporalis muscle: very easily recordable from subjects

with clenched teeth. Large negative peak at 17.2 +/- 1.9 msec and positive peak at 22.8 +/- 2.8 msec;

c) neck muscles: recorded from inion. Begins as early as 7.4 msec. Has multiple components: negative waves at 11.3 +/- 0.2 msec and positive waves at 16.8 +/- 2.4 msec and 33.8 +/- 0.5 msec; and,

d) frontalis muscle: highly variable response. There is usually a distinct positive component at approximately 30 msec. Streletz et al. (1977) have shown this reflex to have a positive wave at 14-18 msec and 22-36 msec.

The wave peaks are not affected by habituation (Salamy and McKean, 1976; Schulman-Galambos and Galambos, 1975; and, Webster, 1971); by lack of attention (Picton and Hillyard, 1974; and, Picton et al., 1971); by sessional experiences (Thornton, 1975); or, by fatigue of the neural system (Schulman-Galambos and Galambos, 1975). However, Huang and Buchwald (1978) suggested that observed decrements in the amplitude of wave one which result from increases in stimulus duration or decreases in inter-stimulus-interval (ISI) may be due to overstimulation and/or fatigue of the system. Jewett (1970) reported that both increases in peak latency and decreases in peak amplitude result after prolonged stimulation in animals. This is compatible with an earlier study in which prolonged auditory stimulation resulted in a progressive amplitude decrement in cats (Hernandez-Peon and Scherrer,

1955). The BER waveform, however, has been shown to be completely adapted by the 3rd to 4th click (Thornton and Coleman, 1975).

Although states of consciousness, whether natural, traumatic or drug-induced, have long been considered not to effect the BER (Amadeo and Shagass, 1973; Goldenberg and Derbyshire, 1975; Schulman-Galambos and Galambos, 1975; Sohmer and Student, 1978; Sohmer et al., 1978; and, Zollner et al., 1976), recently a number of studies have suggested that specific drugs do change the waveform (although as early as 1972, Jewett and Romano noted changes in BER amplitude, in rats and cats, that were dependent on depth of anesthesia). Allen and Starr (1978) showed that, in monkeys, barbituate injections led to amplitude reductions in all waves. Mendel (1977) showed, in guinea-pigs, that, with low-intensity clicks, injections of atropine result in a doubling of the wave amplitude and an increase of up to 0.3 msec in the latencies. Squires et al. (1978) and Starr (1978) have shown that, in humans, intoxicating doses of alcohol result in latency increases, although not amplitude changes.

The interaural latency difference (ILD) has been shown to be less than 0.2 - 0.3 msec (Selters and Brackmann, 1977) and is now used as the cut off for indicating pathology (Clemis and Mitchell, 1977), although one study presented data showing quite marked wave seven differences in normal subjects (Rowe,

1978). Clemis and Mitchell have also shown that bone conduction of the clicks may result in a low amplitude peak five that is delayed 2-4 msec; however, Rosenhamer et al. (1978) have shown, with patients deaf in one ear, that this phenomenon becomes noticeable only with click intensities in excess of 75 dB HL.

The BER components show a complex of effects common to all neural systems (Picton et al., 1977). Decrease in stimulus intensity results in decreases in wave peak amplitude and increases in wave peak latency (Allen and Starr, 1978; Huang and Buchwald, 1978; Hyde et al., 1976; Jewett et al., 1970; Kodera et al., 1977; Portmann and Aran, 1971; Pratt and Sohmer, 1977; Yoshie, 1968; and, Zollner et al., 1976). As the ISI decreases there is a decrease in peak amplitude and an increase in peak latency (Allen and Starr, 1978; Amadeo and Shagass, 1973; Hallman, 1977; Hecox and Galambos, 1974; Jewett and Romano, 1972; Picton et al., 1974; Pratt and Sohmer, 1976; Rowe III, 1978; and, Webster, 1971). These changes are, however, not proportional across all waves (Pratt and Sohmer, 1976). Although the amplitude of wave one appears to increase in a linear fashion with increasing click intensity, the later waves reach a saturation point at intermediate intensities, and may even occasionally decrease in amplitude at high intensities. Latency shifts resulting from changes in stimulus intensity, on the other hand, affect all waves equally. Decreasing ISIs

result in a larger decrease in wave one amplitude as compared to the later waves, whereas, wave one shows no ISI/latency effect as do later waves. In all cases, decreases in ISI result in a greater latency shift in a particular wave as compared to the wave preceeding it. The "accumulation" effect was explained as the result of the converging and diverging tracts of the ascending CAS. Given the increasing numbers of neurons at progressivly higher levels of the CAS (Chow, 1951), the accumulation effect should result in wave five having the largest increase and decrease with changes in stimulus parameter. However, considering the relatively small dynamic range of the later waves (i.e., the "saturation" effect described above) Pratt and Sohmer (1976) suggest the following:

> "... a small number of active, lower order fibers activates a much larger number of higher order neurons (divergence) and each higher order neuron is activated by the synaptic contact with many lower order neurons (convergence). Thus, higher order neurons are already maximally activated at intermediate click levels. As the click rate was increased, the amplitude of N1 decreased, probably because of refractoriness, but the number of active fibers reaching the next nucleus may still have been sufficient to cause the smaller than expected decrease in amplitude of the response from higher order brainstem nuclei. Also as click rate was increased, a particular volley may be more effective since it may find some residual excitation remaining from the previous volley. Further evidence for these hypotheses comes from the observation that at low click levels, when the ... first wave ... is hardly apparent, the fourth wave ... is clearly seen, indicating that a very small number of primary auditory nerve fibers (insufficient to give a clear response in this recording) nevertheless succeed in activating a sufficient number of higher order neurons."

The foregoing may also be a plausible explaination for the prevalent finding that wave five is the largest and most easy to record at low stimulus intensities and is least affected by changes in ISI (e.g., Jewett and Williston, 1971). Naunton and Zerlin (1976) found that the N1 wave (BER wave one) intensity/amplitude function was not monotonic and, in fact, showed the same relative leveling and decreasing function at high intensities that Pratt and Sohmer report for the later waves. This function, however, was dependent on the frequency components of the stimulus. The amplitude of wave one is determined by three factors: a) the total number of activated neurons; b) their rate of firing; and, c) their degree of synchrony. The general increase in amplitude of wave one coincident with increases in stimulus intensities may be caused by two factors: a) increased firing of individual neurons; or, b) increased number of activated neurons that result from the broadening of the basilar tuning curve due to increases in intensity. The third factor, that of synchrony, is a function of the frequency of the stimulus and influences the amplitude of wave one in the following way: Bekesy's traveling wave has its greatest velocity at the basal turn (high frequencies), slowing progressively as it moves toward the apex. A greater number of hair cells are activated near the base in a very short period of time resulting in near synchrony of discharges (or more synchrony for higher as compared to lower

frequencies). The traveling wave for lower-frequency stimuli has a slower rate and thus results in a progressive departure from synchrony and the consequent decreased amplitudes.

For higher frequencies (8000 Hz), the wave one intensity/amplitude function is essentially monotonic (as suggested above) to approximately 70 dB, where its amplitude begins to decrease in comparison with lower frequencies (2000 Hz) of the same intensity. This "saturation" effect may result from a decrease in firing rate due to cells being driven beyond their maximum (Kiang, 1965). Thus, the difference between the intensity/amplitude function of low and high frequency components may be due to the broadened mechanical tuning curve for low frequencies resulting in a greater number of understimulated hair cells than the narrow curve for high frequencies.

Another possible cause of changes due to variations in ISI is normal refractory processes within the CAS (Pratt and Sohmer, 1976; Salamy et al., 1978; Simmons et al., 1966; Webster, 1971; and, Webster and Bock, 1971). This has been questioned, however, by Don et al. (1977). They found that the latency/ISI function of wave five is essentially linear between ISIs of 10 and 100 msec, while that of the eighth nerve AP (Eggermont and Odenthal, 1974) is logarithmic. This, they suggest, indicates that the input/output functions of the CT components are basically different from the PT components.

They state that the ISI effect is not due to refractory changes, but rather to one of incomplete recovery. The primary factors underlying recovery times include: a) receptor adaptation and/or fatigue; b) synaptic transmission changes; and, c) refractory periods. They eliminate refractory changes since this phenomenon has a shorter time course (1-2 msec) than the ISIs normally used. They attempt to eliminate synaptic transmission changes as a factor in a study in which changes in waveform stimulated by a monaural click sequence were not altered by coincident presentation of rapid clicks to the other ear. As they suggest, this does not eliminate the possibility that the click responses are transmitted via a monaural pathway (Babinghian et al., 1975). The third primary factor, and the one they favour, is adaptation and/or fatigue. If this is, in fact, the primary factor, it becomes difficult to explain the rapid latency shift to asymptotic values by the 3rd to 4th click (Thornton and Coleman, 1975), or the very little, if any, change in latency and/or amplitude in infants after more than 15,000 click presentations (Salamy and McKean, 1976). The possible cause underlying this ISI "effect", favoured by the present author, is still that of neural refractory processes. To suggest that since the time course of refractoriness of individual neurons is less than the rate of stimulation it does not contribute to the changes in the BER, one must assume that the CAS "fires" once and only one to every stimulation.

This is simply not true. As stated above, there is considerable evidence that shows individual neurons firing in bursts (after a single stimulation) for periods of time that may be "orders of magnitude" longer than the initial burst. Although no firm conclusion can be stated at present, the fact that little or no changes in either latency or amplitude occur with ISIs of more than 100 msec (e.g., Yoshie, 1968) also points to refractory changes, rather than adaptation or fatigue, at shorter ISIs.

The BER has a threshold between 10 and 20 dB HL (Yamada et al., 1977), and, Mitchell and Clemis (1977) have shown that audiograms derived from BER records compare favourably with conventional audiograms. One clinical consideration is that Coats and Martin (1977) have shown, in pathological cases, that the shape of the audiogram may influence the BER waveform regardless of lesion locus.

One of the basic problems in BER audiometry is that the clearest, unambiguous records are obtained from the presentation of broadband clicks. These, however, provide little information about the frequency specificity of the response (Jewett et al., 1970; Jewett and Williston, 1971; Naunton and Zerlin, 1976; Picton et al., 1974; and, Sohmer and Feinmesser, 1967). Brief and long tone bursts (Kodera et al., 1977; and, Terkildsen et al., 1975), filtered clicks (Davis and Hirsh, 1976), and, narrow bands of masking noise (Eggermont et

al., 1976; and, Elberling, 1974) have been used to compensate for these deficiencies with fairly good results. One of the on-going controversies in BER research is whether the wave peaks (particularly wave five) represents responses to components above or below 2000 Hz. To a certain extent the problem involves instrumentation (e.g., filter setting) and stimulus parameters, however, the camps are divided between those who feel that the BER can reflect all frequencies (e.g., Klein and Teas, 1978; Kodera et al., 1977; Suzuki and Horiuchi, 1977; and, Yamada et al., 1977) and those that see it as reflecting only high frequency activity (e.g., Davis and Hirsh, 1976; and, Manabe, 1976). Brama and Sohmer (1977), however, comparing filtered and unfiltered click stimuli, found that the absolute latency of the BER was dependent on the most prominent stimulus frequency component. This, together with BERs taken from subjects with various hearing loss, suggested that BERs could be obtained from low-frequency stimulation. It is interesting to note that, whereas most researchers have studied the wave peaks (i.e., one through seven), Klein and Teas, and Suzuki and Horiuchi studied the BER slow potential.

Although the correlation of BER records with electrocochleographic records has been questioned (Eggermont and Odenthal, 1974a), others have shown that the latency and amplitude functions of the BER changes slope between 40-60 dB SL (Davis and Hirsh, 1976; Yoshie, 1968; and, Yoshie and

Ohashi, 1969). This is similar to the slopes obtained in promontory or round window electrocochleography (Eggermont and Odenthal, 1974b; and, Portmann and Aran, 1971). Weber and Fujikawa (1977) and Zollner et al. (1976) have suggested that this change in slope may be the result of differential activity of the two populations of hair cells in the organ of Corti.

In general, the BER can be considered a good quantitative and objective measure of auditory sensory function (Picton et al., 1977; and, Starr, 1978) for frequencies below 2000 Hz (Kodera et al., 1977) as well as those in excess of 2000 Hz (Davis, 1976). It must be noted, however, that subjective estimates of loudness do not correlate well with BER levels (Bauer and Galambos, 1975; Coats and Dickey, 1972; Hecox, 1974; Hecox et al., 1976; and, Pratt and Sohmer, 1977). In other words, BER is not hearing. It has been suggested that either a higher level system functions to determine subjective loudness on a priority basis, or the levels obtained by BER records reflect the limits of the instruments used, and the anatomical effects on neural volume conduction (Hecox et al., 1976; and, Pratt and Sohmer, 1977). Or, in terms of wave one (Coats and Dickey, 1972), the temporal dispersion of the action potential may lead to a reduction in amplitude while the total number of active fibers remain constant.

BER GENERATOR SOURCE LOCATION

Interest in this auditory, non-invasive, electro-neural phenomenon stems from the possibility of accurately determining anatomically a series of EEG EP generator sources, obtaining information on the electrophysiological function of the CAS in both normal and pathological individuals, and applying this technique to clinical situations. Since the CAS has been shown to have response patterns that are extremely heterogeneous within each nucleus (Boudreau and Tsuchitani, 1968; and, Whitfield, 1967), it is probably simplistic to speak of the CAS or any of its constituent nuclei as having a unitary function. A number of different techniques have been used in attempting to delineate the anatomical structures involved in the BER. These include: a) correlations between specific scalp evoked potentials and single unit or depth recorded potentials; b) differential analysis of the distribution of each wave over the scalp and at other locations (e.g., nasopharynx and neck positions); c) correlations of the BER before and after destruction of specific tracts and nuclei in animals; d) correlation between normal human BERs and the BERs obtained from individuals suffering from known pathological conditions; and, e) studies of the maturational trends of the BER in both humans and animals.

Attempts to locate neural generator sources via scalp electrodes (the goal of BER research) is wrought with problems (i.e., the effect of volume conduction on the waveform, the effects of the meningies-skull-scalp combination, electrode position, instrumentation, averaging, etc.). This includes the hazard of seeing a particular nucleus as the source of a particular wave peak simply on the basis of temporal coincidence between single unit activity and scalp recorded potentials (Starr and Hamilton, 1976). This problem is probably best illustrated by Stevens (1974) in his list of the possible causes of amplitude changes:

> "If ... a surface positivity or negativity is decreased during a stimulus paradigm, it is impossible to determine from measurements of the surface potential whether the decrease was produced by: (a) a reduction of the amplitude of the presynaptic volley (i.e., a decrease in the number of cells activated); (b) a change in the synchrony of the presynaptic volley; (c) a cancellation of fields due to the generation of currents of opposite polarity at the same location as the test response; or, (d) a cancellation of fields due to generation of field potentials deeper in the structure which, having a bigger current spread, may obliterate more localized or more superficial field potentials" (2B-77).

Most workers have regarded the BER as a composite firing of complex generators including both ascending and descending fibers, and possibly post-synaptic slow waves, since both fast and slow waves can be recorded from a number of CAS nuclei via depth electrodes (Huang and Buchwald, 1977; Jewett et al., 1970; and, Ornitz and Walter, 1975, 1976). Starr and Hamilton

(1976) studying a number of patients suffering from a variety of brainstem pathologies (confirmed at autopsy), found the following correlations: whenever the brainstem is extensively disturbed or destroyed, including the extramedullary eighth nerve entrance, only wave one can be observed in the BER; whereas, waves four through seven require the existence of a normal midbrain. In particular, they placed the origin of wave one in the eighth nerve (possibly before it passes through the internal auditory meatus), wave two at an indeterminate point between the generators for wave one and wave three, and wave three within the combined medullary portion of the auditory system, including the cochlear nucleus, trapezoid body and superior olivary complex. A recent, very interesting study was carried out by Uziel and Benezech (1978) in which behavioural correlates of brainstem pathologies were compared with alterations in the BER. They state:

> "There is good correlation between flexion reactivity which requires the integrity of the brainstem ... and normal BER. Flaccidity which appears in brainstem lesions involving the inferior pons, always corresponded with a loss of the last three waves of the BER (3-5). Extension reactivity, classically identified with Sherrington's rigidity in animals, has in fact been observed even in the absence of anatomical brainstem lesions ... Our study confirms these findings since, out of 11 of our patients with this kind of reactivity, 8 showed BER abnormalities and 3 had normal BERs. Bilateral areactive pupillary dilatation has been observed in pontine or medullary lesions ... It is related in our study to abnormalities of waves P1-P3. The other pupillary abnormalities (myosis and absence of

> light reflex) have been related to mid-brain or diencephalic lesions. They were associated in our study with abnormalities in wave P5 in 40% of the cases, and with the absence of alterations of the BER in the other cases. We have found a good correlation between the presence of vertical automatic eye movements which require intactness of the mesencephalic tegmentum (periaqueductal grey matter) ... and the integrity of the 5 components of the BER. An abnormality of the 5th wave occurred in 60% of the cases in which no vertical eye movement could be observed: this suggests that the 5th wave originates from the midbrain (inferior colliculus). The integrity of the inferior pons is essential in the generation of horizontal automatic eye movement ... A good correlation exists between the disappearance of horizontal automatic eye movements and the loss of wave P3, which suggests that this wave originates from the inferior pons. The procedure is identical for the corneal reflex, which has the same origin."

Lesion studies with animals have shown similar findings to the above, with the expected inconsistencies of this type of gross experimental design (e.g., Goldenberg and Derbyshire (1975) found even bilateral ablations of the IC did not eliminate wave five although it did change the variability of the first three waves, whereas, Buchwald and Huang (1975) showed lesions of the IC eliminated wave five while any lesion above the level of the CN had no effect on waves one or two). The Buchwald and Huang study appears to be more in keeping with the correlation studies of Jewett and others. They found that mid-collicular decerebration had little effect on the first five waves, whereas bilateral aspiration of IC resulted in the elimination of wave five. Severing the efferents from the CN resulted in the elimination of all waves except one and two, while separating the CN from the acoustic nerve left only wave

one. Wave one, unlike the Cochlear Microphonic response, disappeared at death. With midline sagittal sections from the IC to the lower end of the CN, wave four and five decreased in amplitude by 50% or more, while wave three disappeared altogether. This suggests the relative dependence of generators of these waves on crossed and uncrossed projections. A more definitive locus of the wave four generators was determined in these sagittally sectioned animals: lesions of the ventral nucleus of the LL and of the adjacent pre-olivary areas resulted in the elimination of the remaining wave four potential. Allen and Starr (1978), using monkeys, presented somewhat similar data: waves one and five appeared to originate from single lateralized generators; waves two and three from bilateral generators; and, wave four from a mid-line structure. In another comparable study (Achor, 1976), depth recordings of isopotential contour maps indicated that wave two, four and five were generated by two or more sources, while wave three was generated by a single contralateral source, possibly the SOC or lateral lemniscus.

When studies are based on a particular combination of the above anatomical locations other conflicts arise. For example, Huang and Buchwald (1978) and Yoshie (1968) have suggested that the wave peak latencies are entirely determined by the latency of wave one, thus the CAS nuclei are simply relay centers in which no information processing takes place. Within each

nucleus of the CAS there are a group of constant "short-latency" units which are sensitive to onset but not duration (Huang and Buchwald, 1977). These units have been found in the CN (Rodinova, 1971), in the SOC (Galambos et al., 1959) and in the IC (Gersuni et al., 1971); at each CAS level they show a mean response latency which correlates well with the expected BER peak for that level. This would suggest the possiblity that the BER reflects the activity of "phase-locking" neurons, monosynaptically connected, that preset the system for the analysis of the stimulus in much the same way as trigger pulses do in signal averagers. If this is the case, any analytical neural activity not completely locked to the stimulus would cancel during averaging even if it was initiated by the stimulus. In clarification of this hypothesis, other workers have shown the BER to be sensitive only to the onset of the stimulus (Hallman, 1977; Hecox and Galambos, 1974; and, Hecox et al., 1976). Although Hecox et al. state that it is only the onset, not the offset nor the duration of the stimulus, that results in the BER, their use of a 2 msec tone burst may not have allowed enough recovery time for the system to respond, and would have resulted in an off-response wave five (if one exists) occurring beyond their duration of measurement. In any case, the on-response has been implicated as the primary cause of the eighth nerve AP (Goldstein and Kiang, 1958), the 8-50 msec AEPs (Lane et al., 1971; Beitter and Hegan, 1973; and,

Skinner and Antinoro, 1971), and, the 50-500 msec AEPs (Lamb and Graham, 1967; Onishi and Davis, 1968; and, Skinner and Jones, 1968). The hypothesis that the BER reflects only the on-response of the cochlea is not universally accepted. Kodera et al. (1977), using tone bursts of fairly long durations, showed a BER waveform at both the initiation and cessation of the burst. The off-response showed different latency and amplitude functions, as compared with the on-reponse, for changes in stimulus intensity. This, they suggest, may be due to adaptation of the system by the preceeding components of the tone burst. It might be just as reasonable to assume refractory changes; however, more work is needed to understand the process involved in this "off-response".

Since it has been shown that significant variability within each wave peak (two to seven) does exist (Hallman, 1977) and that the between peak latency is not fixed when certain parameters are changed (Hallman, 1977; Ornitz and Walter, 1975, 1976; Pratt and Sohmer, 1976; and, Zerlin and Naunton, 1973), a second possibility, as suggested by Yoshie (1968), is that neural impulses originating from the apical turn of the cochlea are delayed by the travel time of Bekesy's traveling wave and, therefore, tend to cancel rather than summate during averaging. This results in a cancellation of all potentials occurring after the initial basal potentials for each click, and is compatible with studies that indicate, by the use of masking

noise, that the compound action potential (Teas et al., 1962) and the BER (Hecox, 1974; and, Starr, 1978) reflect only the function of the basal turn of the cochlea. Similar to Yoshie, Teas et al. felt this was due to a "superior" synchronization of the APs originating from the basal turn where the velocity of the Bekesy traveling wave is greatest.

Even in the area of eighth nerve APs, long the subject of electrocochleographic study, there is conflict. Those studies reporting eighth nerve pathologies (Daly et al., 1977; Sohmer et al., 1974; and, Starr, 1978) show changes that seem confined to either or both of the first two waves; however, some studies show only amplitude changes while others show both amplitude and latency changes. Thus, although researchers are becoming quite precise in locating the general area generating each BER wave, there is still much to be studied before the functioning of the CAS, and how this relates to changes in the BER, is adequately understood.

Stockard and Rossiter (1977), in overview, have suggested the BER positive peaks result from complex neural structures within the following areas: wave one, the acoustic nerve; wave two, the pontomedullary junction; wave three, the caudal pons; wave four, the rostral pons or midbrain; wave five, the midbrain; wave six, the thalamus; and, wave seven, the thalamus or auditory radiations.

BER MATURATION

The BER has been recorded in premature infants (Schulman-Galambos and Galambos, 1975), neonates (Lieberman and Sohmer, 1973) and young children (Hecox and Galambos, 1974, Lieberman and Sohmer, 1973). Studies have shown a very definite maturational progression that, in humans, reaches adult form somewhere around 18-30 months of age (Mokotoff et al., 1977; Salamy and McKean, 1976; Salamy et al., 1975; Schulman-Galambos and Galambos, 1975; Shah et al., 1978; and, Starr et al., 1977). At birth, wave five shows a prolonged latency and diminished amplitude that matures to adult amplitudes by 12 months (Lieberman and Sohmer, 1973) and adult latencies by 18 months (Hecox and Galambos, 1974). A similar trend has been shown for rat pups and kittens (Jewett and Romano, 1972). In comparison, wave one achieves adult latencies by 7 months (Hecox, 1976) suggesting that the PT components mature before the CT components. The CT component shows three definite stages of development (Salamy and McKean, 1976): an initial abrupt change between birth and six weeks; a period of quiescence through the 6th month; and, a period between six months and one year when latency values are reduced by as much as 0.5 msec. The CT maturational trend has been explained as either a reflection of developmental myelination or synaptogenesis. One series of experiments that attempted to

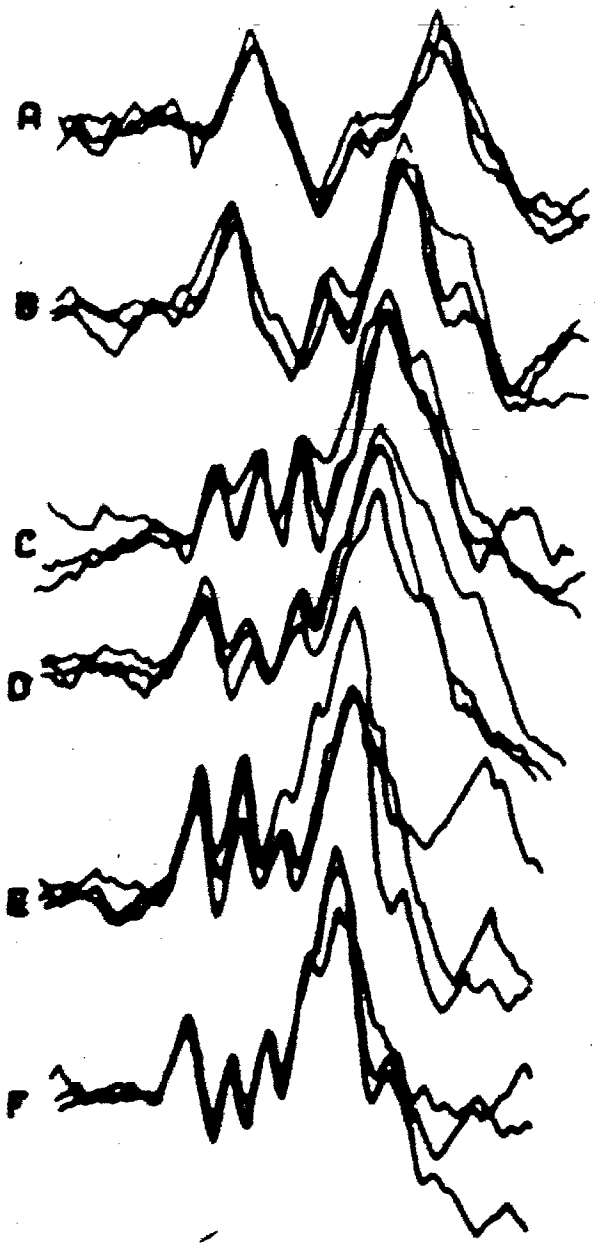
establish the sensitivity of the BER to rates of myelination (Shah et al., 1978) experimentally induced myelin deficiency in animals. The latency prolongation of the wave peaks not only correlated well with those found in infants but also with those found in patients with demyelinating deseases (Starr and Achor, 1975; Starr and Hamilton, 1976; Stockard et al., 1976; Stockard and Rossiter, 1977; and, Wiederholt et al., 1977).

The PT trend has been assumed to either involve maturation of the middle ear or changes within the cochlea itself. Since high frequency components become less effective as maskers the younger the infant is (Hecox, 1976), it has been suggested that there is a maturational progression, in the sensitivity of the cochlea to high frequency stimuli, from an original apical position toward the base. This would, in part, explain the develcpmental shift in wave five toward shorter latencies and greater amplitudes. In support of Hecox's hypothesis, Bredberg (1968) showed in his sample of human newborn cochleas, that the most basal portion was not mature. It is important to note that it is vary difficult to establish the extent of PT contribution to CT maturational changes (Jewett and Romano, 1972), because of the ccnfounding of the PT component by the eighth nerve activity. What ever the cause of BER maturation, it is now possible to both determine if the peripheral and brainstem divisions of the auditory system are functioning in a child as young as a few days of age, and to determine, over

time, if the system is developing normally. This technique can be used with all infants as a first line screening technique for auditory problems (Mokotoff et al., 1977). One advantage of the use of BER techniques with infants is that BER thresholds are significantly lower than that obtained by heart rate change for auditory responsivity (Schulman, 1973; and, Schulman-Galambos and Galambos, 1975). Figure 5 from Salamy and McKean (1976) shows a typical infant BER maturational development.

CLINICAL APPLICATIONS

There is a growing literature that suggests the BER to be a good measure of not only early onset of pathology, but also of the progression, regression, size and locus of such pathologies (e.g., Daly et al., 1977; McDonald and Sears, 1970; Robinson and Rudge, 1977; Rosenhamer, 1977; Selters and Brackmann, 1977; Simon et al., 1976; Sohmer et al., 1974; Sohmer et al., 1977; Sohmer and Student, 1978; Starr, 1978; Starr and Achor, 1975; Starr and Hamilton, 1976; Stockard et al., 1976; Stockard and Rossiter, 1977; Terkildsen et al., 1977; Thornton and Hawkes, 1976; and, Wiederhold et al., 1977). Whether with adults or infants, the BER can be used as evidence of CAS excitation and, by means of derived expected latency curves for wide band clicks, can be used to estimate the extent



BER MATURATION

A = NEWBORN
B = 6 WEEKS
C = 3 MONTHS
D = 6 MONTHS
E = 1 YEAR
F = ADULT

FIGURE 5
(SALAMY AND MCKEAN, 1976)

of high frequency hearing loss (Weber and Fujikawa, 1977). The important feature is that motor responses are not required of the patient, thus the particular level of cognitive functioning is not important.

The clinical use of the BER is still in its infancy and at times may be more confusing than helpful. For example, a commonly found abnormality in the BER record is a reduction in wave peak amplitude and an increase in latency. However, this is not univeral since MS typically results in latency increases with no amplitude changes, whereas tumours or compressive lesions may result in amplitude reductions and no latency changes. However, used as part of a larger diagnostic battery, the BER can provide valuable information for both audiometric evaluations and clinical assessments of the integrity of the CAS and brainstem.

BER ELECTRODE POSITION

In general, the electrode derivations that have been used to record the cochlear AP and/or BER include active electrodes in or at: a) the tympanic membrane (Cullen et al., 1972; and, Pratt and Sohmer, 1976); b) the external auditory meatus (Coats, 1970; Salomon and Elberling, 1971; and, Megill et al., 1975); c) the promontory (Portmann et al., 1968; Yoshie, 1968; and, Eggermont et al., 1974); or, d) the scalp (e.g., vertex,

forehead, mastoid, earlobe and nasopharynx) (Hecox and Galambos, 1974; Jewett et al., 1970; Martin and Coats, 1970; Sohmer and Feinmesser, 1967; and, Thornton, 1975). In all cases, whether recording the cochlear AP or the BER, it is reasonable to assume that all electrode derivations are recording the same volume-conducted potentials.

Many of the experimental parameters one uses indicate particular biases in orientation. BER recordings are no exception. Vertical conduction through the brain is accomplished in an easier manner than is horizontal conduction. The recorded electrical activity is a combination of the algebraic summation of the volume conducted potentials at each electrode; and, the difference between any two electrodes which result from the particular capacitant and resistant properties the conducted current must encounter before reaching the respective electrodes. In the CNS, the average dielectric constant (capacitance) and resistant values of the tissue is neither isotropic not homogenous. These differences (which in orthoganal directions can differ by a factor or 5 to 10) reflect both the geometric and architectonic organization of the CNS (Dondey and Gaches, 1977). It is, therefore, not surprising that the vertex shows the largest waveform (Streletz et al., 1977; and, Terkildsen et al., 1974).

The earlobe, mastoid and nose have been found to be active during auditory stimulation in humans (Picton et al., 1974;

and, Streletz et al., 1977), while no cephalic indifferent points were found in the rat (Plantz et al., 1974) or monkey (Allen and Starr, 1978), thus the placement of the "reference" electrode must also be carefully considered. Plantz et al. (1974) studied the effects of moving the reference electrode to a number of sites and concluded that both the positive nature and the actual form of the BER was dependent on electrode position. If the peaks result from the activity of a number of different sources, it may be difficult to find one location that adequately resolves all of them. Since Smith et al. (1973) and Martin and Coats (1973) have shown that the generators of the peaks mimic particular deep dipoles, it may be more reasonable to record from a number of different sites if one is to measure all components of the BER adequately. This is particularly true since Picton et al. (1974) found that wave two minics a transverse horizontal dipole, whereas wave three mimics both a horizontal and a vertical dipole. Terkildsen et al. (1974) presented data that suggest the cochlear microphonic is best recorded using a mastoid/ contralateral side of the neck derivation, the BER is best recorded with a vertex/mastoid derivation, whereas, the fifth wave is best recorded from a vertex/ ipsilateral side of the neck derivation. Coats and Martin (1977) have demonstrated that nasal pharyngeal electrodes produce waveforms that have greater amplitudes for waves two and three although there is no

latency shift in the waves between this derivation and the classical vertex derivation.

BER PROBLEMS

The BER is, as suggested above, very sensitive to both recording and stimulus parameters. In addition, the way in which one determines peak latency and amplitude necessarily effect the resulting conclusions. There appears to be a great deal of methodological and comparative problems in this area of research, problems that would seem important to consider. The following is a listing of some that either make it difficult to compare between studies or result in some question as to the adequacy of the findings.

a) stimulus problems

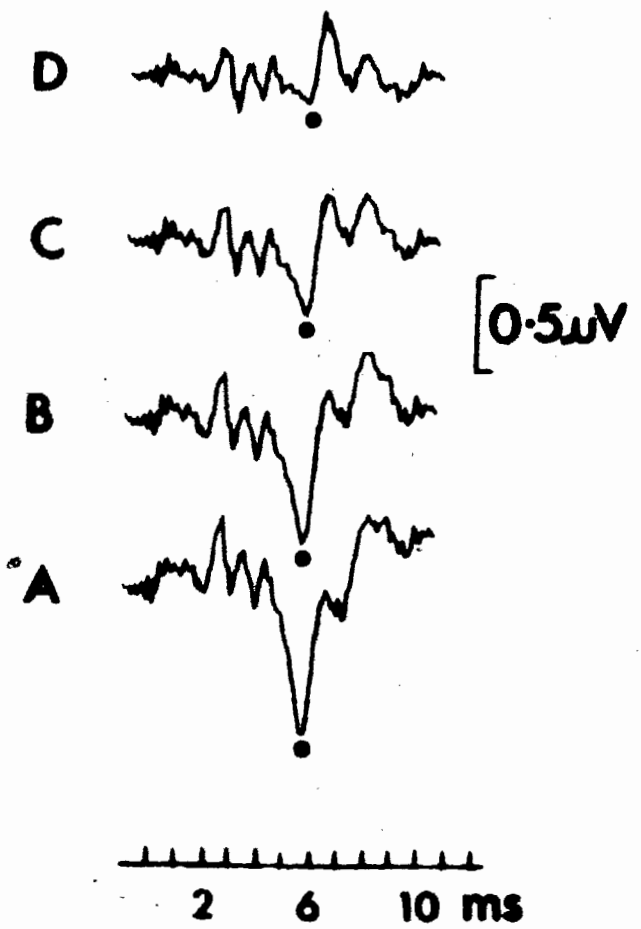
Stimulus parameters, at times, appear to be a matter of convenience rather than one of design. The problem arises when the researcher attempts to compare his results with those in the literature. If the stimulus parameters are disparate, with no factual or theoretical reason for comparison, extrapolations must be carefully considered.

The stimulus may be presented as a click (normally varying from 0.05 msec to 0.5 msec or more), as a tone burst described as a certain rise time (i.e., 4.5 msec), frequency (i.e., 1000

Hz), and, duration, or as a filtered click (i.e., 3000 Hz to 9600 Hz bandpass). The amount of variation among these stimuli is dependent on the particular transducer used; headphones, earphones or speakers (a great deal of difference exists between transducers in both the form and the duration of the sound wave). The ISI may vary from more than 1000 msec to less than 20 msec and may be presented with fixed intervals or randomly around a particular interval. The intensity of the stimulus may be described as any one of the many types of dB, from dBs above the subject's threshold to a constant sound pressure level, and may vary from threshold to 90 dB or more. Stimuli may be presented monaurally (to either ear) or binaurally, and the initial movement of the diaphragm may result in either a rarefaction or a condensation wave (many studies have used an alternation between the two to eliminate electrical transducer artifacts). Finally, the subject may be either human or animal. Although all of these are quite proper stimulus parameters, comparison between parameter combinations without consideration of the differences is suspect. Probably the most serious problem is the failure to report the complete combination used, such as type of transducer, duration of click, whether the stimulation is binaural or monaural, and, if monaural, to which side, etc.

b) recording problems

Recording problems are probably more theoretical than practical, however, they again inject some question into the presented data. Probably the most problematic is that of filter setting. In most recordings, the BER has a slow component (3 msec < frequency > 10 msec), a component that some feel to be extraneous to the BER and, therefore filter it out (i.e., set highpass filters greater than 100 Hz). This results in an obvious change in amplitude measures (see Figure 6) and, quite possibly, latency measures. The BER is composed of at least seven peaks that occur approximately one msec peak-to-peak, therefore, to resolve this waveform adequately by use of an A-D convertor, the lowpass setting should include 1000 Hz, and the sampling time per point must double this frequency (i.e., 500 usec). As one attempts to resolve more and more detail by adjusting the lowpass filter, the sampling time must also be changed. It should be remembered that, theoretically, a filter affects all components differentially, the phase of a 1000 Hz signal is not the same when subjected to a bandpass of 10-3000 Hz as it is when subjected to one of 300-10,000 Hz. Again, a serious problem is the failure to include the necessary comparative information. The following are a few of the studies that either have intrinsic problems, or are simply difficult to compare with other research:



EFFECT OF LOW-FREQUENCY FILTER ON THE BER

A = 0.8 HZ
B = 100 HZ
C = 200 HZ
D = 250 HZ

FIGURE 6
(SUZUKI AND HORIUCHI, 1977)

1) Amadeo and Shagass (1973) 10-80,000 Hz bandpass, 31.25 usec per point; low pass filter should have been set to 16,000 Hz.
2) Buchwald and Huang (1975) 30-3,000 Hz bandpass, no A-D information.
3) Goldenberg and Derbyshire (1975) 10-3,000 Hz bandpass, 200 usec per point; low pass filter should have been set to 2,500 Hz.
4) Hecox (1974) no information.
5) Portmann and Aran (1971) 1-30,000 Hz bandpass, 100 usec per point; low pass filter should have been set to 5,000 Hz.
6) Salamy and McKean (1976) 10-10,000 Hz and 30-10,000 Hz; no A-D information.
7) Sohmer and Student (1978) 250-5,000 Hz bandpass, no A-D information.
8) Terkildsen et al. (1974) 0.5-4,500 Hz bandpass, no A-D information.
9) Terkildsen et al. (1977) 200-4,500 Hz bandpass, no A-D information.
10) Wiederholt et al. (1977); no information.

Electrode position does not appear to be a serious problem since the majority of studies have used a vertex/mastoid derivation. However, with the increased awareness of cephalic active 'reference' sites, and the use of forehead-hairline

active electrodes in clinical settings, this may become a problem in the future.

c) measurement problems

Latency measures are relatively consistent across studies, only varying in what is considered zero time. Amplitude measures, on the other hand, vary considerably:

1) Amadeo and Shagass (1973); amplitude was measured in uV between an estimate of the isoelectric line, as determined from the baseline preceding the stimulus mark. Latency was measured as time to peak after the earphones were stimulated.
2) Goldenberg and Derbyshire (1975); a Z-score for each point was determined by obtaining an average of 700 responses, subtracting the average at each point from the mean of the points sampled prior to the stimulus, and, dividing this difference by the SD of the distribution of prestimulus points.
3) Huang and Buchwald (1978); a mid-point amplitude was used between the rising and the falling phase of each component as an average measure of peak-to-peak amplitude.
4) Picton and Hillyard (1974); a baseline was determined from the first 0.5 msec of the response and extended through the succeeding evoked potential components. If

such a baseline was too difficult to evaluate because of stimulus artifact contamination, an arbitrary baseline was drawn at the midpoint between the troughs following wave one and two.

5) Portmann and Aran (1971); for the amplitude, a linear scale was used, expressed as a percentage of the maximum amplitude obtained during the test.

6) Robinson and Rudge (1977); amplitude of wave components two, three, six and seven were measured to the preceding upgoing peak, and that of component five to the upgoing peak preceding wave four. The amplitude of wave one was measured to the baseline as estimated from the pre-stimulus level. Latency was measured from the onset of the click to the downgoing peak of each component.

7) Rowe (1978); amplitude of wave five was measured from the positive peak of wave five to the following negative trough.

8) Sohmer and Student (1978); CT latencies were measured as the time interval from the negative peak of the auditory nerve response to the positive trough of the response from the inferior colliculus.

9) Thornton and Hawkes (1976); peak-to-peak amplitude was measured as the difference between a particular negative peak to the succeeding positive peak.

d) individual research problems

It is quite probable that most research is plagued with one or more methodological problems. Whether these problems are implicitly or explicitly stated, or not, it is important that BER research be read from a questioning point of view. Following are examples of the type of problems that may be encountered:

1) Amadeo and Shagass (1973); the parameters used in this study result in a waveform in which wave four or wave five is not observable. This, in of itself, is not a particular problem; however, they labelled this peak as wave four. Many studies have shown peak five to be present even in the absence of peak four.

2) Huang and Buchwald (1977); these workers determined the latencies of wave two to five for tone bursts in which the actual duration of the bursts remained constant while the tone varied from 500 Hz to 10,000 Hz. They report that the latencies did not differ significantly (wave two +/- 0.4 msec, wave three +/- 0.3 msec, wave four +/- 0.4 msec, and wave five +/- 0.4 msec). This, they suggest, indicate the presence of short-latency components that vary little with changes in stimulus parameters. What they did not consider, however, is the number of reports suggesting that the BER is sensitive to only the onset of the stimulus. Thus, the difference observed would not be expected to vary a great deal since the onset of a

10,000 Hz tone burst to its first peak is less than 0.5 msec faster than a 500 Hz tone burst.

3) Plantz et al.(1974); this very complex study of the volume spread of BER components over the scalp, based its recording parameters on a theory of Plonsey's (1969). This theory suggests that volume conduction properties of living tissue are essentially resistive up to frequencies of 10,000 Hz. Since they were sampling at a rate of 100 usec per point, they considered that, in terms of Shannon's sampling theorem, they only retained information to 5,000 Hz, well below Plonsey's limit. They concluded, therefore, that any latency differences found at various locations on the head would be due to a change in dipole orientation and not to the electrical properties of the conducting tissue. Plonsey, however, only considered lung, fatty tissue, liver and heart muscle; not CNS tissue. In addition, nowhere in his writings does one find any consideration of the capacitant qualities of the meningies, skull, or scalp layers, or of the non-isotropic and non-homogenous organization of the brain (Clark and Plonsey, 1968; Clark and Plonsey, 1970; Clark and Plonsey, 1971; Heppner and Plonsey, 1970; and, Plonsey, 1969). Since they set their low pass filters at 10,000 Hz and sampled at 10,000 Hz, according to Nyquist, they introduced the possibility of data contamination by alias frequencies.

4) Robinson and Rudge (1975); these workers state:

> "estimates of amplitude and latency are made at the turning-points of the evoked potential waveform which merely reflect the point of maximum synchronization of many interacting fibers."

This is simply not proven. As stated above, the possible causes of a particular peak, considering the state-of-the-art, is infinite (or almost so).

5) Shah et al. (1978); they begin with the premise:

> "there is now little doubt that brain stem activity can be reliably recorded from the surface of the scalp in animal and man, and that the various waves which make up the complex "far-field" response originate primarily from spatially separate structures in the classical auditory system ... although it was suggested that some waves arise from activation of multiple local generators in algebraic summation (Achor, 1976; Jewett, 1970) there is no question that they represent different levels of brain stem integration (Wiederholt et al., 1977)."

Wiederholt et al. (1977), however, only studied three patients, with clinical evidence of central pontine myelinolysis, one of whom died of complications of the pathology. BERs were recorded from only two patients from which they concluded that the waveforms indicated an impairment of function in the auditory pontine pathway; a somewhat hazardous conclusion considering their sample size.

CHAPTER THREE

BINAURAL INTERACTION IN THE CAS

The first comprehensive theory of the neural integration of binaural signals was developed by von Bekesy (1930).

> "He conceived of a region in the CNS that receives inputs from the two sides. The "characteristic of direction" arises when impulses from one side "tune up the cells there" to that particular side. If "all the cells" are activated by stimulation presented to one ear, then the sound is judged to be in the extreme lateral position. When the two sounds are applied simultaneously, the two "excitations" fall in the middle of the cell group, the transmission of excitation stops, and the cell group can transmit its directional relation to a higher level" (Erulkar, 1972).

At a very simple level, localization of sound in space (or lateralization if headphones are used) is accomplished by analysis of the difference in time or intensity of dichotic (or diotic) stimuli (Mast, 1971). In a general sense, low-frequencies (< 1500 Hz) are located by disparities in time, while high-frequencies (> 1500 Hz) are located by disparities in intensity (Jeffress, 1975). The processes involved are not quite so simple, however, since the CAS has been shown able to extract temporal information from the "envelope" of the

stimulus waveform when this envelope contains only high frequencies (Jeffress, 1975); and, with headphones, differences in intensity may be detected even for low-frequencies (Mills, 1960). Jeffress concluded that there are two CAS mechanisms operating: a) units that phase-lock to the stimulus, firing at particular points in the sound cycle; and, b) units whose firing is determined by the level of the stimulus, independent of the frequency. It is important to make the distinction between the psychophysical properties of the ear and the neurophysical properties of the CAS. Whereas both time and intensity interact in the initiation of neural action potentials (Deatherage and Hirsh, 1959; and, Hallman, 1977), once initiated, localization is entirely dependent on the neural interactions of temporal patterns. The loci of these interactions appear to be spread throughout the CAS (Rosenzweig, 1961), as suggested by the following:

A) Interactions at the level of the Cochlea

The first possible site of binaural interaction is within the cochlea where the efferent system, initiated via stimulation of the contralateral ear, may influence afferent activity (Gulick, 1971). Galambos et al. (1950) were able to show evidence for the presence of a cochleo-cochlear pathway in cats, although the minimal transmission time (1.25 msec) was too long for the mediation of localization in most cases

(Walsh, 1957). No evidence for an equivalent pathway has been shown in humans.

B) Interactions at the level of the Cochlear Nucleus

The second possible site is that of the CN, although Gulick (1971) stated that no binaural interaction has been observed at this level. Rasmussen (1960) described connections of the trapezoid bodies with the lateral superior-olivary nuclei, which have direct efferent connections with the contralateral VCN. Thus, a direct link does exist between the two VCN via a chain encompassing only two synapses. Similarly, Mast (1971) showed that, in Chinchillas, binaural interaction can take place at the level of the DCN, mediated, presumably, by centrifugal pathways. Pfalz (1962) showed that deafferented CN cells are inhibited (never excited) by auditory stimulation of the opposite intact cochlea. He concludes that the role of this interaction remains obscure.

C) Interaction at the level of the Superior Olivary Complex

The SOC has been the most studied in terms of the localization of sounds in space; it is probably the lowest level of functionally significant binaural interaction (Moushegian et al., 1972). SOC cells receive short-latency inputs from the CN (Brugge and Geisler, 1978), and respond differentially to dichotic clicks (Hall, 1965; and, Moushegian

et al., 1964). Moushegian (1971; in Jeffress, 1977) has shown, in agreement with Jeffress, that the SOC contains two groups of fibers; one whose firing rate is completely determined by the frequency of the stimulus, and another whose rate of firing is determined by the difference in level between stimuli, independent of frequency.

1) The Lateral Superior Olive (S-Segment)

Within the S-segment, Boudreau and Tsuchitani (1968) have shown that, with the exception of cells with characteristic frequencies (CF) below 1000 Hz, contralateral, simultaneous stimulation will inhibit the majority of cells. Both the contralateral and ipsilateral CF's tend to be the same, and are tonically acting. The AP of the S-segment is not a function of the absolute stimuli intensity, but rather the relative dichotic intensity difference.

2) The Medial Superior Olive (Accessory Nucleus)

On the basis of anatomical data (Stotler, 1953), physiological data (van Bergeijk, 1962; Galambos et al., 1959; Moushegian et al., 1964; and, Tsuchitani and Boudreau, 1964), and behavioural data (Masterton et al., 1967; and, Masterton et al., 1968), the medial superior olive (MSO) has been implicated in the localization of sounds in space. Frequently regarded as a nucleus whose main function is to encode interaural time differences (Brugge and Geisler, 1978; Galambos et al., 1959; Goldberg and Brown, 1969; Masterton et al., 1967; and,

Masterton et al., 1968), there is considerable evidence pointing to its role in the encoding of intensity differences as well (Gulick, 1971; Hall, 1965; Hind et al., 1963; and, Worden et al., 1966). The activity of the MSO units show, in fact, the expected cancellation of time and intensity trades.

The MSO receives symmetrical and equal innervation from the CN; these terminating on the opposite sides of the same cell (Gulick, 1971). Stotler (1953), in cats, showed that MSO cells have two large dendrites that extend approximately 200u horizontally in opposite directions. Projections of the contralateral CN reach the medial dendrites, while projections of the ipsilateral CN reach the lateral dendrites; thus a polarity exists between CN inputs. When a particular dichotic stimulation results in a convergence of CN inputs on one MSO cell, the cell fires maximally. As the locus of the stimulus changes, the inputs do not converge and the cell firing rate decreases. For some of the MSO cells, the best interaural time delay is frequency independent (Moushegian et al., 1971), thus they are able to decode and encode information from a complex low-frequency sound source. In addition, the presence of a population of such cells suggests that the MSO involves a place mechanism for this decoding/encoding (Goldberg and Brown, 1969; and, Rose et al., 1966). Although it is common in CNS sensory systems to find topography preserved in successive populations of neurons, in this case the CNS representation of low-

frequency auditory space is created by temporal and spatial interactions at the level of the MSO.

The MSO units exhibit intrafiber volleying patterns with intervals which are the same as, or multiples of, the stimulus period (Deatherage and Hirsh, 1959). Galambos et al. (1959) found a slow wave component of the MSO AP. This slow wave was negative, as recorded by a depth electrode placed ventromedial to the MSO, when contralateral stimuli were presented, and positive when ipsilateral stimuli were presented. Their explanation for this phenomenon was that contralateral stimulation results in a partial depolarization of the medial dendrites, while ipsilateral stimulation results in partial depolarization of the lateral dendrites. Since the cell body acts as a sink, the recorded wave for these two types of stimulation would have opposite polarities. This phenomenon of phase reversal in the region of the SOC has been reported by others (Moushegian et al., 1964; Hall, 1965; and, Tsuchitani and Boudreau, 1964), and, with animals, is used as an indicator that depth electrodes are within the vicinity of the MSO. The slow wave pattern has been shown to be distinctly different at each point within the MSO, and to change depending upon whether the stimuli are rarefaction or condensation clicks (Galambos et al., 1972).

The above discription of MSO functioning is a good fit to a theory of Jeffress (1948) in which he suggested that

interaural time differences may be decoded as a difference in place. Sets of binaural neurons were hypothesized which receive monaural inputs from either ear. Each input reaches the cells after a transmission delay that is a function of nerve fiber conduction rates and spatial summation of PSPs (both functions of stimulus intensity parameters), and interaural time parameters. Each cell functions as a coincidence detector; thus, a difference in neural conduction time is represented as a difference in place. It is necessary to assume that the cells only fire when, and if, they receive simultaneous excitation from the two sides, however, this model easily handles both temporal and intensity disparities.

Binaural beats, considered to be an indication that the CAS preserves information about stimulus phase, are also likely to result from a function of the MSO (Wernick and Starr, 1968). Using a subtraction method to determine binaural interaction in the SOC of cats, these researchers found that when tonal stimuli of differing frequency were presented, an envelope periodicity equal to the frequency difference between the two tones resulted. The frequency of the binaural beats is known to be the difference between the frequencies of the stimuli presented.

D) Interactions at the level of the Inferior Colliculus

Nauman (1958, in Endo, 1976) presented evidence suggesting little or no binaural interaction at the level of the IC or above. The IC appears however, to have a primary function in the processing of interaural time differences, which is consistent with its direct links with the MSO. Erulkar (1959) found 60% of his sampled cells had latencies dependent on the azimuth of the sound source. Similarly, Rose et al. (1966) found cells in the IC of cats whose rate of firing changed as a function of interaural time difference. Nelson and Erulkar (1963) found groups of cells that either changed their PSP configuration, or latency, depending on which ear was stimulated. They also found, by changing the time delay of dichotic stimuli, that the inputs could be made to interact either in an inhibitory or facilatory manner.

BINAURAL INTERACTIONS REPRESENTED IN THE BER

If the BER wave peaks do represent the activity of auditory brainstem nuclei, any binaural interactions occurring in these nuclei should be represented in the recorded BER, given that these interactions are consistent and the instrumentation used is of sufficient sensitivity. Three

procedures have been used to investigate these hypothesized interactions: a) comparisons between binaural BERs and summed monaural right and left BERs; b) comparisons between rarefaction and condensation BERs; and, c) comparisons between monaural right and left BERs.

In general, studies comparing binaural with summed right and left monaural BERs show little or no latency shift in the peaks, although amplitude changes do occur. Gerken et al. (1975), comparing frequency following responses (FFR) rather than BERs, found no binaural/monaural differences. They concluded that the resulting binaural waveform was simply the sum of two monaural FFRs (representing two independent populations of neurons), and that a vertex/mastoid derivation is not sensitive to auditory binaural interactions. Starr and Achor (1975) also concluded that there are no substantial BER differences between monaural and binaural stimulation, although a comparison of the waveforms presented in their article shows the amplitude of binaural BERs to be somewhat less than twice the amplitude of monaural BERs. This finding has been seen in our lab, and is of interest since it suggests that the auditory projections from each ear do, in fact, have common neuronal populations. This is consistent with the discussion above concerning CAS convergence and divergence. Huang and Buchwald (1978), using cats, found a decrement in binaural wave four amplitude as compared with summed monaural amplitude, although

no change was observed in waves one, two or three. Endo (1976), using polarity alternating clicks with humans, found both a decrease in amplitude and latency for binaural wave five. Using the subtraction technique developed by Wernick and Starr (1968), he found a negative "interaction" peak that occurred approximately 0.3-0.4 msec after the wave five peak.

Since it has been shown that the first peak of firing in single units of the eighth nerve occurs sooner after rarefaction clicks than condensation clicks (Goblick and Pfeiffer, 1969; Kiang, 1965; and, Salomon and Elberling, 1971); and, that the MSO units are differentially sensitive to these two types of stimuli, it is reasonable to expect the BER to reflect these differences. The earliest studies were by Ornitz and Walter (1975, 1976), in which they showed a phase shift in the first five waves for a percentage of their subjects. The greatest difference was observed for wave four; in all cases, latencies were shorter for rarefaction than for condensation clicks. Hallman (1977) presented similar results, although in this case the precedence effect of rarefaction clicks reversed by wave four. He concluded that the probable cause was neuronal conduction speed rather than any specific information processing. Coats and Martin (1977), using subjects with high frequency hearing loss, showed waves two to four to be out of phase, with little or no difference for wave five. They concluded that their findings were compatible with a dual

system view of the component sources of the BER. One system results in a slow wave "whose maximum vertex positivity creates peak V", and a second system that results in "a series of four or five oscillations ... creating peaks II to VI". Rosenhamer et al. (1978), however, could find no significant latency difference in wave one, three or five, whether the stimulation was condensation, rarefaction or alternating clicks. They were also unable to find a change in the peak-to-peak amplitude of wave five as stimulus phase changed.

Since the SOC appears specifically tuned to differentiate between left and right stimuli (Clark and Dunlop, 1968; Galambos et al., 1959; Tsuchitani and Boudreau, 1964; and, Worden et al., 1960), it is reasonable to expect some difference in the shape, amplitude or latency of BER wave three, or the negative peaks on either side. Martin and Coats (1973) and Coats and Martin (1977) have demonstrated that nasal pharyngeal electrodes produce waveforms that have greater amplitudes, and better definition, for waves two and three when ipsilateral clicks were presented; however, wave three becomes almost nonexistent when contralateral clicks were presented. Little other evidence has been shown in the literature; in fact, as stated above, interaural differences greater than 0.2-0.3 msec are considered to be an indication of pathology.

In summary, it appears that the contribution of binaural interaction, or left/right differences, to the BER is far from clear. The following study was designed to provide some clarification of the representation of binaural integration in the BER. If this neural processing can be clearly observed then increased understanding of the neural generators producing the peaks will have been ascertained.

CHAPTER FOUR

This study used two approaches to ascertain the extent to which binaural processing in the CAS is reflected in the BER: 1) a comparison of binaurally presented condensation (C) and rarefaction (R) stimuli. Four phase conditions were used (R-right, R-left; R-right, C-left; C-right, R-left; and, C-right, C-left). At the outset it was only possible to speculate on the types of changes expected since no research had been published in which the binaural nature of the four phase pairs had been studied. In terms of the whole BER waveform, since the majority of monaural studies comparing rarefaction with condensation stimuli had shown latency shifts with R initially evoking shorter latencies, a similar pattern was expected when comparing RR (right-R and left-R) with CC waveforms. However, since the only difference between RC and CR stimuli is the lateral position of the two phases, and since it is reasonable to assume the anatomy of the CAS to be bilaterally symmetrical, no difference between the evoked waveforms (as seen by a vertex electrode) was expected. In general, differences in latency, as well as amplitude, found in the early waves resulting from changes in phase were expected to be reflected in at least the first five waves, as has been shown for monaural stimulation (Coats and Martin, 1977;

Hallman, 1977; and Ornitz and Walter, 1975, 1976). In terms of peak-specific effects, since Galambos et al. (1959) were able to demonstrate differential responding of the SOC to R and C stimuli, changes in latency and/or amplitude were expected for peak three.

2) The comparison between a binaurally evoked BER waveform, and the algebraic sum of two monaural waveforms. If the BER reflects binaural integration, a comparison between binaural and combined monaural waveforms should result in the following differences:

a) decreased binaural amplitudes at those peaks representing neural integration. This is based on the assumption that binaural activation of a CAS structure will not produce the same amplitude as the algebraic summation of a left and right monaural activation of the same structure. If a particular wave peak is found to have a smaller amplitude when evoked by a binaural stimulus than that produced by algebraically summing responses to two equivalent monaural stimuli, it can be argued that this peak is an indicator of binaural integration. If the neural inputs from the two cochleas, through neuronal divergence (a known auditory phenomenon) results in the activation of binaurally innervated neuronal populations at higher levels, then stimulation of the left or the right ears separately will result in some activity within these populations. However, stimulation of both ears simultaneously

will result in a different contribution to the BER due to the overlap of the activation of these populations. It follows that for those generator sources in which binaural neuronal populations exist, the BER evoked by binaural stimulation should not equal the sum of the responses to separate right and left monaural stimulus presentations: Binaural RL < Monaural R + Monaural L. If there are, in fact, two separate monaural systems involved, or if no binaural integration occurs in the generator sources of the BER, no difference in amplitudes between binaural and combined monaural waveforms should occur. b) since dual inputs to a binaurally activated CAS system should result in the pool of affected neurons reaching threshold sooner than single monaural inputs (Pratt and Sohmer, 1976), a decreased latency for integrating peaks is expected for binaural stimulation.

Additional conditions studied:

a) pictorial comparison between binaural and combined monaural waveforms where the stimulus conditions are a change in time (Delta T) or a change in intensity (Delta I) of one ear relative to the other. This phase of the present experiment was purely exploratory. No previous studies of the BER have used these stimulus conditions, probably because of their confounding nature. That is, by delaying the click to one ear, relative to the other, two BER waveforms will be produced in

which all peaks will be delayed for one. If binaural integration does occur, it will be confounded by the complexity of the combined waveform. Similarly, by decreasing the amplitude to one ear relative to the other, a similar delay in all BER peaks generated by the decreased stimulation will result. Therefore, simply for exploratory purposes, pictorial comparison was used. To help eliminate the expected confounding (and since the interest is in possible binaural interaction and not the known monaural changes caused by these stimuli), for both the delays in latency and decreases in amplitude, waveforms were averaged across subjects and all Delta I or Delta T conditions, and the BI/MON differences plotted. For this part of the study, rarefaction clicks were the phase chosen since, in our lab, it has proven easier to delineate the wave peaks clearly with this type of stimulus.

b) statistical determination of significant interaural differences for peak five. This is also an exploratory part of the study. Since it is now common, in clinical settings, to use interaural differences in BER waveforms as indications of pathology, it is necessary to determine if the interaural latency differences within the normal range (i.e., less than 300 usec) are random (i.e., no significant difference in normal subjects), or, if not, which ear shows longer latencies. This latter possiblity also has implications for those interested in the laterality of CNS systems.

c) pictorial comparison of left and right waveform differences measured by a mastoid/mastoid derivation. Although the mastoid has been shown to be an electrically active site in the past, because of its continued use as a reference site in BER research, it seemed important to reiterate this point. In addition, since researchers have pointed to differences in mastoid/mastoid waves, as compared to conventional vertex/mastoid waves, a comparison was made between the latencies of the obtained waveforms to determine which (if any) of the peaks and troughs correspond, and the direction of difference.

d) comparison of the waveforms obtained by a conventional vertex/mastoid derivation with a vertex/non-cephalic derivation. Since a number of studies have shown both that there are different dipole orientations for different BER generator sites and that the mastoid is an active site, it was considered that a non-cephalic reference might delineate binaural interaction effects more clearly.

e) measurement and analysis of latencies and amplitudes for both BER peaks and troughs. Traditionally, troughs of the BER waveform have received little or no attention. However, it is important to determine if the troughs represent actual processing in the CAS or simply the restoration of peak activity to a resting level. If they, in fact, represent CAS processing, their study may be important in understanding the nature of the full system.

METHOD

Subjects

Ten male and ten female paid subjects (mean age of 22.45 years; range of 18-34 years) were recruited from the Simon Fraser University student body. All subjects were screened for hearing defects (Bekesy Audiometer, type E800, Grason-Stadler); six potential subjects were not used due to hearing losses in excess of 25 dB (1964 ISO values) for the pure tone frequency range of 100-10000 Hz. This cut-off was chosen after personal consultation with Dr. P. Gannon.

Apparatus

Beckman EEG disc, silver/silver chloride electrodes (16mm) were attached to the vertex, forehead, seventh cervical process and both mastoids. Redux electrode paste (Hewlett-Packard) was used for abrading the skin and for providing the electrode/skin interface. The impedance between pairs of recording electrodes was less than 5000 ohms in all cases. The recording derivations were as follows: vertex active, right mastoid reference; vertex active, seventh cervical process reference; left mastoid active, right mastoid reference; and, forehead ground. Amplification was accomplished by either a Tektronix

Type 122 or Type RM122 first stage amplifier (bandpass 8-10K Hz), and a Grass 7P5-A/DAC second stage amplifier (bandpass 10-40 KHz). EEG output was recorded on a Tandberg 115 FM taperecorder (bandpass 0-5 KHz) for later averaging offline. Final offline signal conditioning (Krohn-Hite Model 3323 dual filter) set the bandpass to 80-3000 Hz. A Fabritek 1072 signal averager was used with an A/D sampling rate of 20 usec per point for 512 points; each average was comprised of 2048 stimulus presentations. The averager was set to nine bits of resolution, thus for an approximate amplification of 100,000x, comparison of plotted signals can be accurate to no closer than 0.039 of a uvolt. Averaged signals were transferred to disc storage (Data General Nova 3, mini computer) for further processing and final plotting (Houston Omnigraphic, model E-6650). Initial calibration of the system was accomplished using a Bioelectric Instruments, CA5 calibrator set to deliver a two msec 2 uvolt squarewave. Daily calibration was thereafter accomplished using a 50 uvolt signal (Grass Model SWC-1B Square Wave Calibrator).

Click duration was set at 0.2 msec, generated by either a Wavetek model 184 or 185 signal generator triggered by the function generator described below. This duration was chosen so that comparisons might be made between this study and those of Endo (1976), Hallman (1977), and Ornitz and Walter (1975, 1976). Inter-stimulus-interval (ISI), set to 50 msec, was generated by a Hewlett Packard Variable Phase Function Generator (model 203A). This ISI was chosen for two reasons:

a) the greatest rarefaction/condensation (R/C) effect occurred in Hallman's study at this ISI; and, b) recording times per stimulus condition are of reasonable length for subjects. Relative delays in the stimulus latency (Delta T) were obtained by changing the phase of the variable output of the function generator. Both stimulus intensity and delay were monitored on a Philips oscilloscope (model PM 3232). Online monitor averaging was carried out on a Fabritek 1052/LS signal averager. Clicks were transduced by TDH-39 earphone speakers mounted in MX41/AR rubber cushions. The modification of the click by the mechanical properties of the transducer is shown in Figure 7. Clicks of equivalent duration have been shown to have peak energy in the 3000-4000 Hz frequency range (Hallman, 1977: Ornitz and Walter, 1975, 1976). Rarefaction and condensation waves were defined as the direction of first movement of the transducer diaphragm and were produced by reversing the polarity of the square wave from the signal generator. Click intensity was set at 65 dB HL as defined by the formula: dB = 20 log [voltage of signal/voltage of threshold] (Picton et al., 1973). This dB level was chosen since it approaches the intensity levels at which peak amplitude plateaus are reached (i.e., the CAS is being driven near its BER maximum) and the intensity level is neither harmful nor discomforting to the subject. Approximate equivalent dB level, at an ISI of 2 msec, was 96 dB SPL, as measured by a General Radio Co. sound level meter (type 1551-C) fitted with a one-half inch condenser microphone (type

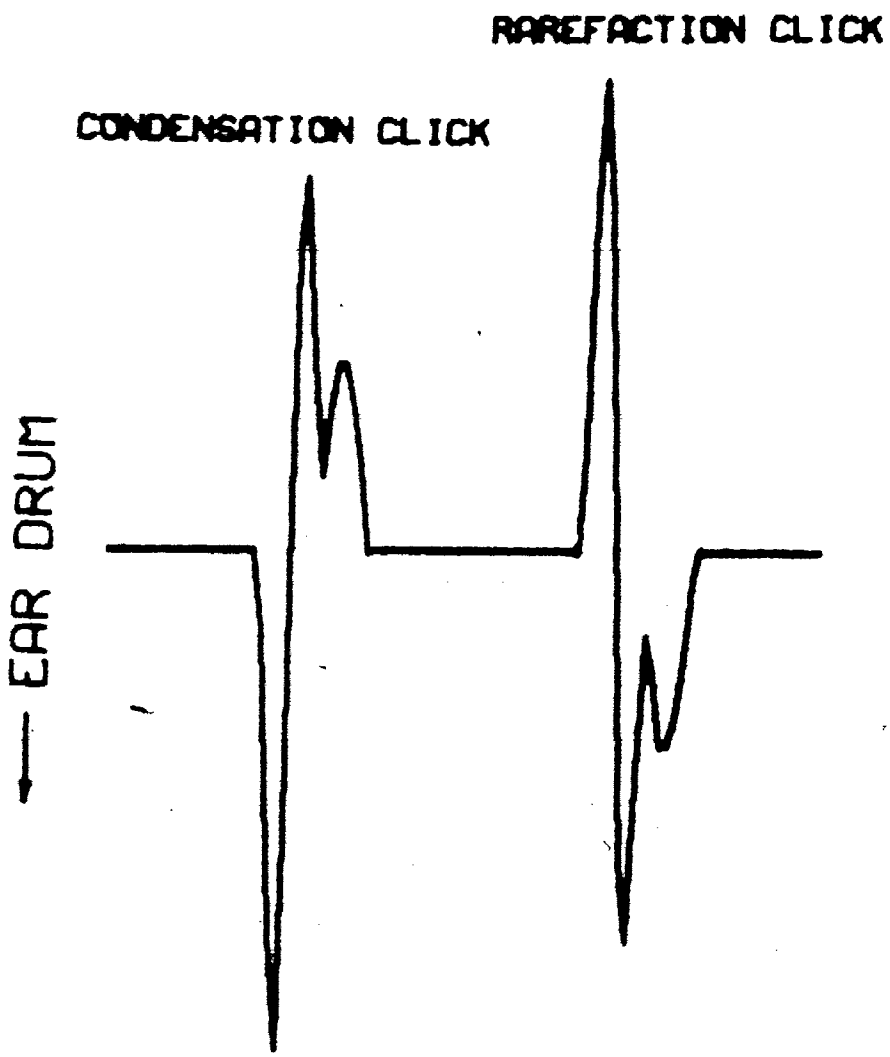


FIGURE 7
TRANDUCER MODIFICATION
OF SQUARE WAVE

1560-P5) and a General Radio Co. earphone coupler (type 9A). The sound level meter was set at "slow" with a "B" weighting.

The recording room was neither electrically insulated nor sound attenuated; in order to eliminate possible external electrical interference, first stage amplifiers were placed in the room within reach of the electrode lead wires. No observable increase in background noise resulted from this placement.

PROCEDURE

Preceeding each recording session the amplification system was adjusted for d.c. offset and calibrated. Subjects were placed in a semi-supine position on an "easy-boy" type of recliner rocker. All were told to rest, relax and, if possible, go to sleep. Lights were turned off, clicks were turned on, and, after monitoring showed substantial muscle relaxation (i.e., a leveling of muscle artifact in the EEG) and/or five minutes had elapsed, recording commenced. Two potential subjects were eliminated from the study due to excessive muscle artifact contamination. The F.M. taperecorder CRT allowed continuous monitoring of background muscle activity; if the subject showed a build up of muscle tension, recording was interrupted and the subject was given a "stretch" break. Average recording times lasted approximately 1 1/4 to 1 1/2 hours.

All subjects received the following ten randomly presented stimulus conditions:

1) rarefaction - left; rarefaction - right;
2) rarefaction - left; condensation - right;
3) condensation - left; rarefaction - right;
4) condensation - left; condensation - right;
5) nothing - left; rarefaction - right;
6) nothing - left; condensation - right;
7) rarefaction - left; nothing - right;
8) condensation - left; nothing - right;
9) rarefaction - left using right transducer; nothing right; and,
10) condensation - left using right transducer; nothing right.

Subjects were divided into two groups (each comprised of five females and five males) for the second half of the experiment. Group A received the following 16 randomized stimulus conditions using only rarefaction clicks:

1) 45 dB - left; 65 dB - right;
2) 45 dB - left; 0 dB - right;
3) 50 dB - left; 65 dB - right;
4) 50 dB - left; 0 dB - right;
5) 55 dB - left; 65 dB - right;
6) 55 dB - left; 0 dB - right;
7) 60 dB - left; 65 db - right;

8) 60 dB - left; 0 dB - right;

9) 65 dB - left; 60 dB - right;

10) 0 dB - left; 60 dB - right;

11) 65 dB - left; 55 dB - right;

12) 0 dB - left; 55 dB - right;

13) 65 dB - left; 50 dB - right;

14) 0 dB - left; 50 dB - right;

15) 65 dB - left; 45 dB - right; and,

16) 0 dB - left; 45 dB - right.

Group B received the following 16 randomized stimulus conditions using only 65 dB rarefaction clicks. All clicks, whether right or left, were referred to the pulse generated by the Wavetek driving the right transducer:

1) right click; left delayed 0.2 msec;

2) no right click; left delayed 0.2 msec;

3) right click; left delayed 0.4 msec;

4) no right click; left delayed 0.4 msec;

5) right click; left delayed 0.6 msec;

6) no right click; left delayed 0.6 msec:

7) right click; left delayed 0.8 msec;

8) no right click; left delayed 0.8 msec;

9) right click; left advanced 0.2 msec;

10) no right click; left advanced 0.2 msec;

11) right click; left advanced 0.4 msec;

12) no right click; left advanced 0.4 msec;

13) right click; left advanced 0.6 msec;

14) no right click; left advanced 0.6 msec;

15) right click; left advanced 0.8 msec; and,

16) no right click; left advanced 0.8 msec.

RESULTS

For the following:

T = negative waves (i.e., troughs of waveforms);

P = positive waves (i.e.,peaks of waveforms);

VM = vertex/mastoid;

VN = vertex/non-cephalic;

RR = rarefaction right, rarefaction left;

RC = rarefaction right, condensation left;

CR = condensation right, rarefaction left; and,

CC = condensation right, condensation left.

Latency and amplitude measures were taken blind from the plotted waveforms; that is, stimulus conditions were not known at the time of measurement (other than the subjects from which the waveforms were obtained), and both latencies and amplitudes were measured in centimeters with conversion to msec and uvolts occurring after computer analysis. This was carefully done to eliminate the possible experimenter bias if either the stimulus condition, or the wave peak latencies were known.

Peak/trough determination in BER research is extremely problematic. This is especially true when one attempts to

compare differences in latency in the 10's of microseconds. No explanation of exact deterination procedures has been reported in the literature to date. Since the BER waveform is a complex potential, and since each potential is somewhat unique to each subject, a detailed determination procedure is necessary to enable replication. A system for peak/trough determination was developed for this study (see Appendix A) and was used on all waveforms. Two questions arose concerning this procedure: a) would other researchers, using this procedure, agree in their gross identification of peaks and troughs; and, b) how much variability in rater's determination of microvolt and millisecond values can be expected? The second question is most important since it will set the lower limits for differences that can be regarded as significant results of experimental condition's regardless of statistical significance of the data. In an effort to answer these questions, six individuals (raters), with little or no experience with BER's determined the peaks and troughs of the waveforms for two randomly selected subjects (i.e., a total of 32 waveforms). From these data, for each peak and trough, the an estimation of the variance of the raters was obtained (see Table One, a), where X is a raters determination for a single peak or trough, $\bar{X}$ is the mean of all raters for that particular peak or trough, and R is the number of raters; and, an estimate of the variance of the author, (see Table One, b), where Y is the author's previous determination. An F ratio was formed from these two

TABLE ONE
TABLE OF FORMULAE FOR
RATER STATISTICS

a) $$\hat{\sigma}_s^2 = \frac{\Sigma\Sigma(X-\bar{X})^2}{32\times(R-1)}$$

b) $$\hat{\sigma}_A^2 = \frac{\Sigma(Y-\bar{X})^2}{32\times(1+1/R)}$$

c) $$F = \frac{\hat{\sigma}_s^2}{\hat{\sigma}_A^2}$$

d) $$ESE = \sqrt{\frac{\hat{\sigma}_s^2 + \hat{\sigma}_A^2\,(R-1)}{R}}$$

variances (see Table One, c) with a df of 32 and 160. Significance would indicate that the raters and the author were selecting different component to be measured. The following F's were obtained:

a) Latencies

P1: F = 0.134/0.237 = 0.565

T1: F = 0.050/0.056 = 0.771

P2: F = 0.024/0.030 = 0.802

T2: F = 0.026/0.098 = 0.265

P3: F = 0.028/0.023 = 1.211

T3: F = 0.009/0.026 = 0.356

P4: F = 0.026/0.047 = 0.555

P5: F = 0.025/0.102 = 0.245

T5: F = 0.018/0.066 = 0.275

P6: F = 0.039/0.117 = 0.335

T6: F = 0.027/0.104 = 0.259

b) Amplitudes

P1: F = 0.094/0.153 = 0.616

T2: F = 0.026/0.047 = 0.55

P2: F = 0.021/0.030 = 0.696

T2: F = 0.044/0.040 = 1.09

P3: F = 0.029/0.035 = 0.829

T3: F = 0.020/0.041 = 0.485

P4: F = 0.040/0.144 = 0.278

P5: F = 0.077/0.266 = 0.289

T5: F = 0.029/0.070 = 0.414

P6: F = 0.054/0.122 = 0.443

T6: F = 0.029/0.134 = 0.261

Since no F reaches significance, this indicates that both the author and the raters were identifying and measuring the same BER components. In addition, since the majority of F's are less than 1, the author's determinations are closer to the raters' means than the raters are to their own mean. In other words, the author shows less variation in peak/trough determination as might be expected from the difference in experience with BER waveforms.

Two estimated standard errors (ESE) were obtained. The first ESE was for the author (the square root of the variance (multiplied by a conversion factor to msec (0.128) and uvolts (0.04)) which comprised the lower limit of difference accepted as significant. A second was the pooled ESE for the raters plus the author. This pooled ESE (see Table One, d) is the best estimation of peak/trough determination deviation of any single individual chosen at random to serve as a rater.

Authors' ESE:

	Latencies	Amplitudes
P1:	+/- 0.047 msec	+/- 0.01 uvolts
T1:	+/- 0.029 msec	+/- 0.01 uvolts
P2:	+/- 0.020 msec	+/- 0.01 uvolts
T2:	+/- 0.021 msec	+/- 0.01 uvolts
P3:	+/- 0.021 msec	+/- 0.01 uvolts
T3:	+/- 0.012 msec	+/- 0.01 uvolts
P4:	+/- 0.021 msec	+/- 0.01 uvolts
P5:	+/- 0.020 msec	+/- 0.01 uvolts

T5:	+/- 0.017 msec	+/- 0.01 uvolts
P6:	+/- 0.025 msec	+/- 0.01 uvolts
T6:	+/- 0.021 msec	+/- 0.01 uvolts

Pooled ESE:

	Latency	Amplitude
P1:	+/- 0.060 msec	+/- 0.02 uvolts
T1:	+/- 0.032 msec	+/- 0.01 uvolts
P2:	+/- 0.022 msec	+/- 0.01 uvolts
T2:	+/- 0.027 msec	+/- 0.01 uvolts
P3:	+/- 0.020 msec	+/- 0.01 uvolts
T3:	+/- 0.019 msec	+/- 0.01 uvolts
P4:	+/- 0.027 msec	+/- 0.01 uvolts
P5:	+/- 0.038 msec	+/- 0.02 uvolts
T5:	+/- 0.029 msec	+/- 0.01 uvolts
P6:	+/- 0.041 msec	+/- 0.01 uvolts
T6:	+/- 0.039 msec	+/- 0.01 uvolts

Even with the procedure outlined in Appendix A, P4, P5, P6 and T6 were not clearly observable in some waveforms. When this occurred, the subject was eliminated for all analyses of the affected peak or trough. Although the waves were not "missing" in all stimulus conditions, the underlying assumption of this study was that they were in fact present in all waveforms, but camouflaged by neural activity of unknown origin or cause in some cases. Thus it was felt to be more reasonable to eliminate all data for the affected peak or trough than to attempt a missing data analysis.

Two latency measurements were obtained; the first with

time zero being the sweep initiation of the signal averager (raw latency), and the second with T1 as time zero (derived latency). Thus, according to the classical BER literature (e.g., Huang and Buchwald, 1978; and, Salamy et al., 1975), both peripheral and central latency components were studied separately. T1 was chosen over P1 since P1 has proven difficult to determine exactly.

Two amplitude measurements were obtained; one using T1 as arbitrary zero voltage (zero baseline amplitude), and a second using the absolute value of peak-to-trough amplitude differences (peak-to-trough amplitude). Thus, influences that affect the overall waveform as well as those affecting separate peak/trough amplitudes were studied.

Since little or no difference in latency or amplitude as a result of binaural interaction has been reported in the literature, if significant changes do occur, they are likely to be extremely small. This results in little observable difference when raw waveforms for different conditions are plotted (see Figure 8). In an effort to better represent waveform changes in plotted form, two techniques were used: a) whenever there was only a single comparison between two means (binaural vs monaural and vertex/mastoid vs vertex/non-cephalic) a difference between the means was plotted; (monaural minus binaural, and vertex/non-cephalic minus vertex/mastoid) and, b) for phase, the difference of each individual phase mean from the grand mean of all four phase

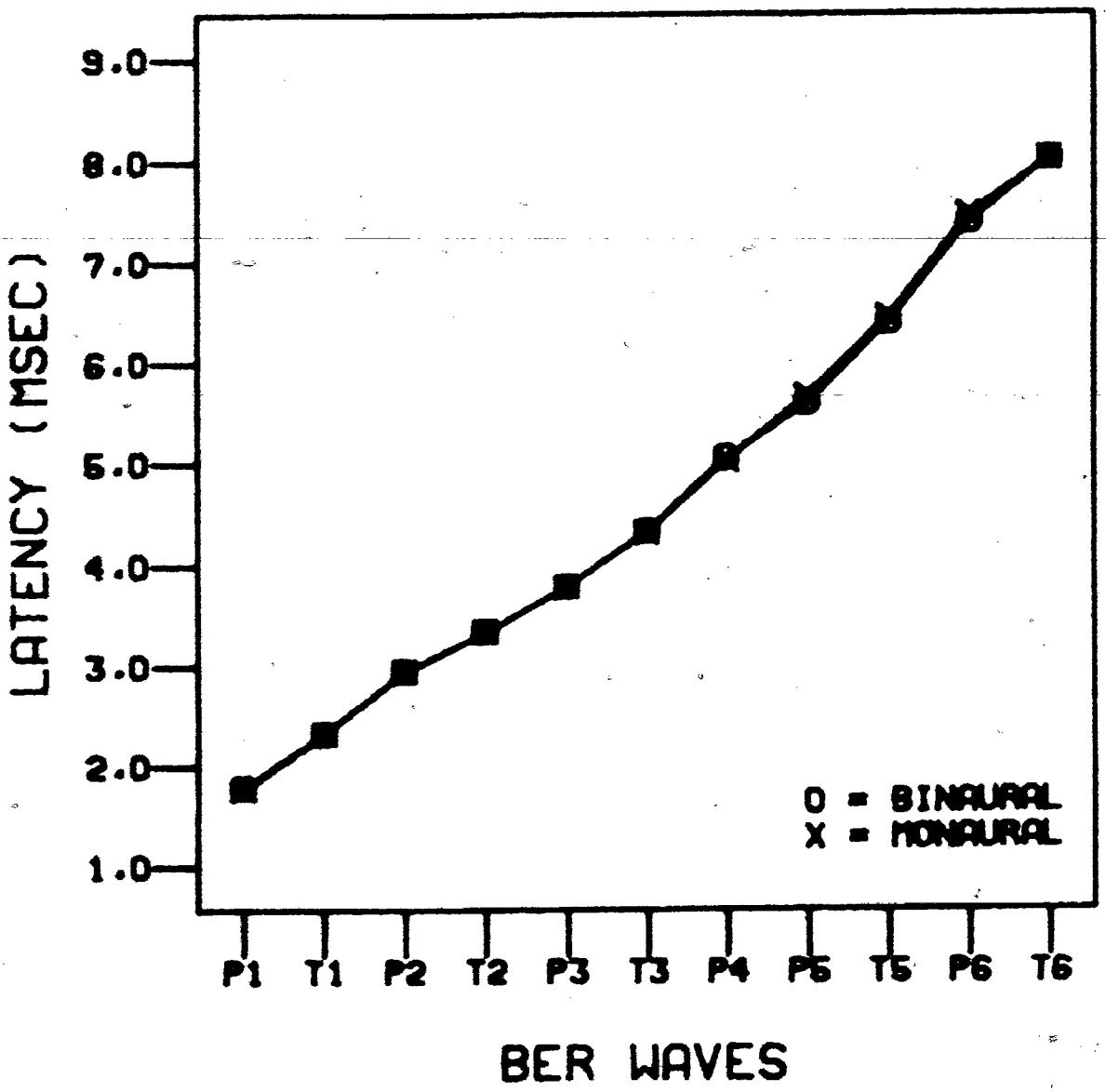


FIGURE 8 -- EXAMPLE PLOT
RAW LATENCIES

conditions (at each individual peak and trough) was plotted. In all cases, plotting is simply a representation of the ANOVA findings and does not represent an analysis of this linear transformation.

Initial analysis of variance indicated little or no sex differences, thus all ANOVAs were carried out ignoring sex. A three factor (2 x 2 x 4) within subjects analysis of variance design was used to evaluate amplitude and latency measures (electrode derivation x binaural/monaural stimulation x stimulus phase). Each peak and trough was analysed separately resulting in 40 analyses. There is no theoretical reason to consider the two latency analyses and the two amplitude analyses to be orthogonal, thus alpha levels were adjusted to a probability level of 0.013 for determining significance (although considered too restrictive for the present study, an alpha level adjusted for the 40 analyses would have to be set to 0.001). A test of the symmetry of the variance/covariance matrix was carried out for all analyses. All significant results that failed to show symmetry at a probability level of 0.05 were reassessed using the conservative test procedure outlined by Myers (1972, p. 177). Any significant difference failing to reach the 0.025 level of probability with this test was not considered.

Analysis of variance computations were carried out using BMDP2V (Analysis of Variance and Covariance including repeated

measures) of the Health Sciences Computing Facility, UCLA, 1977. One further analysis of variance (sex by ear) was carried out for P5. The left P5 and right P5 latencies, for this analysis, were obtained by averaging the monaural raw latency measures for VM-C and VM-R presented to each ear. The same transducer was used for both ears to eliminate possible mechanical differences. All computer calculated means were verified by hand calculation to ensure the data had been properly read.

ANOVA Tables for all peaks and troughs for the two latency measures and the two amplitude measures are presented in Appendices B through E. Frequency histograms of each peak and trough for raw latency and zero baseline data are presented in Appendix H. Figure 9 illustrates the composite waveform across all subjects and conditions, and Table Two shows the raw latency and zero baseline amplitude means for all waves and troughs. All means are within the bounds reported by other authors, other than P2 (published range 2.4-2.8 msec). It is of interest that with the particular recording parameters used, P5 shows a smaller "waveform" positivity (as measured from an arbitrary waveform baseline) than that of P4 (a not uncommon finding in the literature; e.g., Schulman-Galambos and Galambos, 1975; and, Starr and Achor, 1975). In addition, throughout the blind peak determination, it seemed apparent that P6 and T6 were occurring in only approximately half the

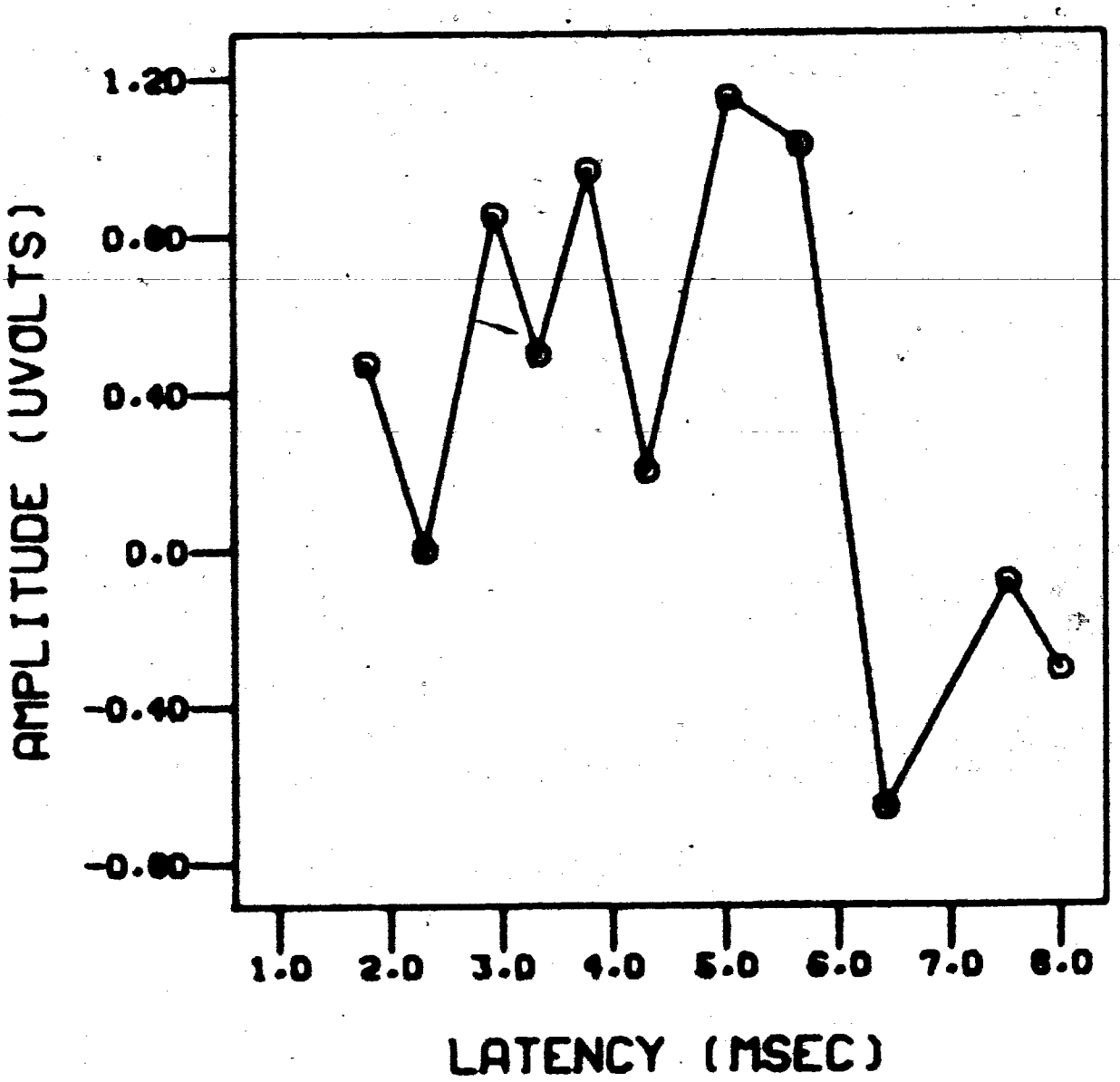


FIGURE 9
COMPOSITE BER WAVEFORM
ZERO BASELINE AMPLITUDES
X RAW LATENCIES

TABLE TWO -- LATENCIES AND AMPLITUDES FOR COMPOSITE BER

Wave	Raw Latency	Baseline Amplitude
P1	1.80	0.47
T1	2.33	0
P2	2.94	0.86
T2	3.33	0.50
P3	3.78	0.97
T3	4.31	0.20
P4	5.04	1.15
P5	5.67	1.03
T5	6.42	-0.66
P6	7.53	-0.09
T6	7.99	-.031

Note: Latencies are in msec and amplitudes in uvolts.

subjects, thus these peaks and troughs were not measured. However, once arbitrary latency units had been converted into msec, it became apparent that the peak thought to be P7 was, in actuality, P6 and the eliminated peak probably the same peak discussed by Don et al. (1977). Further study of the properites of this wave is indicated.

A. Interaural Peak Five Latency Differences

Neither a sex effect nor a lateralization effect was found (see Appendix I). The mean difference between the right and left ears was 0.042 msec, well within the bounds of clinical normalacy.

B. Electrode Derivation Effects

1) Latency Measures

Figures 10 and 11 illustrate the mean difference between Vertex/Mastoid and Vertex/Non-cephalic derivations. In all cases where a significant difference occurs, VM results in shorter latencies. For raw latencies the following p values

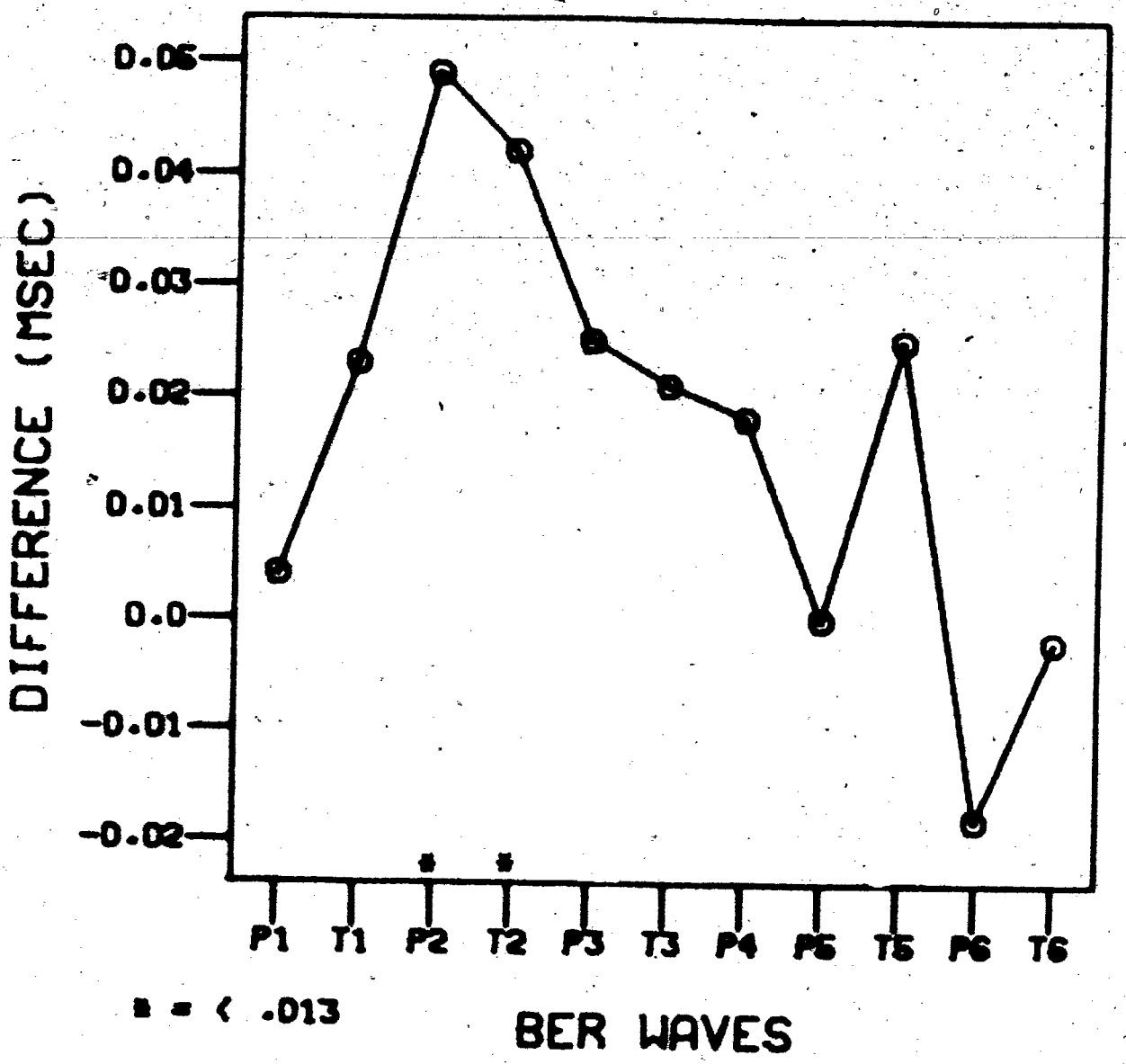


FIGURE 10
RAW LATENCY - VN MINUS VM

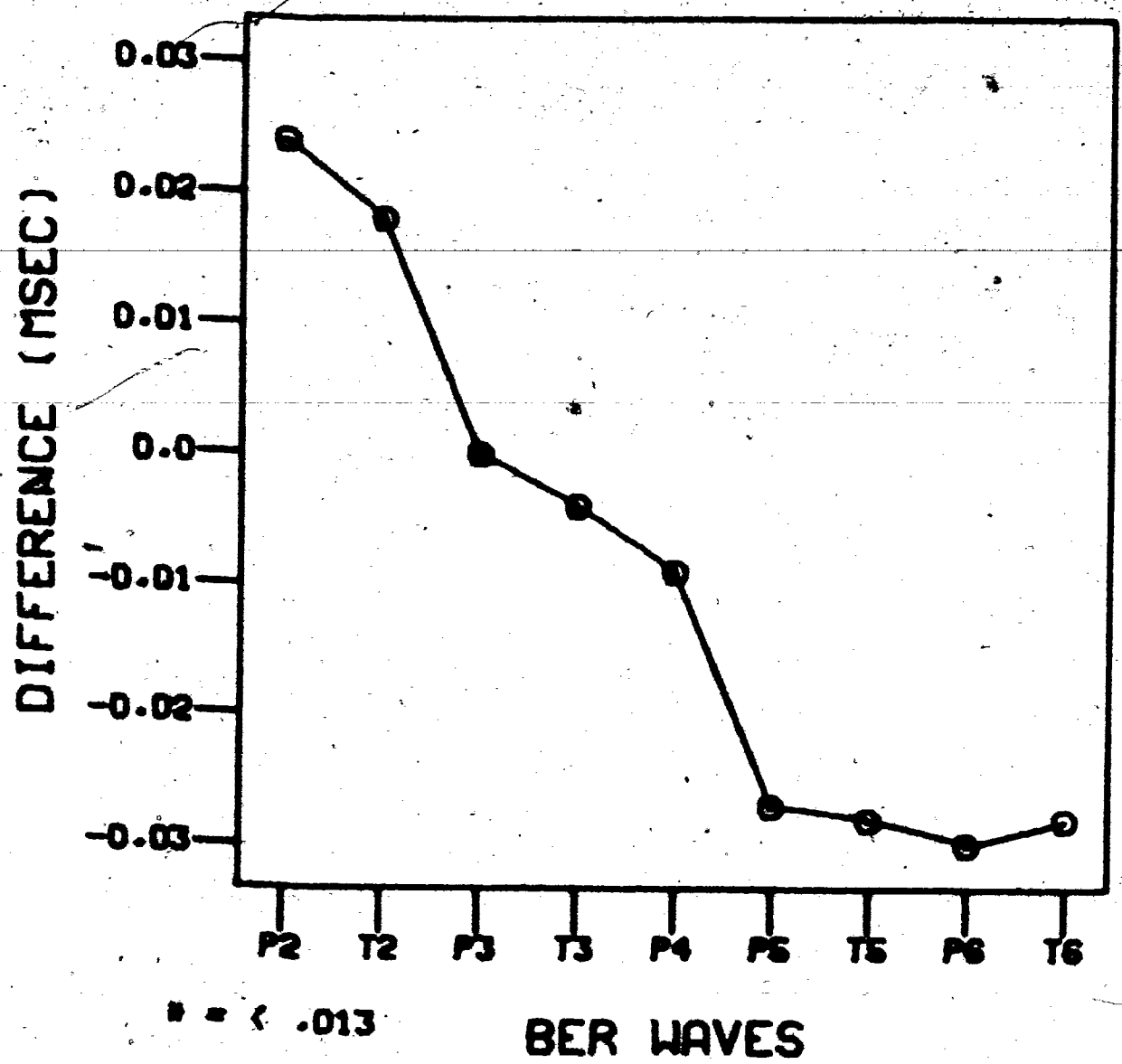


FIGURE 11
DERIVED LATENCY - VN MINUS VM

were obtained (P3 and T5 do not meet the minimum msec difference requirements):

P1: $p > .100$

T1: $p = .054$

P2: $p < .001$

T2: $p < .001$

P3: $p < .001$

T3: $p = .046$

P4: $p = .041$

P5: $p > .100$

T5: $p = .003$

P6: $p > .100$

T6: $p > .100$

For latencies measured from T1 (derived latencies), no significant differences were shown:

P2: $p = .074$

T2: $p = .062$

P3: $p > .100$

T3: $p > .100$

P4: $p > .100$

P5: $p = .015$

T5: $p > .100$

P6: $p = .036$

T6: $p > .100$

A derivation by phase interaction for raw latency at P4 ($p = 0.010$) proved significant (Figure 12). As shown in the Newman-Keuls Studentized Range Statistic (Appendix J), and in terms of the minimum cutoff for mean latency differences, shorter latencies were found for the VM condition when a rarefaction stimulus was presented to the left ear.

2) Amplitude Measures

Figures 13 and 14 illustrate the VM/VN differences for the baseline amplitude and the peak-to-trough amplitude data. In all cases where significance occurres, VN represents a larger waveform than VM; thus, for P2 to P5 VN is more positive, and for T5 to T6 VN is more negative. For zero baseline amplitude data the following p values were obtained:

P1: $p > .100$

P2: $p < .001$

T2: $p < .001$

P3: $p < .001$

T3: $p = .007$

P4: $p < .001$

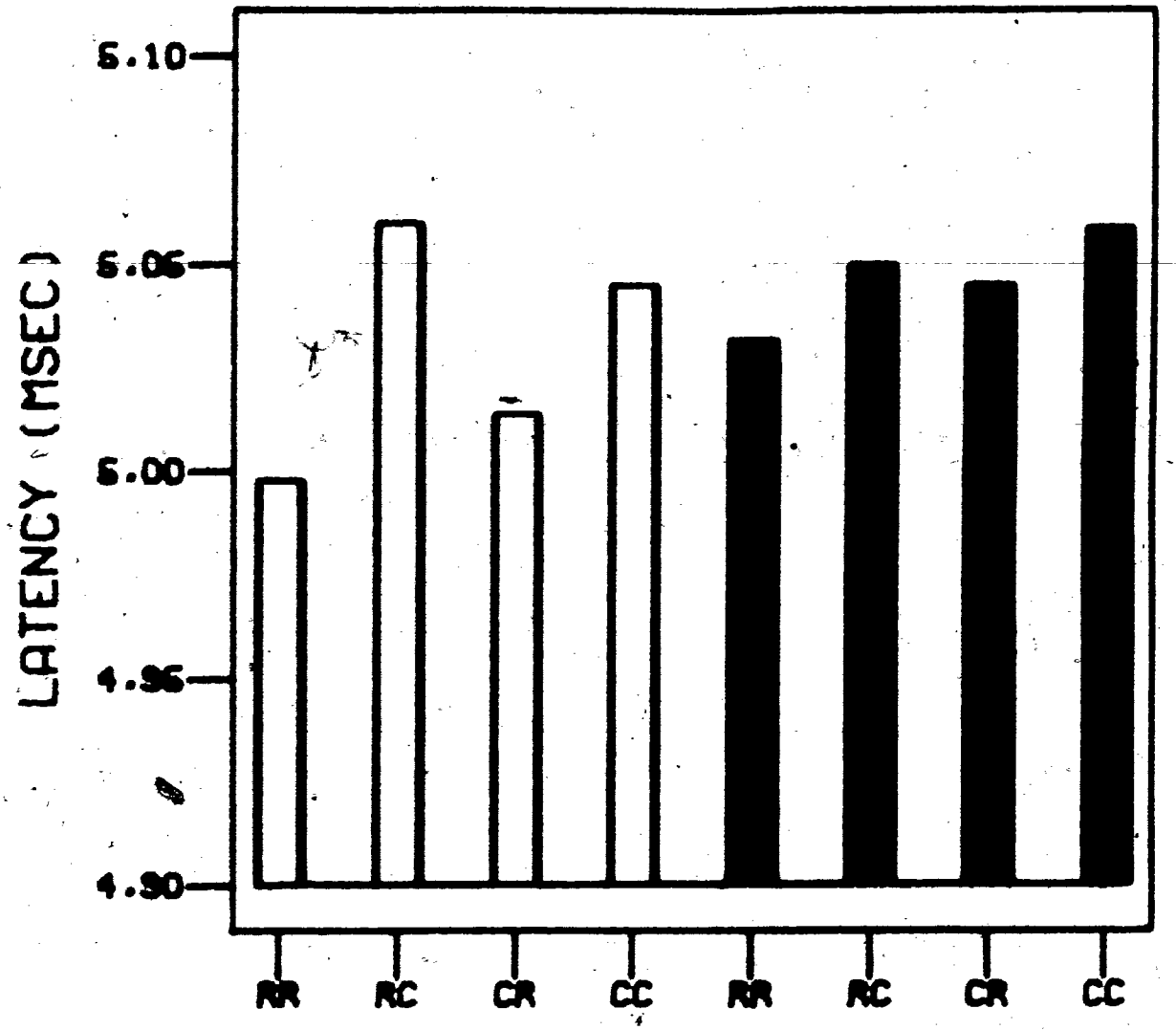
P5: $p < .001$

T5: $p < .001$

P6: $p = .004$

T6: $p < .001$

FIGURE 12
RAW LATENCY - P4



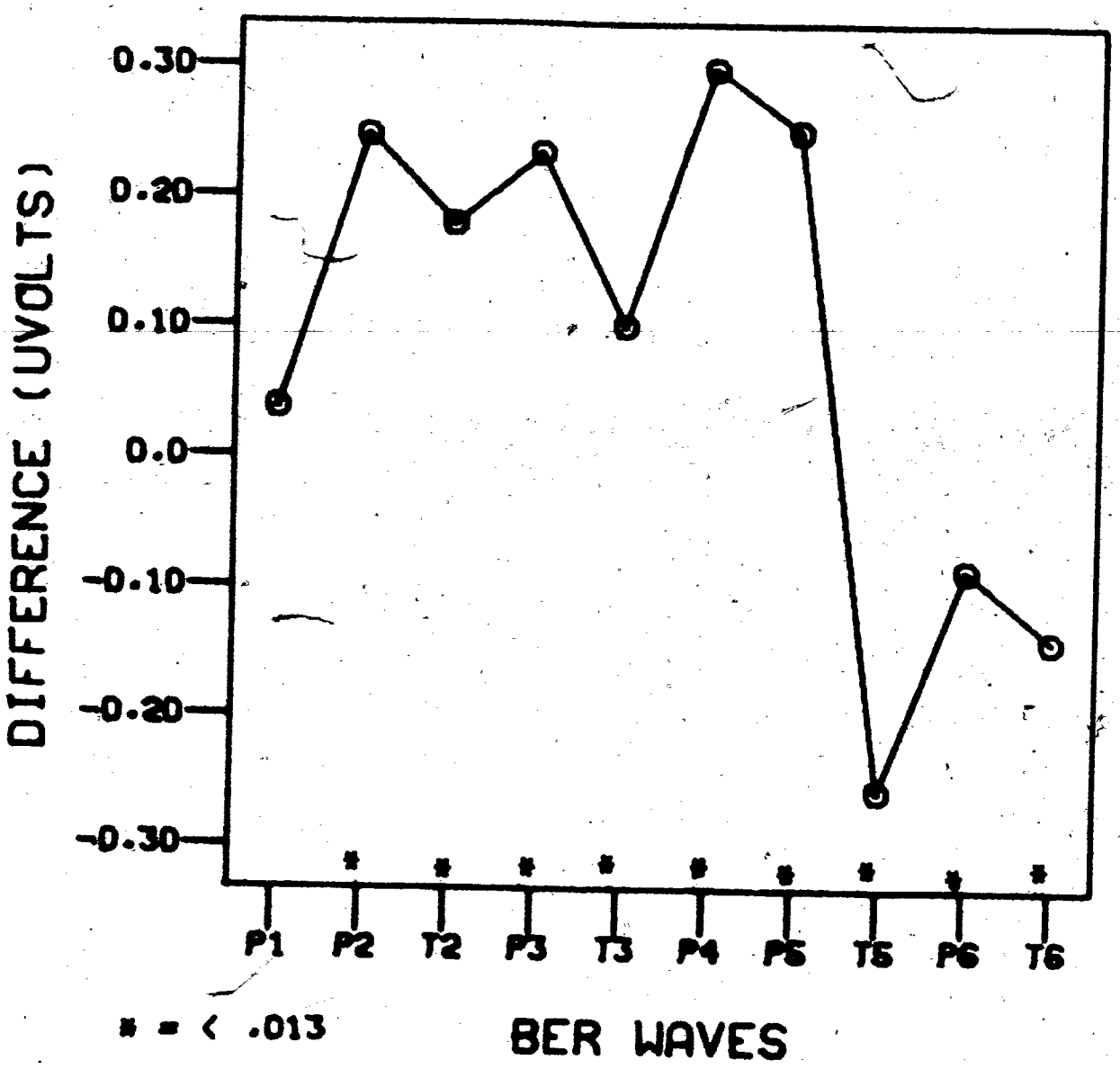


FIGURE 13
ZERO BASELINE AMPLITUDES
VN MINUS VM

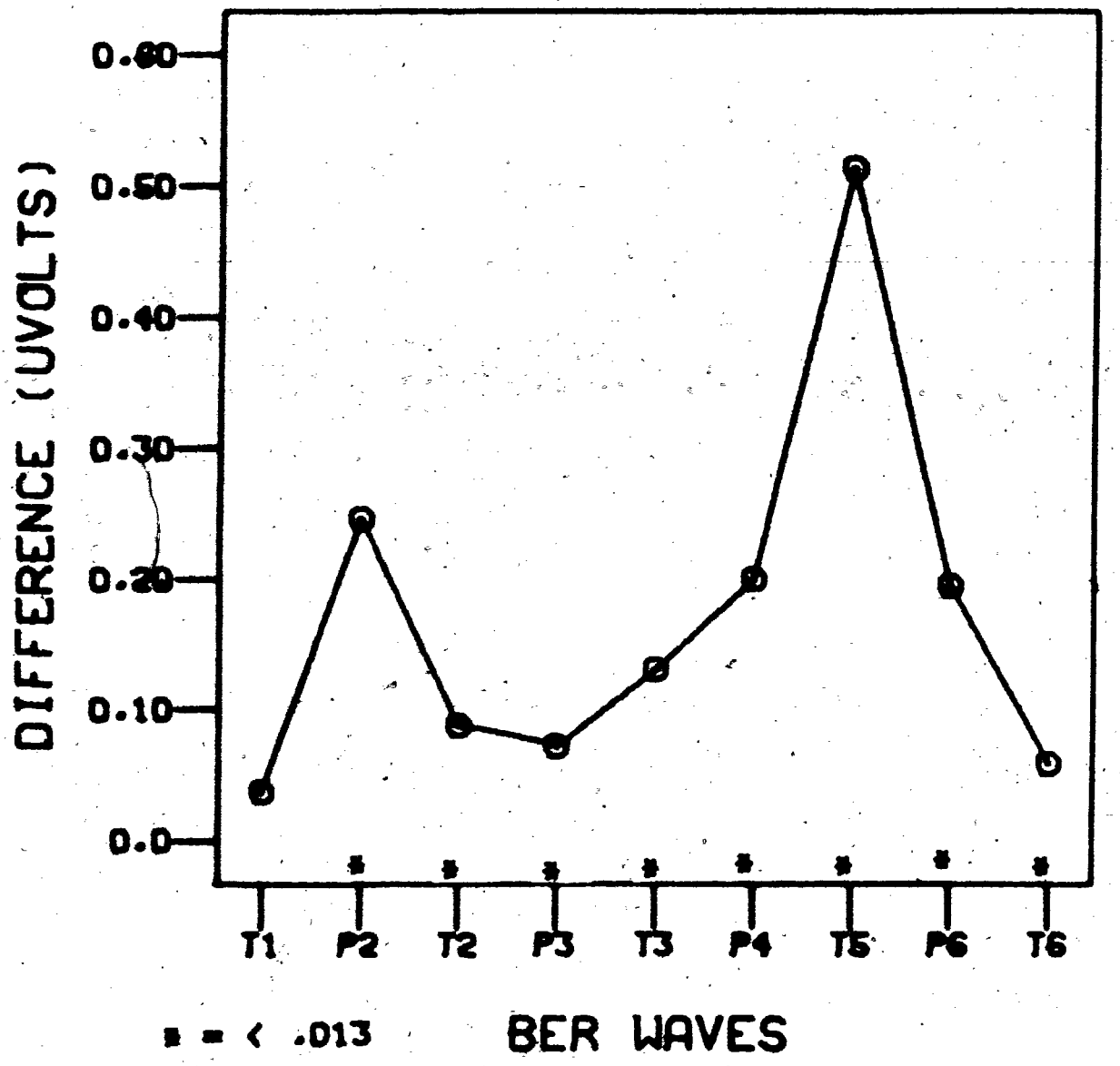


FIGURE 14
PEAK-TO-TROUGH AMPLITUDES
VN MINUS VM

For peak-to-trough amplitude data the following p values were obtained:

T1: $p > .100$

P2: $p < .001$

T2: $p < .001$

P3: $p = .001$

T3: $p < .001$

P4: $p < .001$

T5: $p < .001$

P6: $p < .001$

T6: $p = .012$

P5 was not calculated for peak-to-trough amplitude data since data for T4 were missing. In comparing zero baseline amplitude data with peak-to-trough amplitude data it must be kept in mind that whereas zero baseline amplitude data represent the amplitude of peaks and troughs from an arbitrary baseline, peak-to-trough amplitude data represent the absolute value of peak-to-trough differences. Thus peak-to-trough amplitude for any one peak or trough represents the combined effect of that peak or trough and the preceeding trough or peak.

P1, T5 and T6 showed significant derivation x phase interactions for zero baseline amplitude data, although they did not meet the minimum cutoff for non-symmetry.

Three derivation x phase interactions proved significant for peak-to-trough amplitude data:

T1: $p = .004$

P4: $p = .004$

p6: $p = .004$

As illustrated in Figures 15 through 17. For T1, RR/RC differences for both VM and VN show the same pattern -- RR results in greater amplitudes than does RC. The CC/CR differences, however, are not similar between VM and VN. Although the direction of difference is the same, the VN condition results in a much larger difference (CR resulting in greater amplitudes than CC). In addition, the VN CC/CR amplitudes are greater than the VM CC/CR amplitudes. For P4, the pattern is reversed with RC and CC showing greater amplitudes than RR and CR respectively. This is in accordance with the finding for Phase discussed below.

One derivation x BI/MON interaction was also significant for peak-to-trough amplitude data (Figure 18), T6: $p < .001$. VN is more positive than VM for binaural amplitudes but shows little or no difference for combined monaural data.

FIGURE 15
PEAK-TO-TROUGH AMPLITUDES - T1

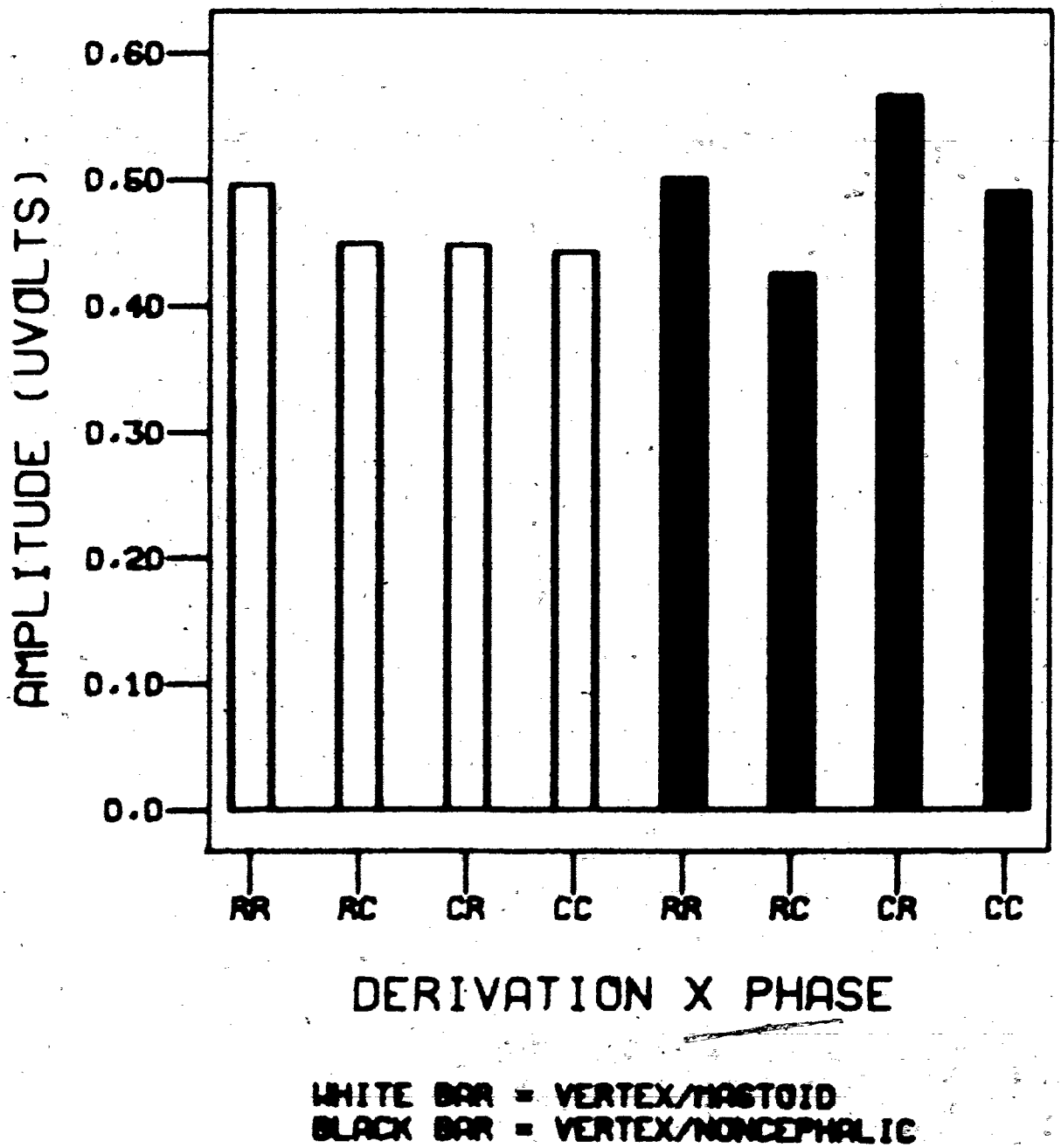


FIGURE 16
PEAK-TO-TROUGH AMPLITUDES - P4

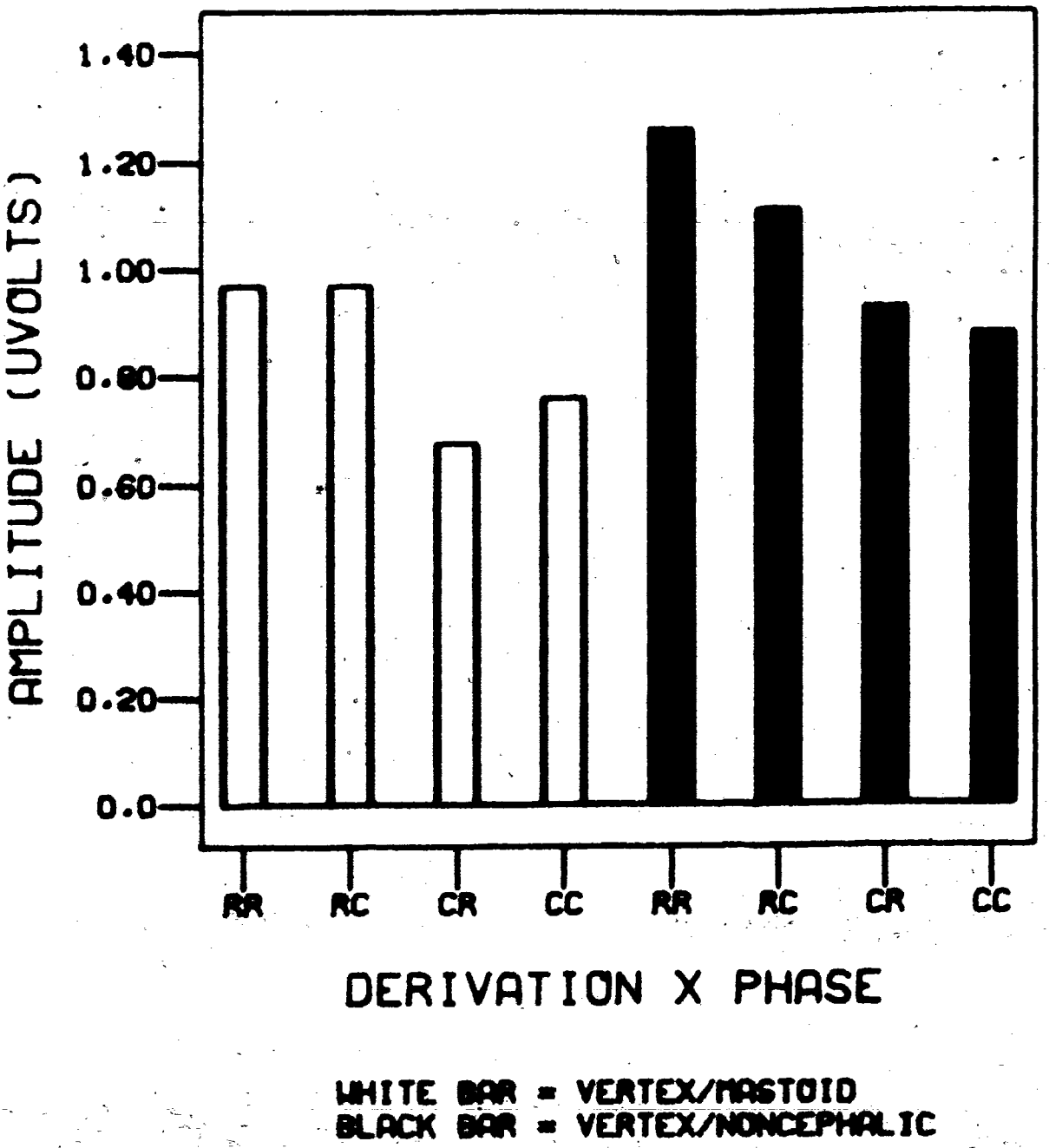
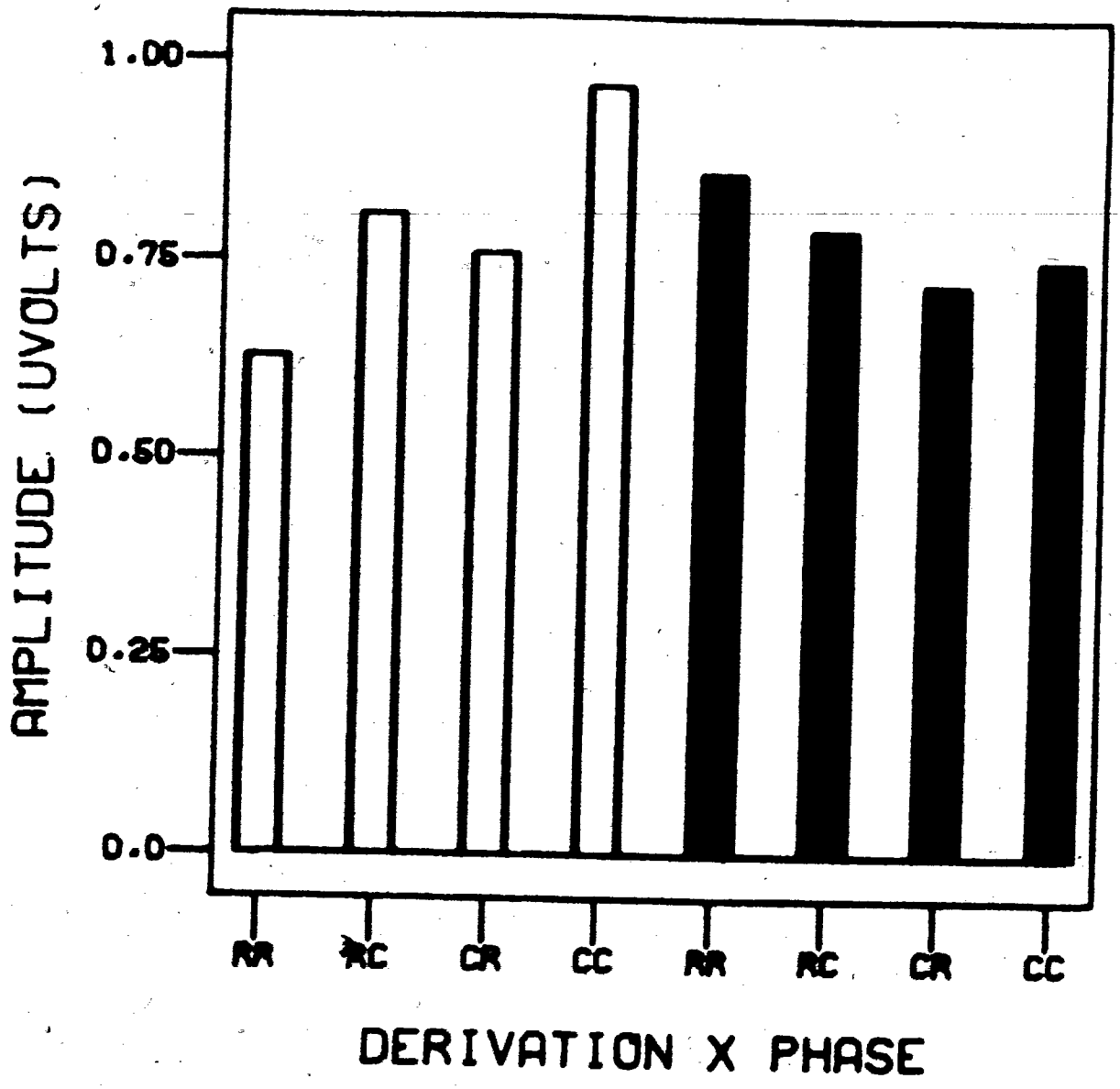
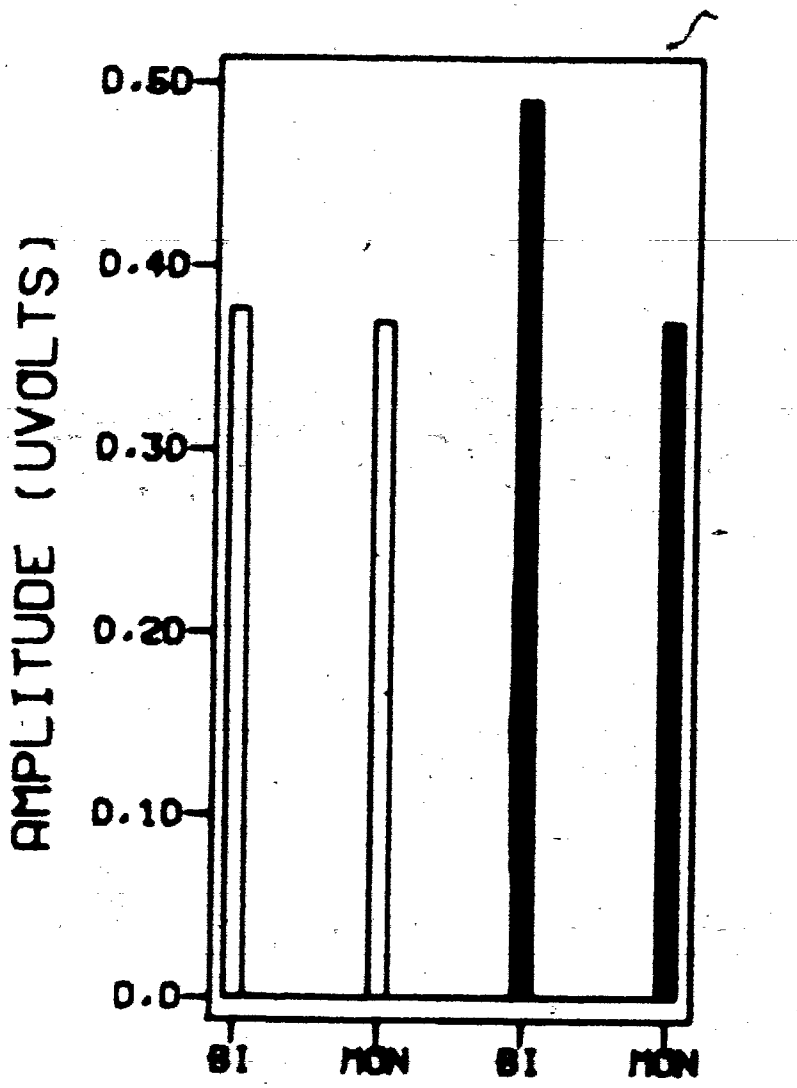


FIGURE 17
PEAK-TO-TROUGH AMPLITUDES - P6



WHITE BAR = VERTEX/MASTOID
BLACK BAR = VERTEX/NONCEPHALIC

FIGURE 18
PEAK-TO-TROUGH AMPLITUDES - T6



WHITE BAR = VERTEX/MASTOID
BLACK BAR = VERTEX/NONCEPHALIC

C. Binaural vs Combined Monaural Effects

1) Latency Measures

Figures 19 and 20 illustrate the BI/MON differences for latencies. Raw latencies for BI are significantly shorter at P5 and T5. A similar pattern is found for derived latencies. The following are the p values obtained for raw latencies:

P1: $p = .015$

T1: $p > .100$

P2: $p > .100$

T2: $p > .100$

P3: $p > .100$

T3: $p > .100$

P4: $p > .100$

P5: $p = .003$

T5: $p = .001$

P6: $p = .086$

T6: $p > .100$

The following are the obtained p values for derived latencies:

P2: $p > .100$

T2: $p = .050$

P3: $p > .100$

T3: $p > .100$

P4: $p = .077$

P5: $p < .001$

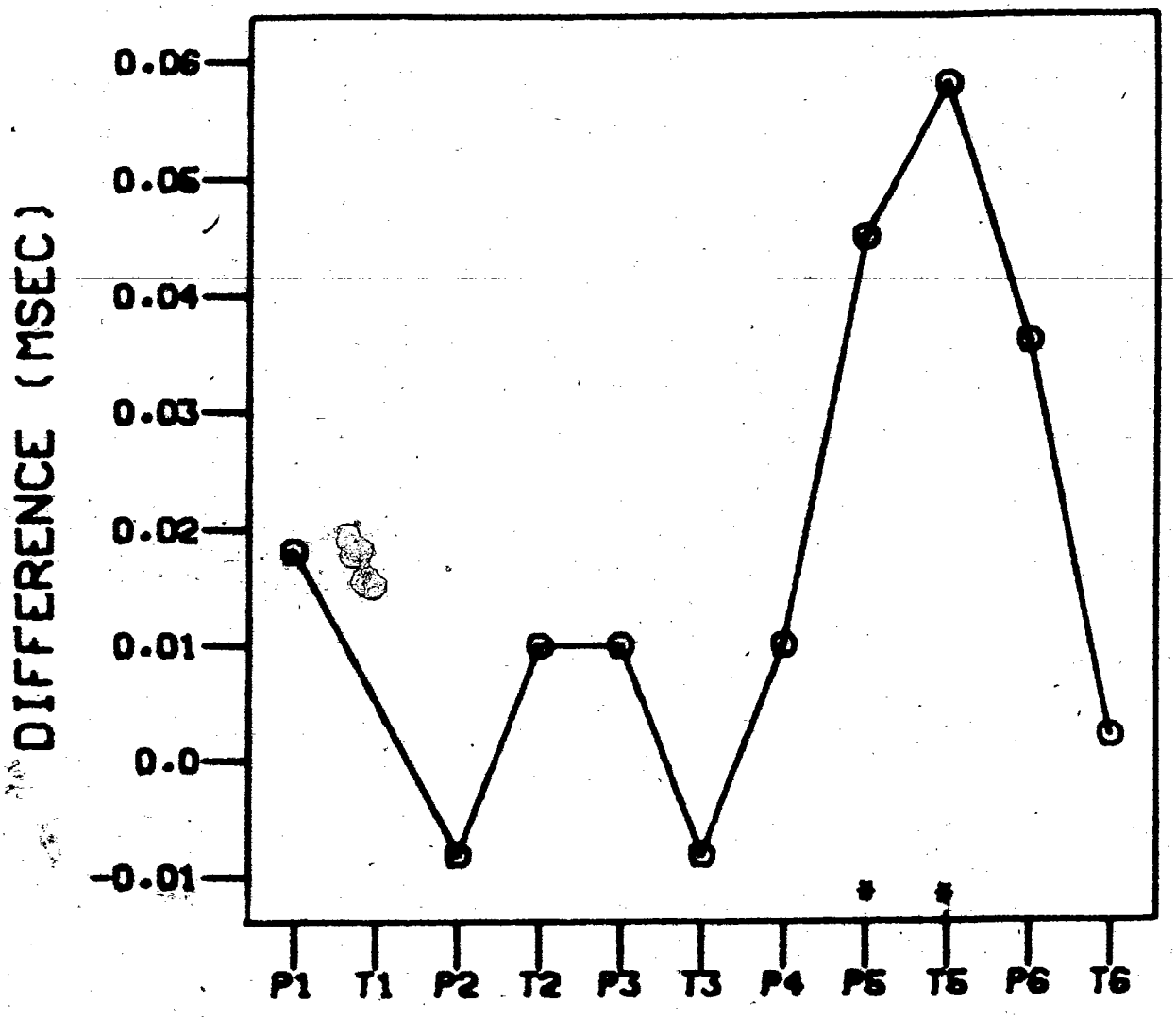


FIGURE 19

RAW LATENCIES - MON MINUS BI

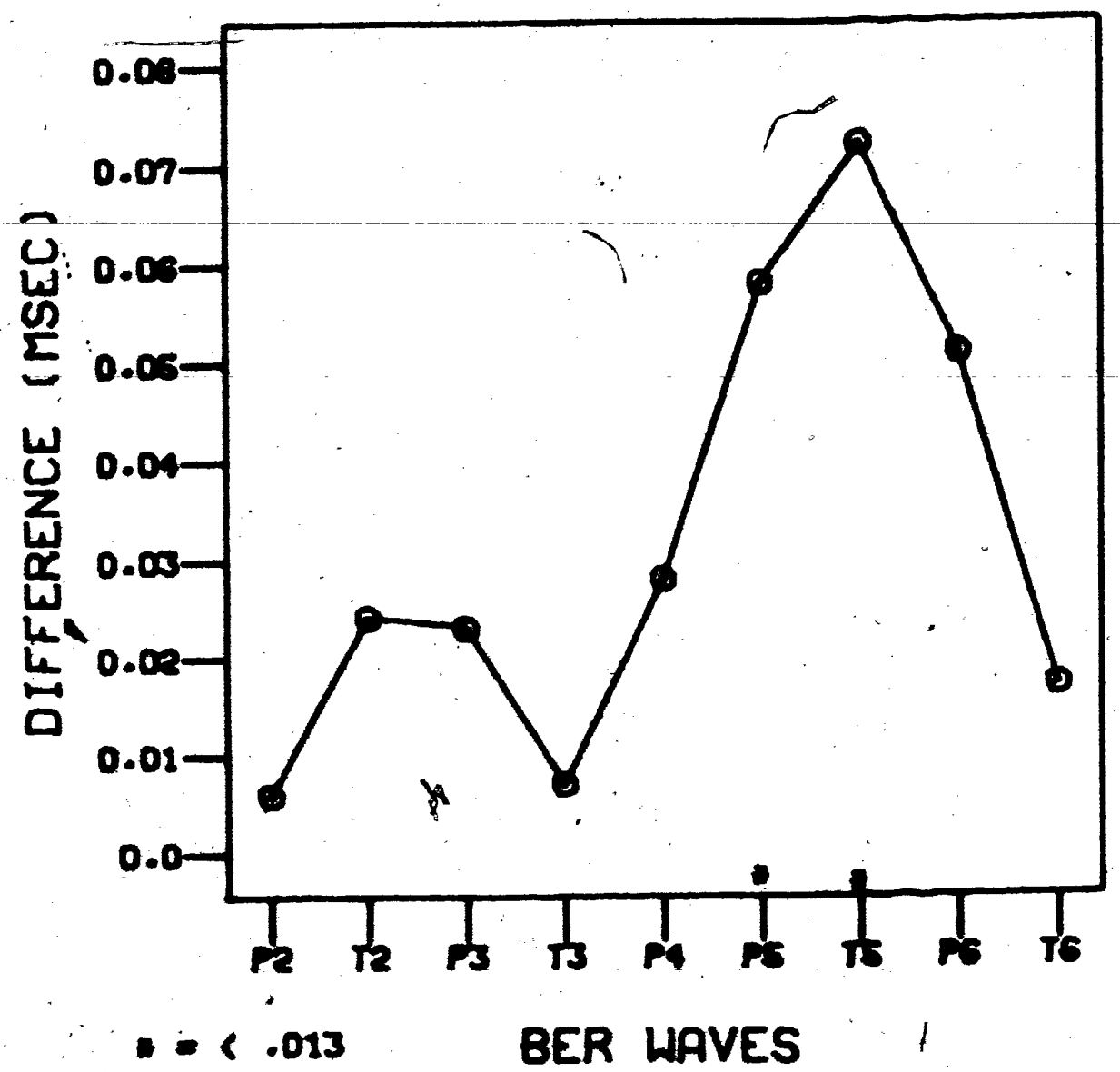


FIGURE 20
DERIVED LATENCIES
MON MINUS BI

T5: p = .002

P6: p = .035

T6: p > .100

2) Amplitude Measures

Figures 21 and 22 illustrate the BI/MON differences as a function of amplitude. In all cases where significance occurs, MON amplitudes are more positive than BI amplitudes. The following are the obtained p values for zero baseline amplitude data:

P1: p = .012

P2: p = .013

T2: p = .081

P3: p = .021

T3: p = .051

P4: p = .003

P5: p = .003

T5: p > .100

P6: p > .100

T6: p > .100

The following are the p values for peak-to-trough amplitude data:

T1: p = .009

P2: p = .013

T2: p > .100

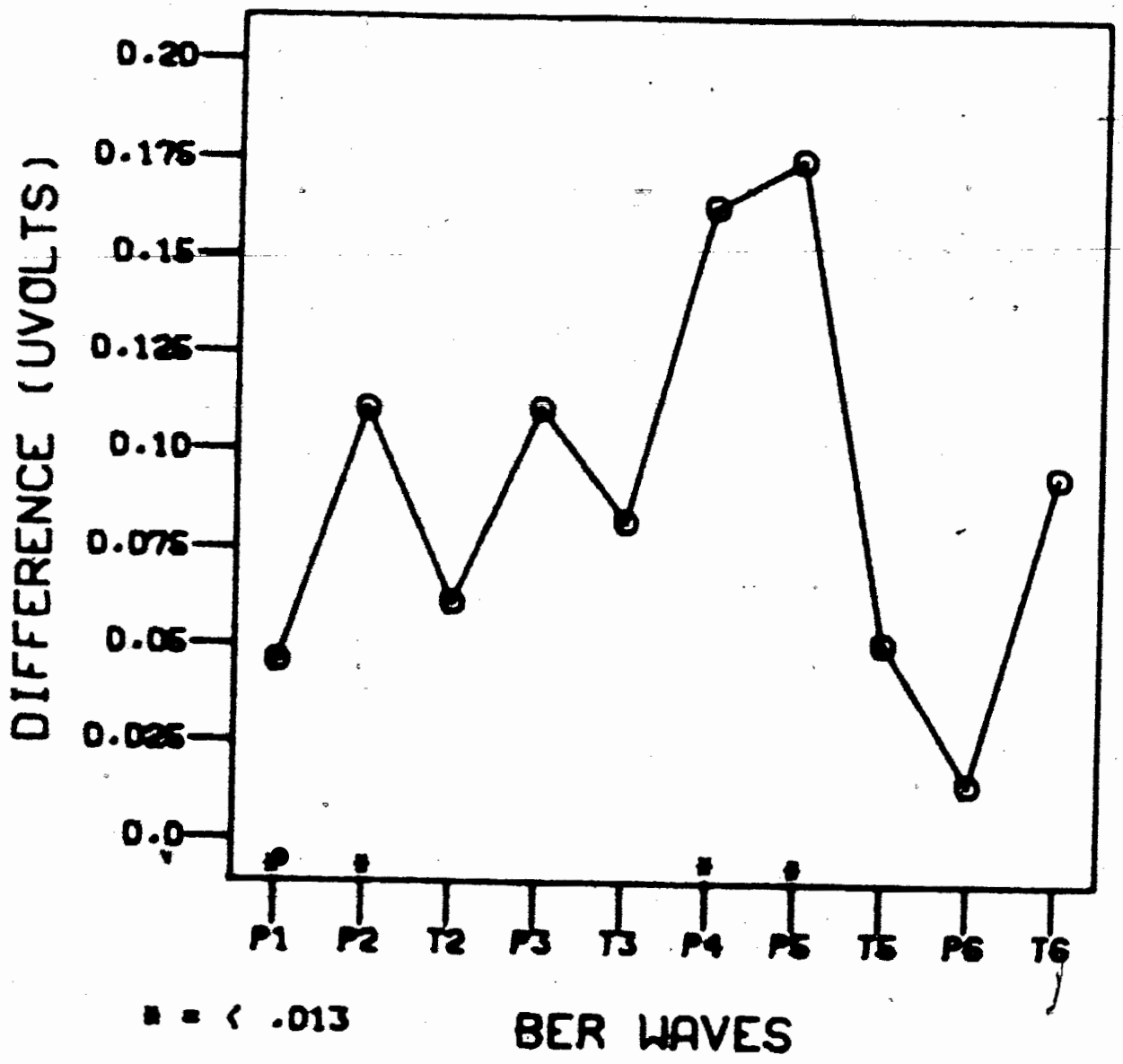


FIGURE 21

ZERO BASELINE AMPLITUDES
MON MINUS BI

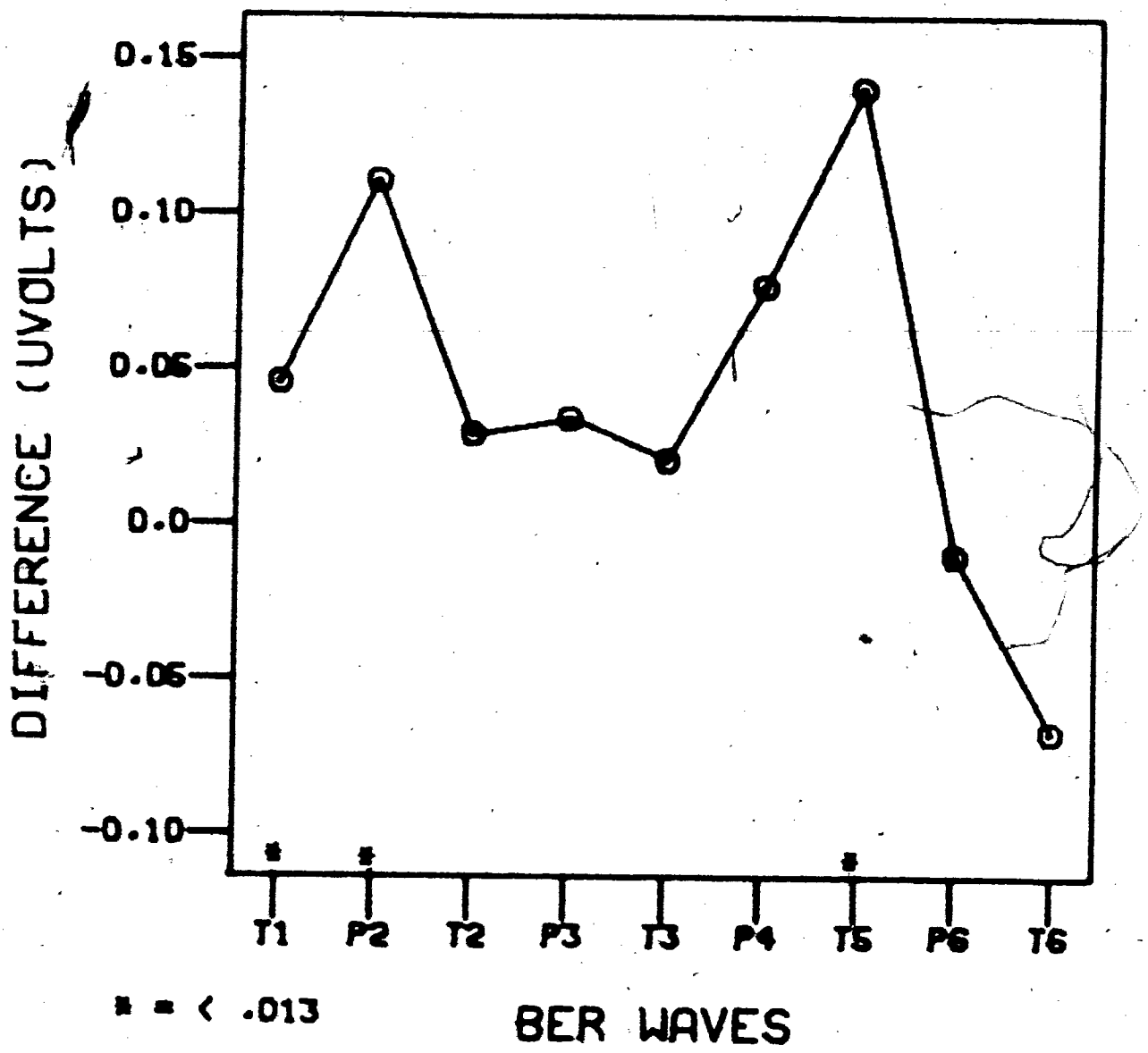


FIGURE 22

PEAK-TO-TROUGH AMPLITUDES
MON MINUS BI

P3: $p = .084$

T3: $p > .100$

P4: $p = .020$

T5: $p = .004$

P6: $p > .100$

T6: $p = .025$

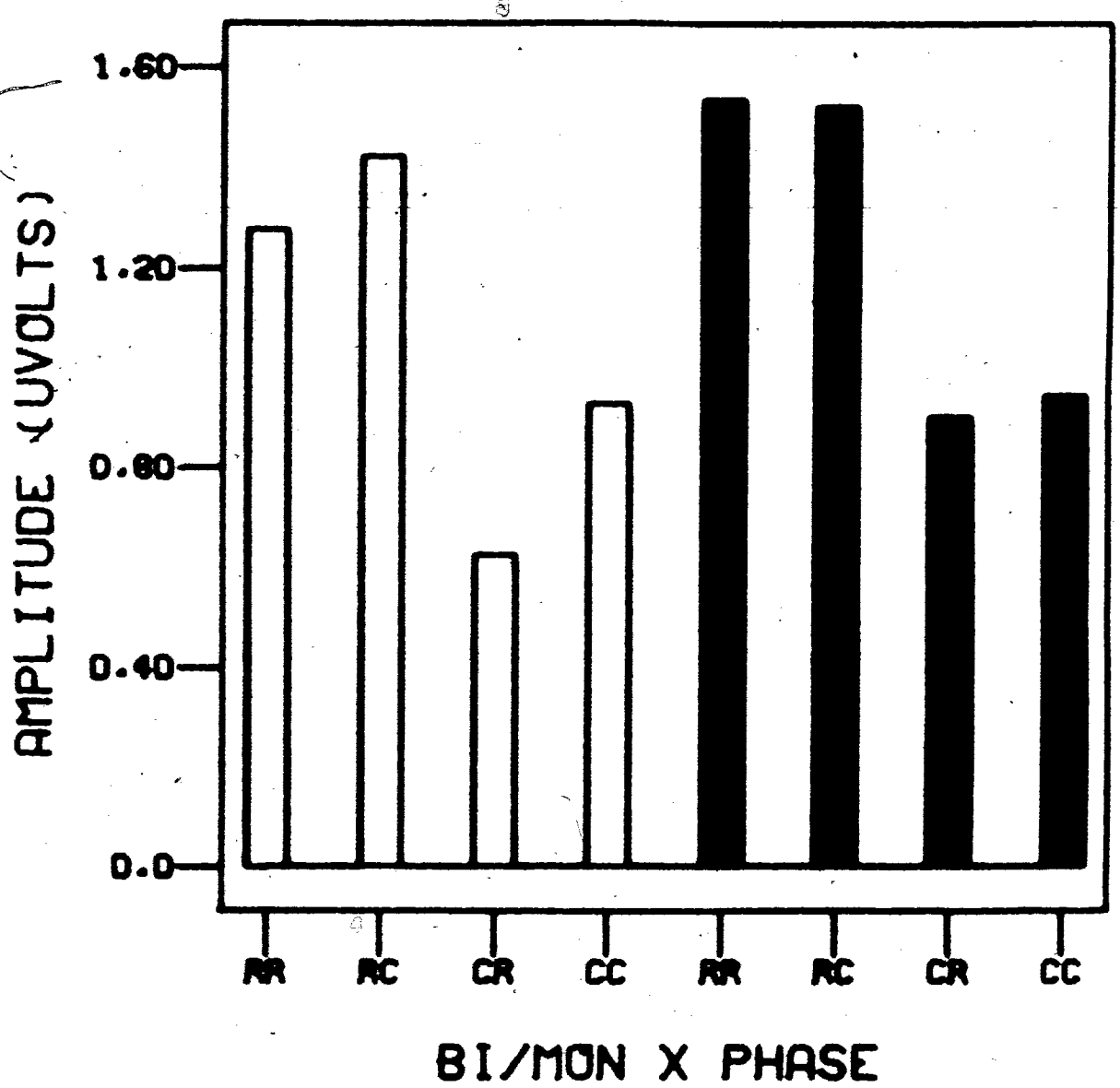
BI/MON x phase interactions also occurred for both conditions (Figures 23 through 25): at P4 ($p = .012$) and P6 ($p = .005$) for zero baseline amplitude data, and P6 ($p < .001$) for peak-to-trough amplitude data. The patterns shown are essentially the same as the derivation x phase interactions for the same peaks and troughs discussed in section B.

D. Phase Effects

1) Latency Measures

Figures 26 and 27 illustrate the latency differences for the four phase conditions at all peaks and troughs. The raw latency main effects at T5 ($p < .010$) and P6 ($p = .001$) do not occur for derived latencies. However, P3 ($p < .001$) occurred for derived latency data and not for raw latency data. T1 (raw latency data) did not meet the minimum requirements for non-symmetry.

FIGURE 23
ZERO BASELINE AMPLITUDES - P4



WHITE BAR = VERTEX/MASTOID
BLACK BAR = VERTEX/NONCEPHALIC

FIGURE 24
ZERO BASELINE AMPLITUDES - P6

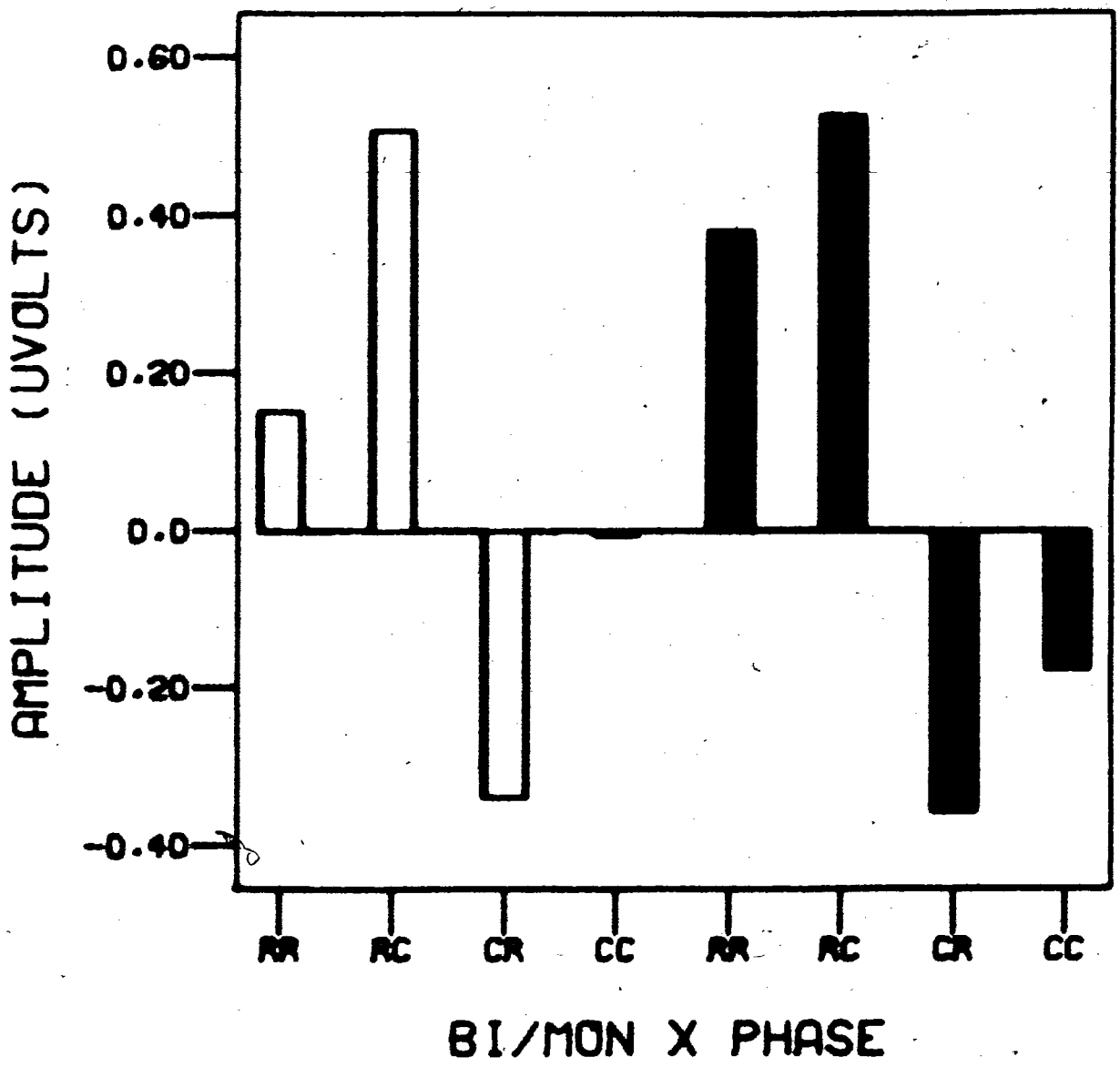
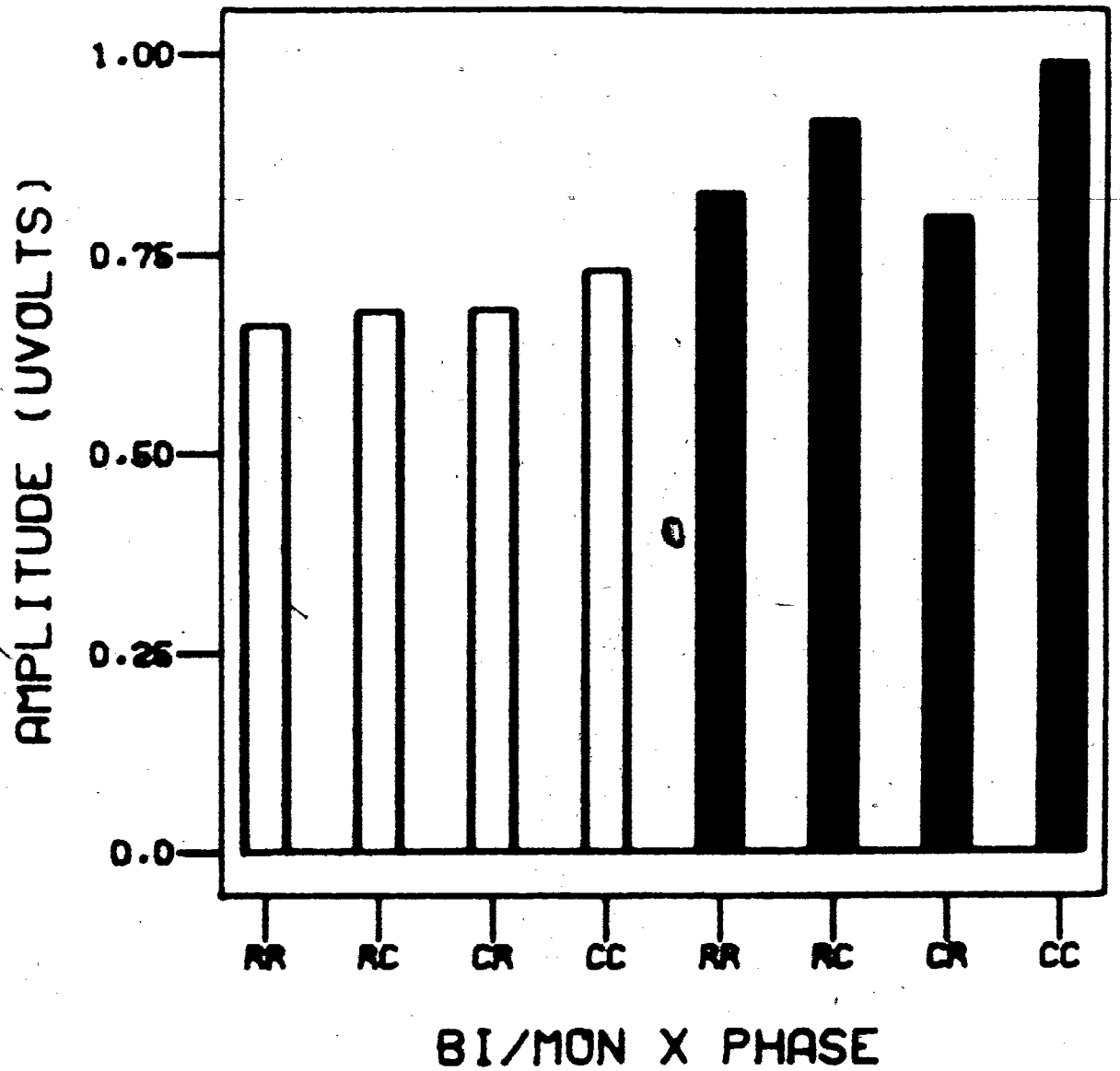


FIGURE 25
PEAK-TO-TROUGH AMPLITUDES - P6



WHITE BAR = VERTEX/MASTOID
BLACK BAR = VERTEX/NONCEPHALIC

FIGURE 26
RAW LATENCIES

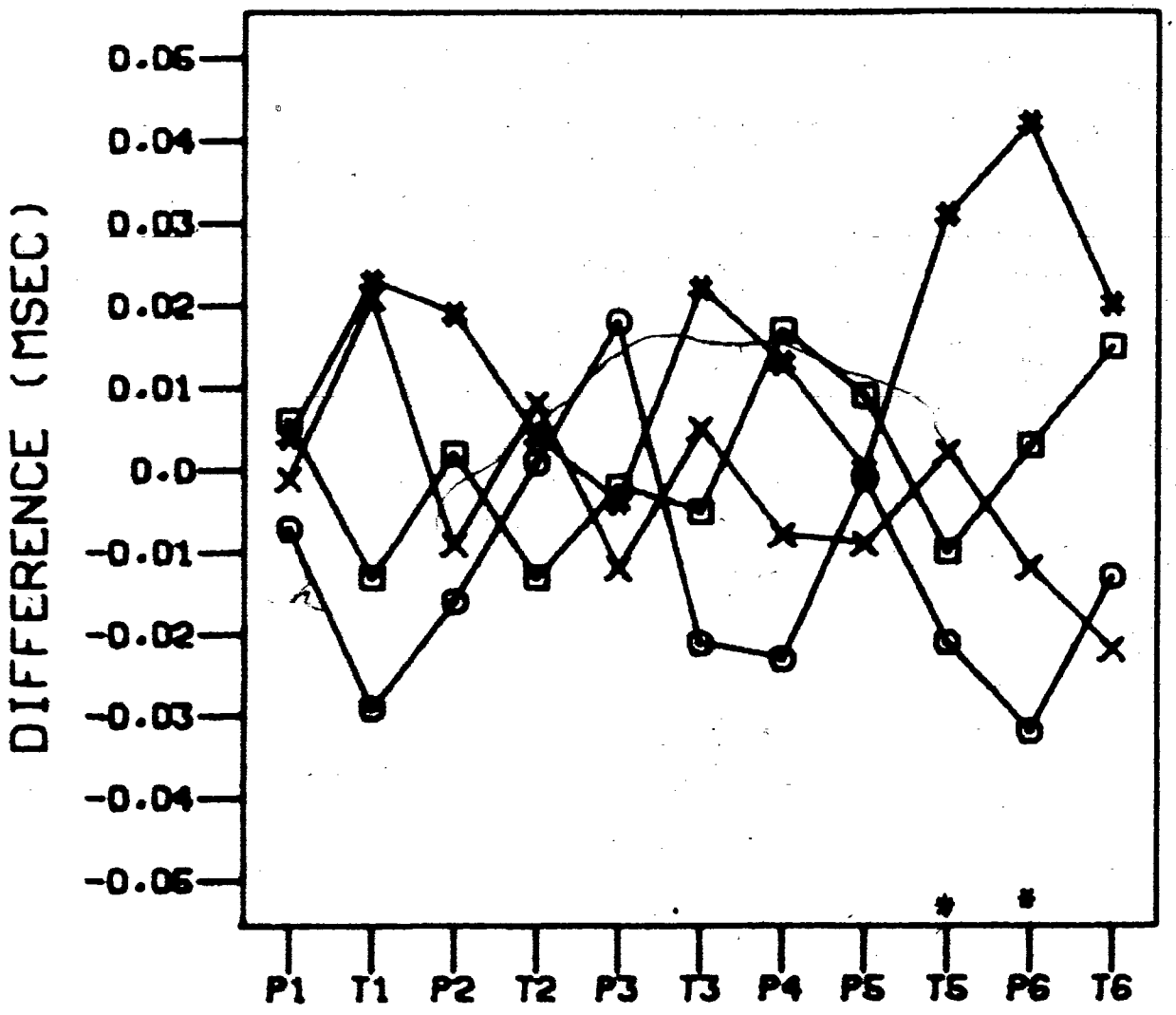
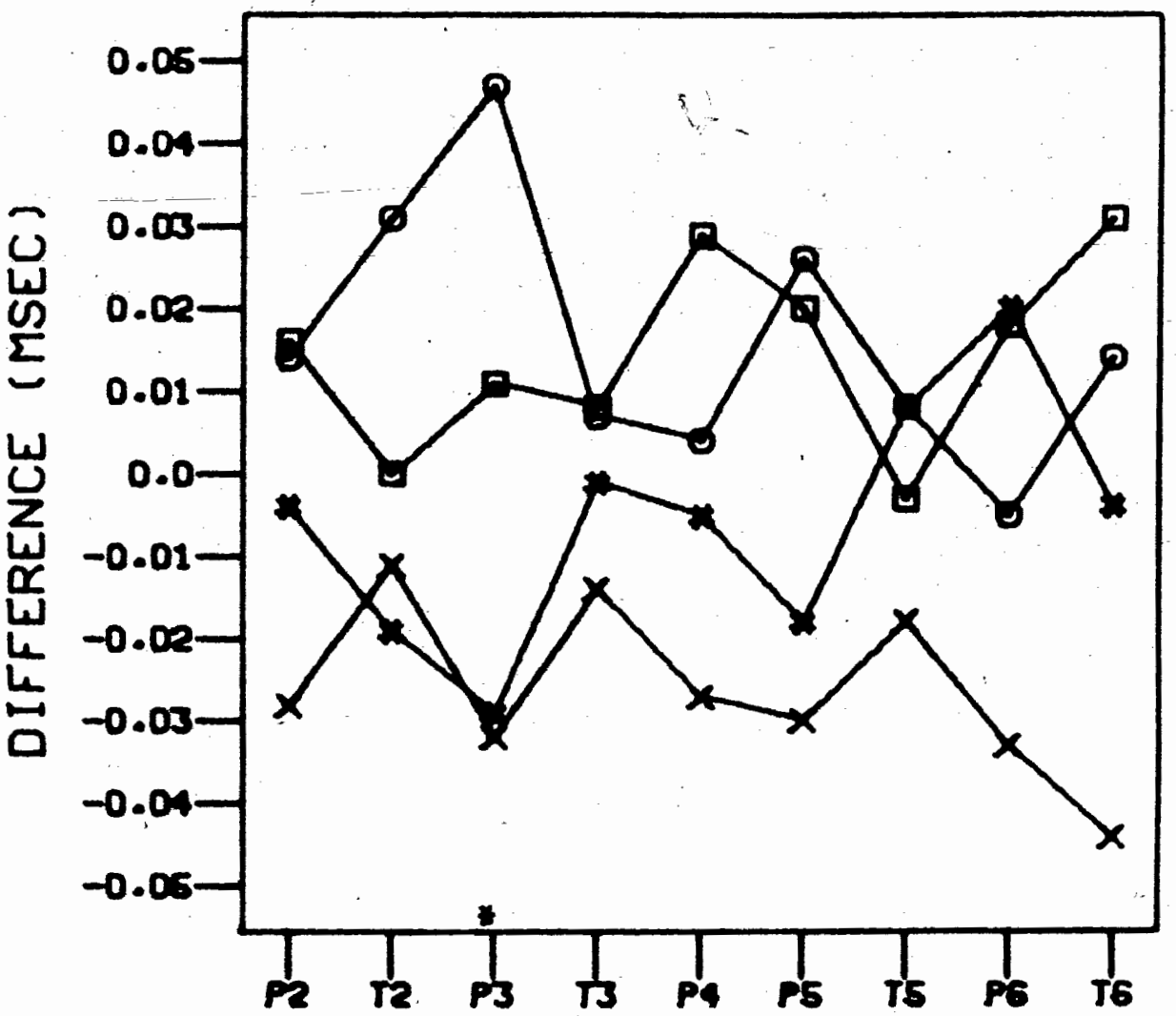


FIGURE 27
DERIVED LATENCIES



* = < .013

O---O = RR
□---□ = RC
X---X = CR
--- = CC

2) Amplitude Measures

Figures 28 and 29 illustrate the amplitude differences for the four phase conditions at all peaks and troughs. P2 through P6 main effects proved significant for zero baseline data:

P1: $p > .100$

P2: $p < .001$

T2: $p = .001$

P3: $p < .001$

T3: $p = .001$

P4: $p < .001$

P5: $p < .001$

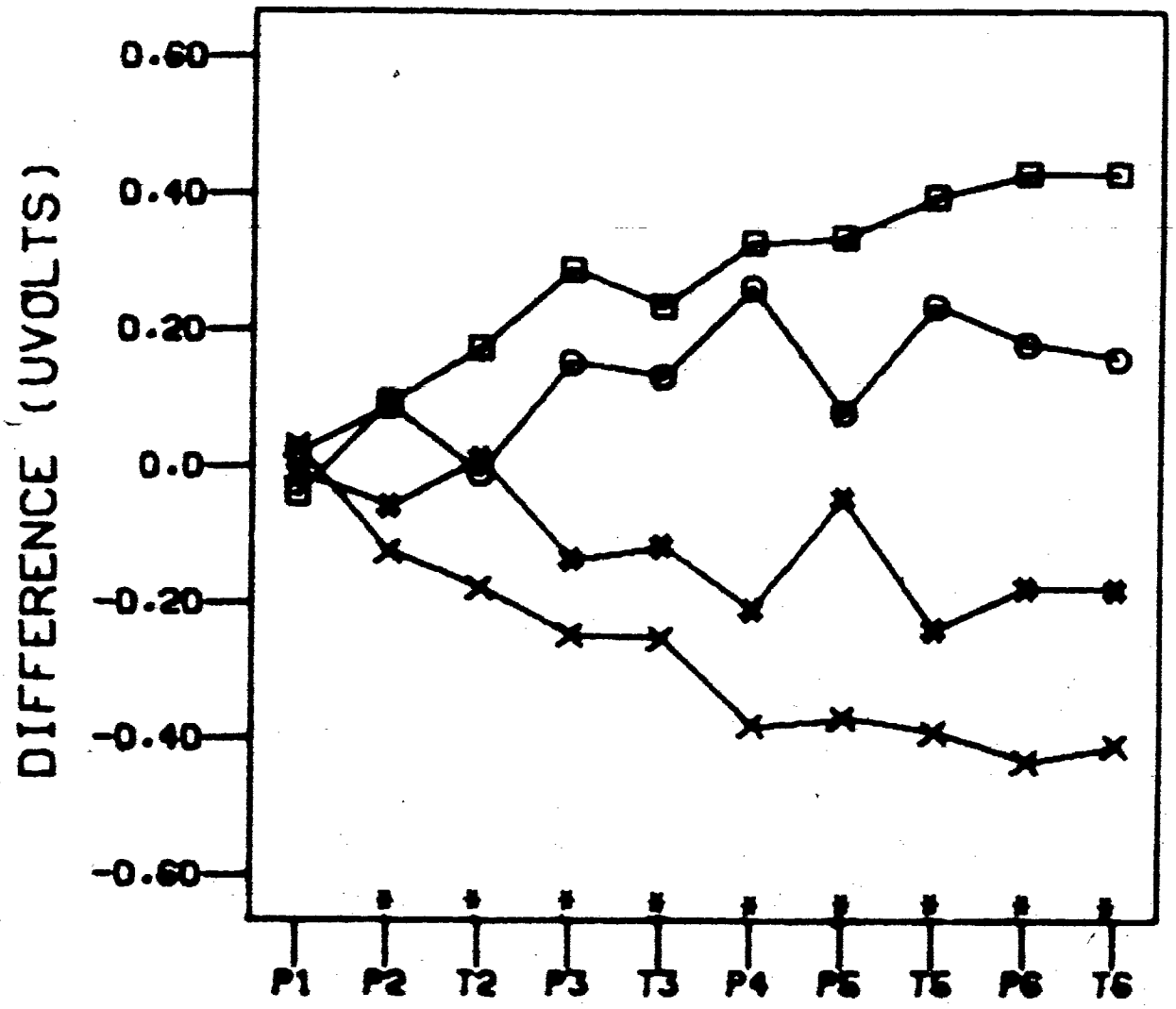
T5: $p < .001$

P6: $p < .001$

T6: $p < .001$

The pattern is two-fold: for the right ear (with the left ear held constant, either R or C) a change from a rarefaction to a condensation click results in a decrease in the amplitude of the peaks and troughs; for the left ear (with the right ear held constant) a change from a rarefaction to a condensation click results in an increase in the amplitude of the peaks and troughs. The Newman-Keuls Studentized Range Statistic (Appendix F) shows, however, other than the CC/CR difference at T2 and P5, the effect of stimulus phase presented to the left ear can only be considered a non-significant trend.

FIGURE 28
ZERO BASELINE AMPLITUDES

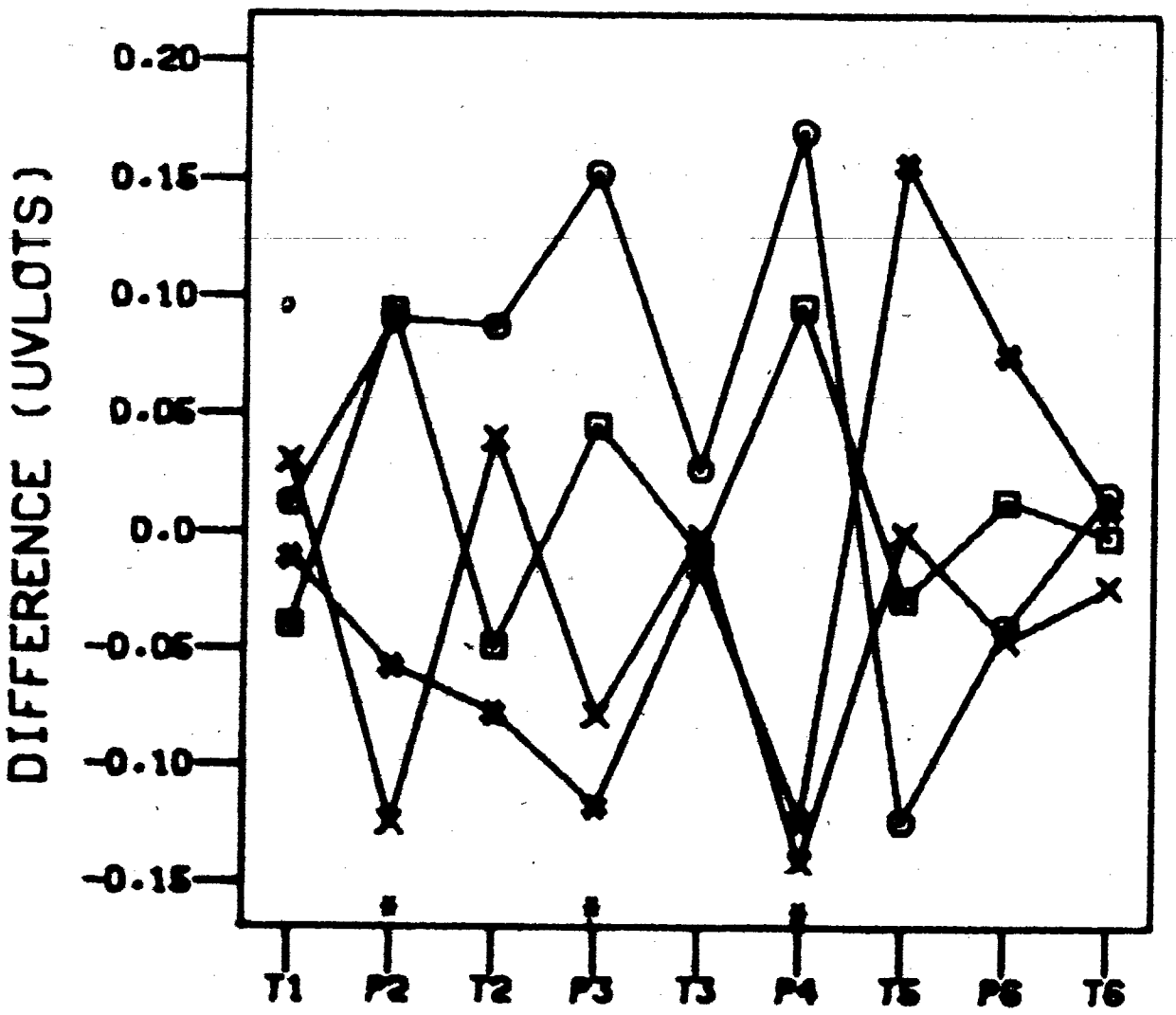


* = < .013

BER WAVES

O---O = RR
□---□ = RC
X---X = CR
--- = CC

FIGURE 29
PEAK-TO-TROUGH AMPLITUDES



* = < .013

O---O = RR
□---□ = RC
X---X = CR
--- = CC

Peak-to-trough amplitude main effects showed the following p values, although T2 and T5 did not meet the minimum requirements for non-symmetry:

T1: $p > .100$

P2: $p < .001$

T2: $p = .004$

P3: $p < .001$

T3: $p > .100$

P4: $p < .001$

T5: $p = .005$

P6: $p > .100$

T6: $p > .100$

The RC/CR means show P2, P3 and P4 (for RC) to be more positive for peaks and more negative for troughs (i.e., it indicates a larger waveform, as compared to the CR waveform, for these peaks and troughs). The RR/CC means show that RR is more positive for P2, P3 and P4, (see Newman-Keuls Tables Appendix F and G). It is interesting to note that the RR/RC and CC/CR differences do not reach significance. Thus, as with zero baseline data, differences arise primarily from a change in phase presented to the right ear.

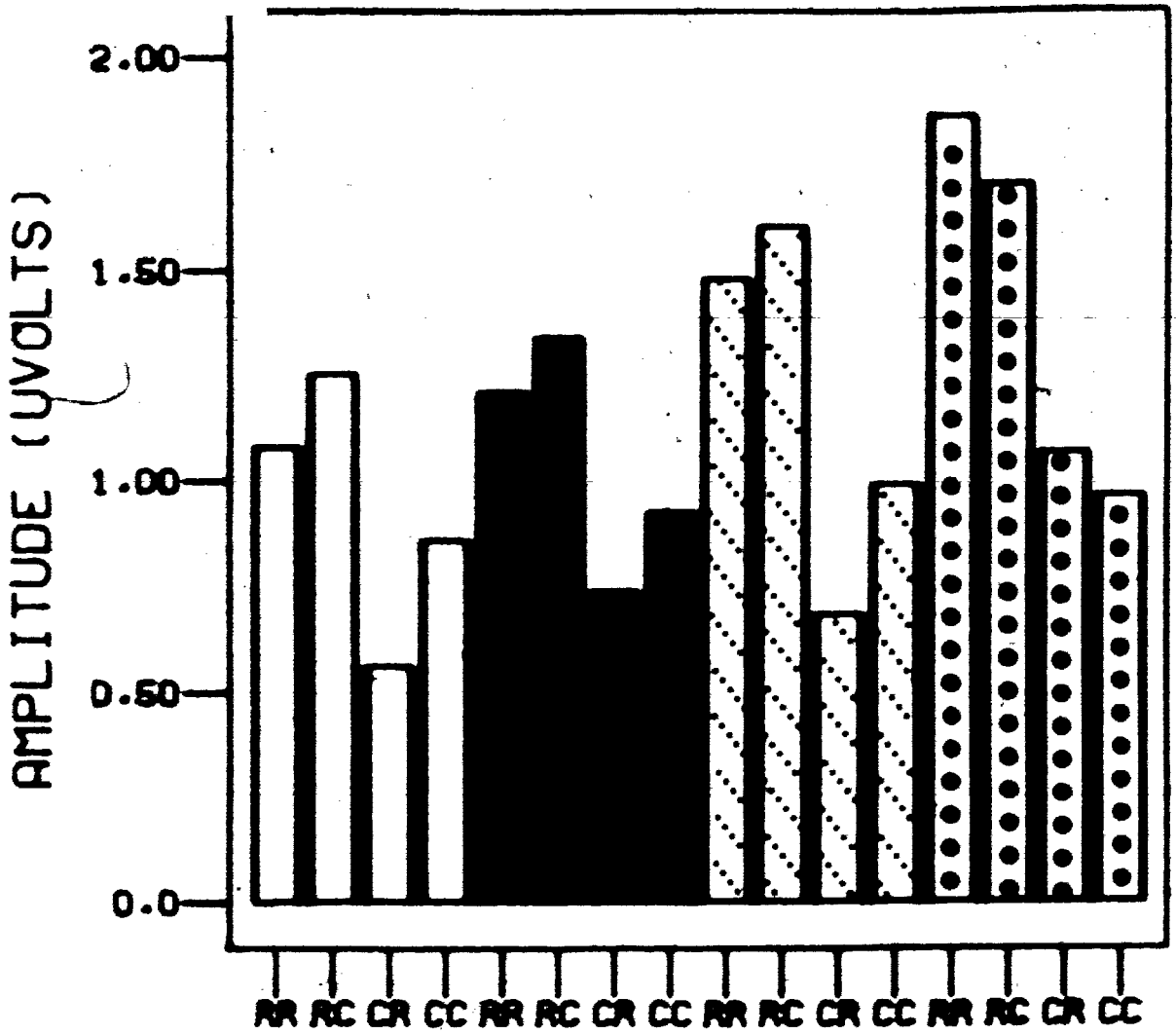
All interactions have been discussed above. These include derivation x phase interactions at T1, P4 and p6 for peak-to-trough amplitudes; and, BI/MON x phase interactions at

P6 for peak-to-trough amplitudes. Two three-way interactions occurred for zero baseline data at P4 ($p = .009$) and P6 ($p = .011$) (Figures 30 and 31). They basically show the same functions discussed above; VN results in larger waveforms than VM, and R presented to the right ear results in greater amplitudes than C presented to the right ear. For P4, there is also the indication that R presented to the right ear results in greater amplitudes in the MON condition than in the BI condition.

E. Mastoid/Mastoid (MM) Derivation

Figure 32 shows the composite waveforms for the MM derivation (left mastoid active). Since a consistent waveform was obtained it is clear that the mastoid is an active BER site. The waveforms for monaural left and right stimulation are almost mirror images, and when either summed monaural or binaural waveforms are plotted, these mirror images essentially cancel (Figure 33). Figure 34 shows the composite waveform for right minus left waveforms. The peaks and troughs, as labelled, compare closest with the peak and trough latencies of

FIGURE 3D
ZERO BASELINE AMPLITUDES - P4

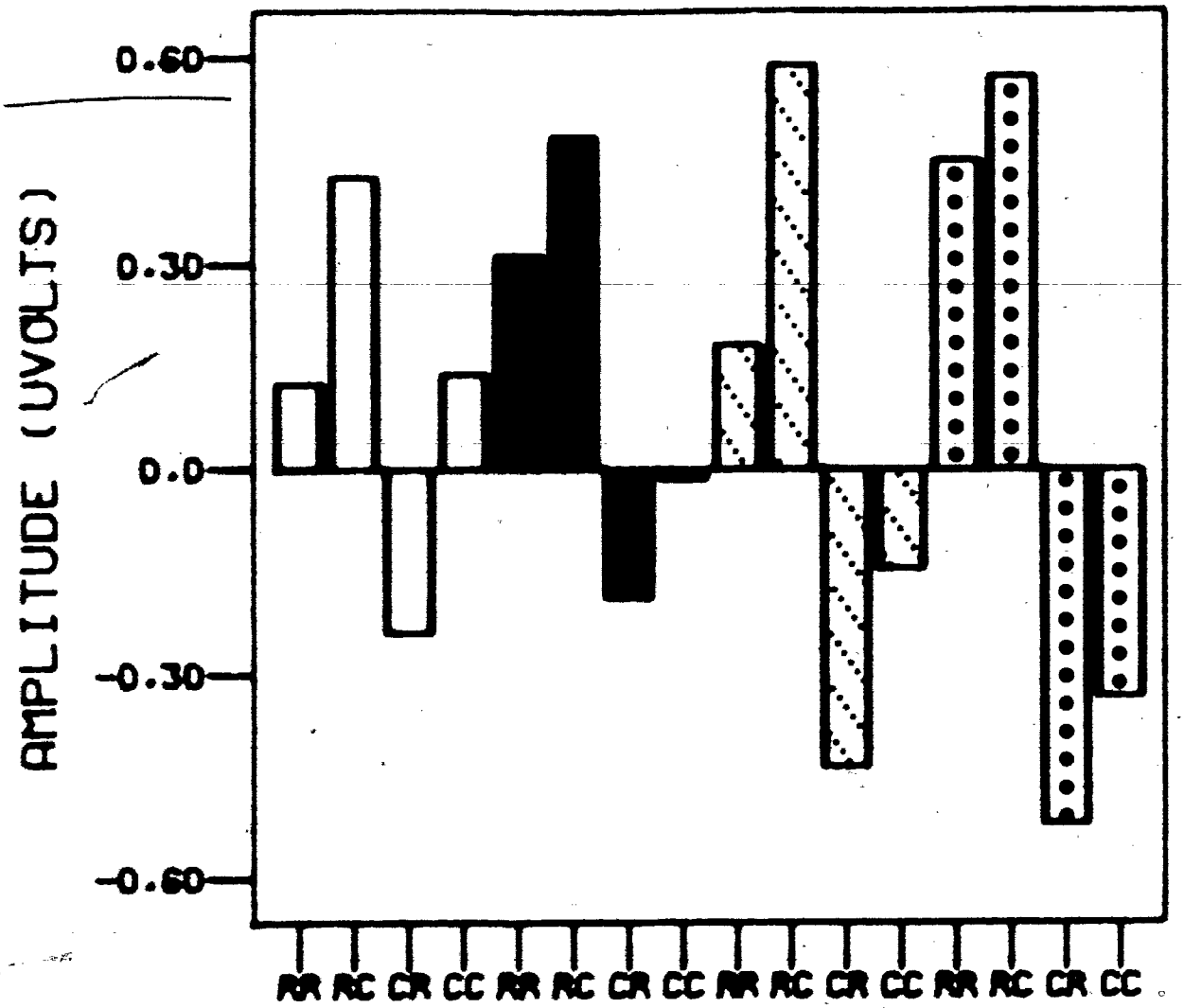


DERIVATION X BI/MON

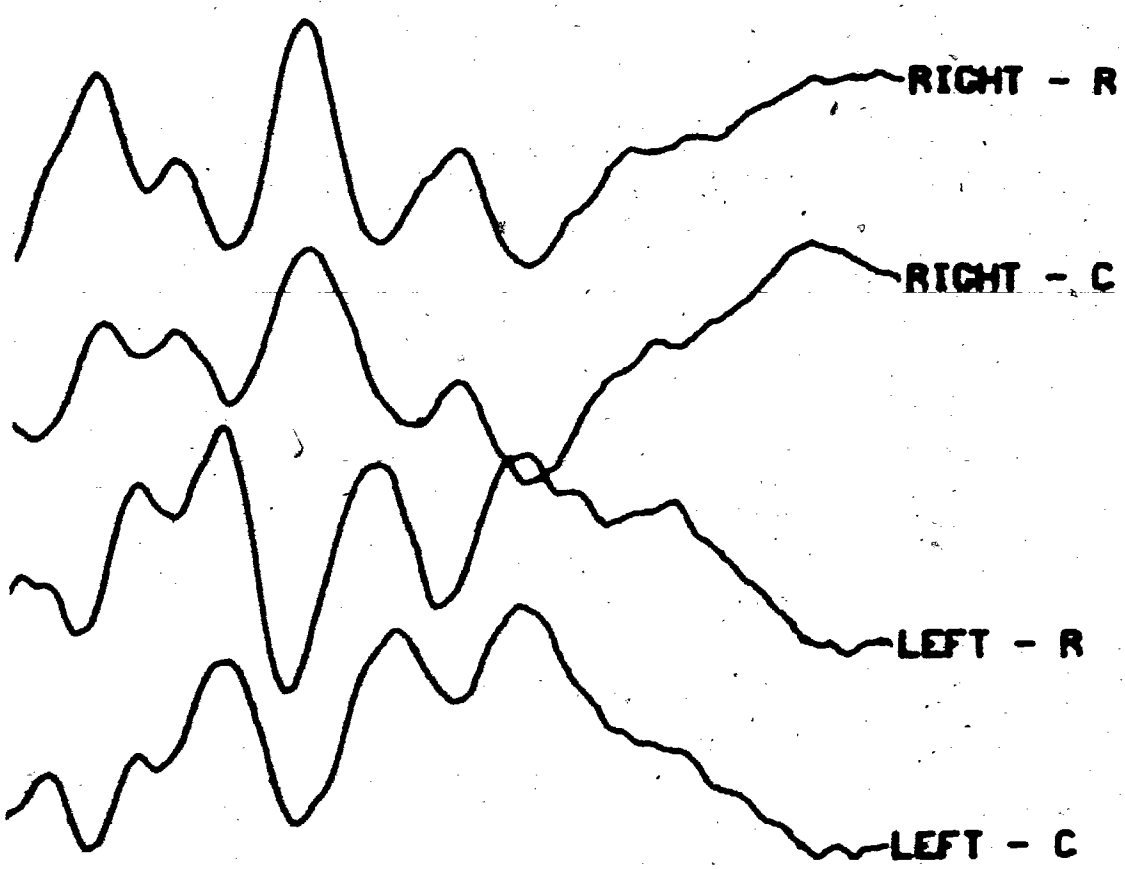
X PHASE

WHITE BAR = VM - BI
BLACK BAR = VM - MON
STRIPED BAR = VN - BI
DOTTED BAR = VN - MON

FIGURE 31
ZERO BASELINE AMPLITUDES - P6



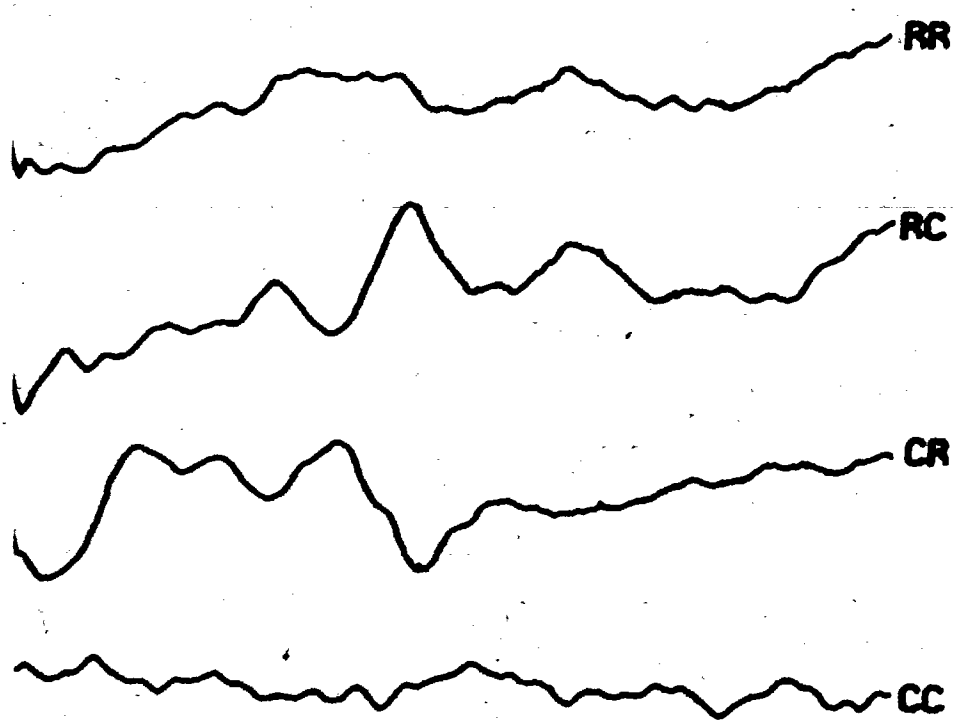
WHITE BAR = VM-BI
BLACK BAR = VM-MON
STRIPED BAR = VN-BI
DOTTED BAR = VN-MON



COMPOSITE MONAURAL
MASTOID/MASTOID WAVEFORMS

FIGURE 32

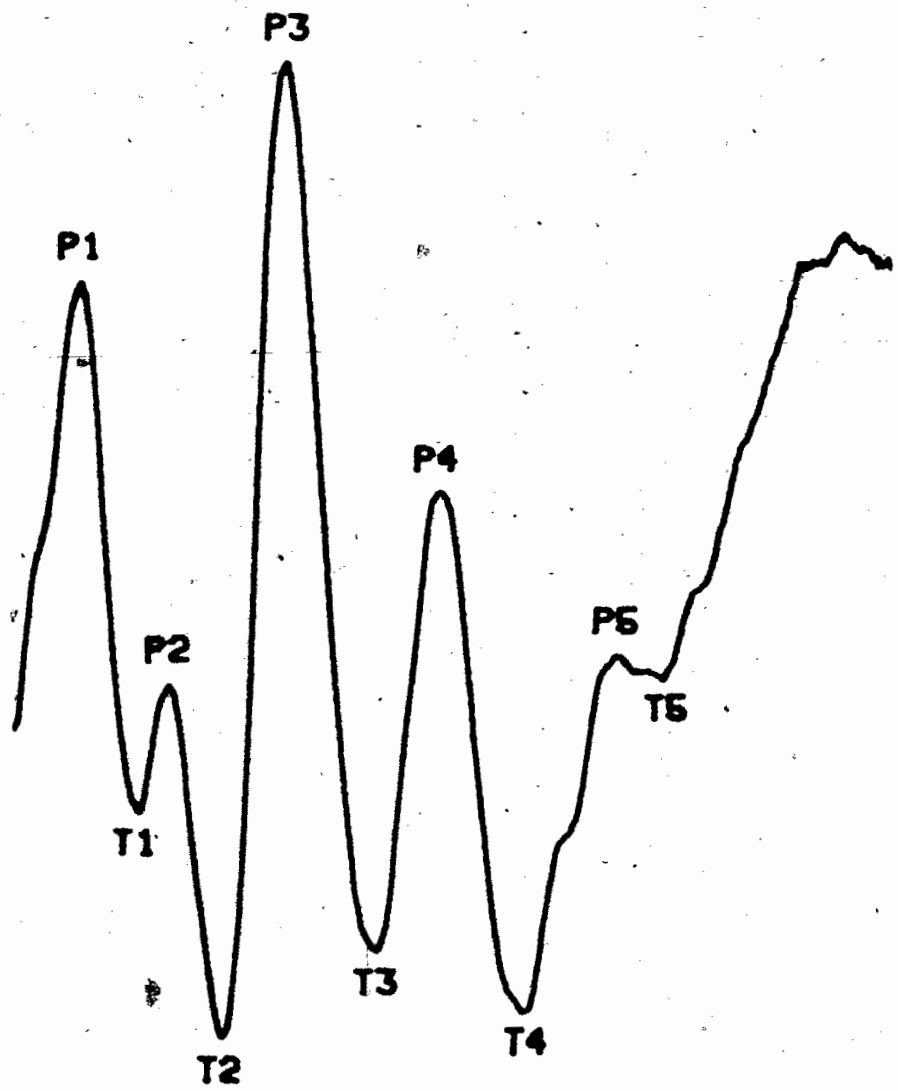
1 INCH = 2.56 MSEC
3.0 CM = 0.25 UVOLTS



COMPOSITE BINAURAL
MASTOID/MASTOID WAVEFORMS

FIGURE 33

1 INCH = 2.56 MSEC
3.0 CM = 0.25 UVOLTS



COMPOSITE MASTOID/MASTOID WAVEFORM

FIGURE 34

1 INCH = 2.56 MSEC
6.5 CM = 0.25 UVOLTS

Figure 9 as follows:

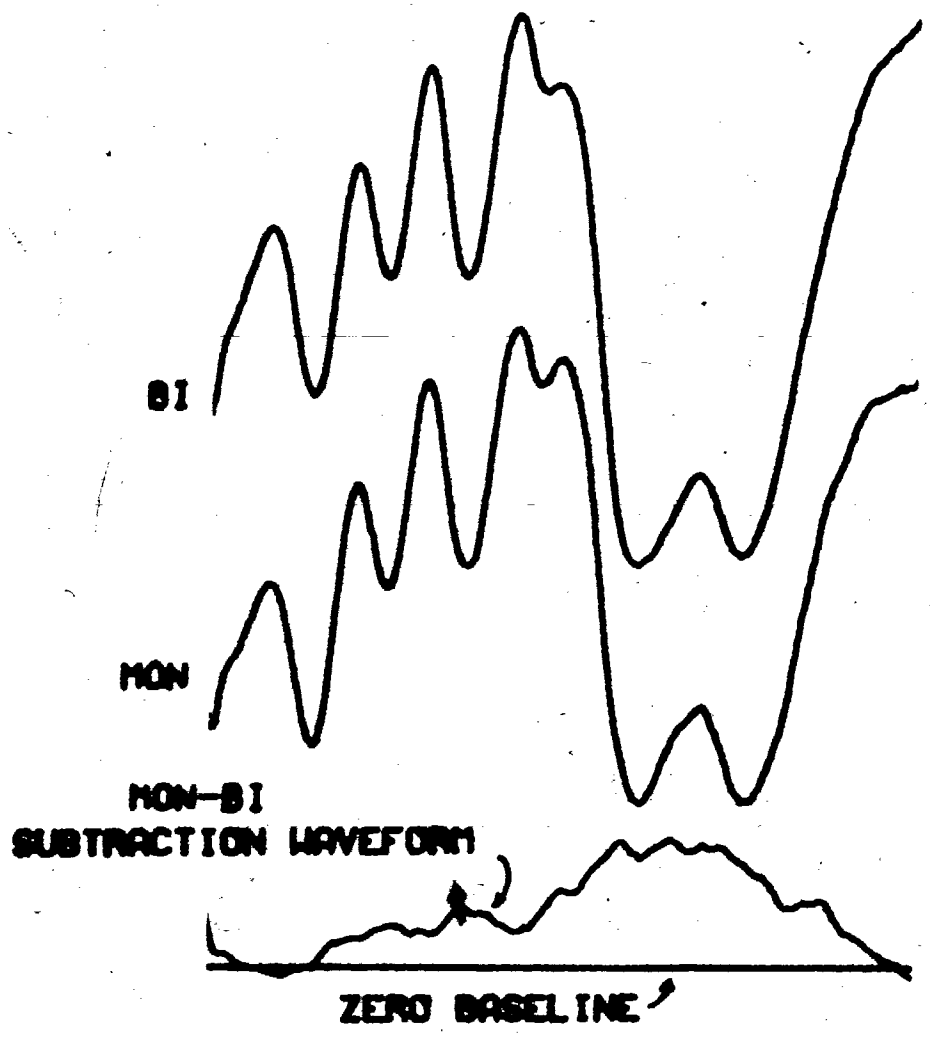
Mastoid Active		Vertex Active
P1 = 1.73 msec	--	P1 = 1.80 msec
T1 = 2.27 "	--	T1 = 2.32 "
P2 = 2.59 "	--	P2 = 2.94 "
T2 = 3.10 "	--	T2 = 3.33 "
P3 = 3.87 "	--	P3 = 3.78 "
T3 = 4.74 "	--	T3 = 4.31 "
P4 = 5.44 "	--	P5 = 5.67 "
T4 = 6.27 "	--	T5 = 6.42 "
P5 = 7.26 "	--	P6 = 7.53 "
T5 = 7.78 "	--	T6 = 7.99 "

What is of interest is that only P3 and T3 show longer latencies for the mastoid active waveform as compared with the vertex active waveform.

F. Composite Monaural Combined vs Binaural Waveforms for Delta I and Delta T.

1) Delta I (Figure 35)

The difference waveform was obtained by subtracting the composite BI waveform (average waveform across all subjects and conditions) from the composite MON waveform. What is apparent is that Delta I did not result in a BI/MON latency shift at the peaks and troughs, but did result in a generally more positive



DELTA I COMPOSITE WAVEFORMS

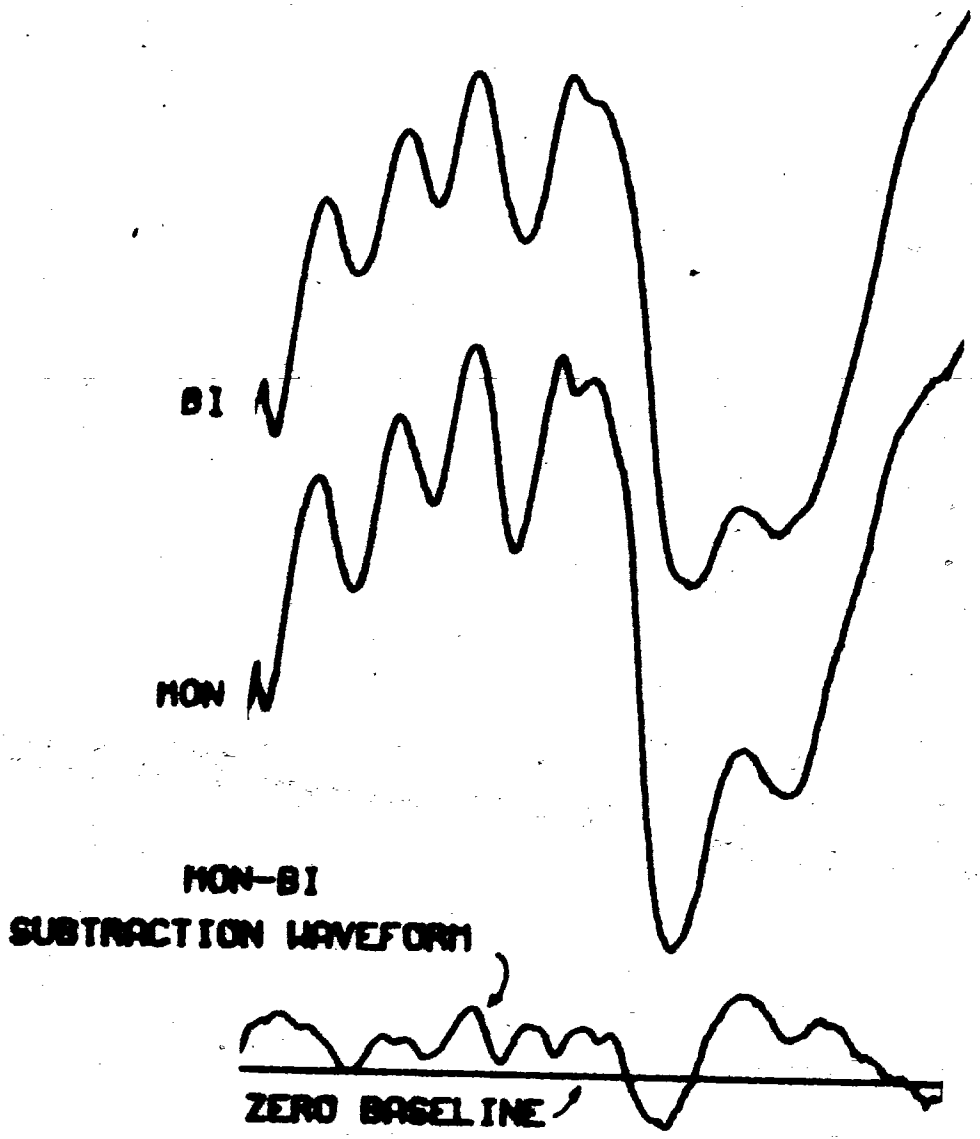
FIGURE 35

1 INCH = 2.56 MSEC
2.0 CM = 0.32 UVOLTS

(with T1 baseline) waveform for MON, and larger differences for BI at P3/T3, P4/T3 and P5/T5.

2) Delta T (Figure 36)

The difference waveform was obtained in the same manner as Delta I. The waveforms do not show the same amplitude function as Delta I (although there appears to be a tendency toward an overall larger MON waveform). The specific amplitude differences appear to be a decreased peak-to-trough amplitude at T1/P1, T3/P3, T5/P5 and P6/T5.



DELTA T COMPOSITE WAVEFORMS

FIGURE 36

1 INCH = 2.56 MSEC
2.0 CM = 0.32 UVOLTS

DISCUSSION

A. Derivation

Amplitude differences between VM and VN derivations were expected. As early as 1974, Terkildsen et al. showed that by moving the reference electrode from an aural region to either the homolateral or contralateral side of the neck, the action potential complex reduces in size while later neural potentials gain in both definition and amplitude. Similar findings were reported by Allen and Starr (1978) in monkeys. The basic hypothesis is that the early waves comprising the AP complex (BER peaks one through three) are heterophasic when comparing the mastoid to the vertex, while the later waves are homophasic. Thus, a change in reference from a mastoid to a non-cephalic, or neutral BER site should result in amplitude decreases (and possibly latency changes) in the early waves, with the opposite effect in the later waves.

In the present study, using a seventh-cervical process reference, the VN derivation did give a larger potential, however, this occurred for all waves other than P1. In general, for both the waveform amplitudes (amplitudes based on zero voltage at T1) and the peak-to-trough absolute amplitudes, VN resulted in an overall larger waveform. Why do the present

data not show the same P1 shift in amplitude as shown by Terkildsen et al., and Allen and Starr? In both the above studies, monaural stimuli were used (4000 Hz tone pips in the Terkildsen et al. study with no information as to direction of initial transducer diaphragm movement; and, rarefaction 10 usec clicks in the Allen and Starr Study), whereas binaural stimuli were used in the present study. Although the definitive experiment comparing monaural and binaural stimuli as they effect P1 has not been done, it may be that the opposite polarity potential produced at the contralateral mastoid (as compared to the ipsilateral mastoid), shown in Figure 33 and also reported by Picton et al. (1974), may have resulted in a reduction of amplitude at the mastoid. If one can assume the heterophasic properties suggested by Terkildsen et al. then the proposed cancellation effect of the binaurally produced mastoid potential as seen by the differential amplifier would result in a smaller P1 when compared with ipsilateral monaural stimulation. In effect, the binaural evoked potential with a mastoid reference would more closely resemble the potential recorded with a non-cephalic reference than the equivalent monaural comparison. On the other hand, the neck reference site used by Terkildsen et al. may not have been a completely neutral site (as suggested by Allen and Starr with monkeys) and may have resulted in a potential (as seen by the differential amplifier) that may have decreased the early potential recorded

at the vertex. The conclusion reached is that VN results in an overall larger waveform as compared to VM, which indicates a homophasic pattern for all waves at both the vertex and mastoid as compared with the non-cephalic site when binaural stimuli are presented.

The latency effects of these two electrode derivations indicate an important feature: latencies for P2 and T2 are significantly shorter for VM than VN for raw latency measures, and become nonsignificant when the latency of T1 is subtracted. This gives support to the suggestion (Picton et al., 1974) that the P2 generator source mimics a transverse horizontal dipole. In addition, there is an indication that the two troughs do reflect CAS information processing suggesting the need to include both peak and trough measurements in all BER studies.

B. Binaural vs Combined Monaural Stimulation

The comparison of binaural evoked responses with combined equivalent monaural evoked responses was expected to indicate the presence of binaural interaction effects. As stated previously, a decreased amplitude at any peak in the BI condition, as compared with the MON condition, would be taken as an indication of binaural interaction within the generator source. For amplitude data, the results are not as definitive as expected. Other than the zero baseline differences at P4, both amplitude analyses show the same peak significances (i.e.,

originates from the SOC. However, since the mean difference shown by P2 and P3 for Zero Baseline data are essentially the same, it may also be that a greater variation in responding is shown by P3, resulting in smaller F's. Speculation must stop at this point since implying that P2 reflects SOC involvement is at odds with past correlational studies, and, to suggest that CN binaural processing has been shown is at odds with single unit studies. This must be viewed with caution. The P1 differences are only slightly greater than the minumum cutoff of 0.04 uvolts and may reflect more T1 functioning than P1 functioning. No reasonable explanation for this effect can be given at this time, and must wait until replication has been shown. Conclusions based on the amplitude differences resulting from the BI/MON conditions suggest that there is both an early binaurally acting component (possibly the SOC) and a later component (P5 - possibly reflecting IC involvement). The difference between zero baseline data and peak-to-trough data for P4 may indicate that the T3/P4 difference is not the critical measure for P4. A second possibility is that the BER waveform is the result of two overlapping components. One results in the BER slow wave (i.e., a wave with greatest positivity in the P4-P5 range) and the other in fast activity resulting in the six peak/trough discursions riding on the slow waveform. If both components result in an increase in the P4 range, whether these increases

are significant or not for the slow wave, the coincidental algegraic summation of the two may have resulted in the zero baseline amplitude showing significance. The Derivation x BI/MON x Phase interaction suggests that the P4 difference for BI/MON is the result of the rarefaction click stimulus presented to the right ear. This suggests (as discussed in C below) that a basic difference exists between the ears in the coding of differences in phase.

The latency effects are of particular interest: since the significantly shorter latencies seen at P5 and T5 for BI raw latency data are also present for derived latency data, support is given to Hallman's (1977) finding that the peaks and troughs are not simply relay stations of end organ coding, but also represent CAS information processing. Since Figures 19 and 20 do not show a progressive decrease in latency for BI in the peaks and troughs preceeding P5, the difference is not simply an accumulation effect; that is, it is not the result of nuclei reaching threshold progressively sooner along the CAS as a result of greater BI input. Therefore, it must be concluded that P5 and T5 represent binaural coding resulting in a specific latency decrease, concomitant with the decreased amplitude at P5 when compared with MON. In addition, since significance was not obtained for P1, T1 or P2, this may be an indication of differential binaural information coding of the early binaurally acting component as compared with the later

component. Stotler (1953) has shown MSO cells to have two large dendrites that extend horizontally in opposite direction. Contralateral CN inputs terminate on the medial dendrites, while ipsilateral CN inputs terminate on the lateral dendrites. Galambos et al. (1959) showed that stimulation of one ear resulted in MSO potential fields of opposite polarity when compared to stimulation of the opposite ear. These two findings may provide a reasonable explanation for the differential BI/MON response of T1-P2 and P5. If the MSO neurons are involved in a form of divergence, rather than convergence, along the line of Jeffress's place theory for lateralization; it may be that a greater variety of neurons are involved in BI stimulation. The result should be greater BI amplitude with no particular decrease in latency. However, since any MSO neurons binaurally innervated would result in potential fields that tend to cancel, one may postulate a decrease in recorded BI voltage.

C. Phase

Phase was expected to result in a function that would be relatively constant over the first four or five peaks and troughs. This was supported by the relatively linear zero baseline amplitude functions, and the lack of derived latency changes (other than P3). Since P2 is the first wave showing significance for the zero baseline amplitude phenomena, in

terms of classical BER research, the CN is the first nucleus that is likely to be specifically involved. However, since binaural interaction at this level can occur only via two synaptic delays from the opposite CN, it is doubtful whether the time factor is great enough to be accounted for by this OCB input; in addition, this type of binaural interaction has not been shown in humans (Gulick, 1971). Thus, the effect is likely the result of coding at the cochlea. Since the effect appears relatively linear over all peaks and troughs, along with the results of the BI/MON comparisons for P4 discussed above, it may be that the CAS is comprised of an information processing duality: one system encodes the time and intensity components of auditory stimuli and transmits this information relatively unchanged to the cortex; and, a second system further processes these neural transmissions to extract information pertaining to sound location in space. In addition, a second duality is suggested (similar to that of Coats and Martin, 1977) by the different phase function for peak-to-trough amplitude data and the lack of any phase latency effects (when comparing raw latency with derived latency). This is based on the hypothesis that if the zero baseline amplitude phase effects were peak/trough specific, no difference should have occurred between zero baseline amplitude and peak-to-trough amplitude functions, and the latency measures should have tended toward shorter latency for waves

showing greater amplitudes (a known BER phenomenon). The problem is that the amplitude differences may have been too small to result in a significant change in latency. However, the implication is that one system results in the fast peak and trough activity, while the second results in a slow wave component that algebraically summates with the fast activity to produce the BER. The two are not likely to be mutually exclusive; since the summating properties of the averaging technique does not allow the separation of the two different coincidental evoked potentials, this is still an open question. At odds with the suggestion for the BI/MON conditions, the T3/P4 differences are significant for the phase conditions. This again suggests either overlapping contributions to the BER peaks and troughs, or the different contribution of subunits within specific nuclei.

The zero baseline amplitude phase effects, in general, show a left/right difference in the coding of the specific rarefaction and condensation stimuli used -- a totally unexpected and intriguing finding. The function is such that a significant R/C difference for the right ear with the left ear held constant is shown as a relative increase in peak/trough positivity for R. For the left ear, with the right ear held constant, this same difference results in a non-significant trend in the opposite direction. Figures 37 and 38 (the functions for VM and VN plotted spearately) indicate the same

FIGURE 37
ZERO BASELINE AMPLITUDES - VM

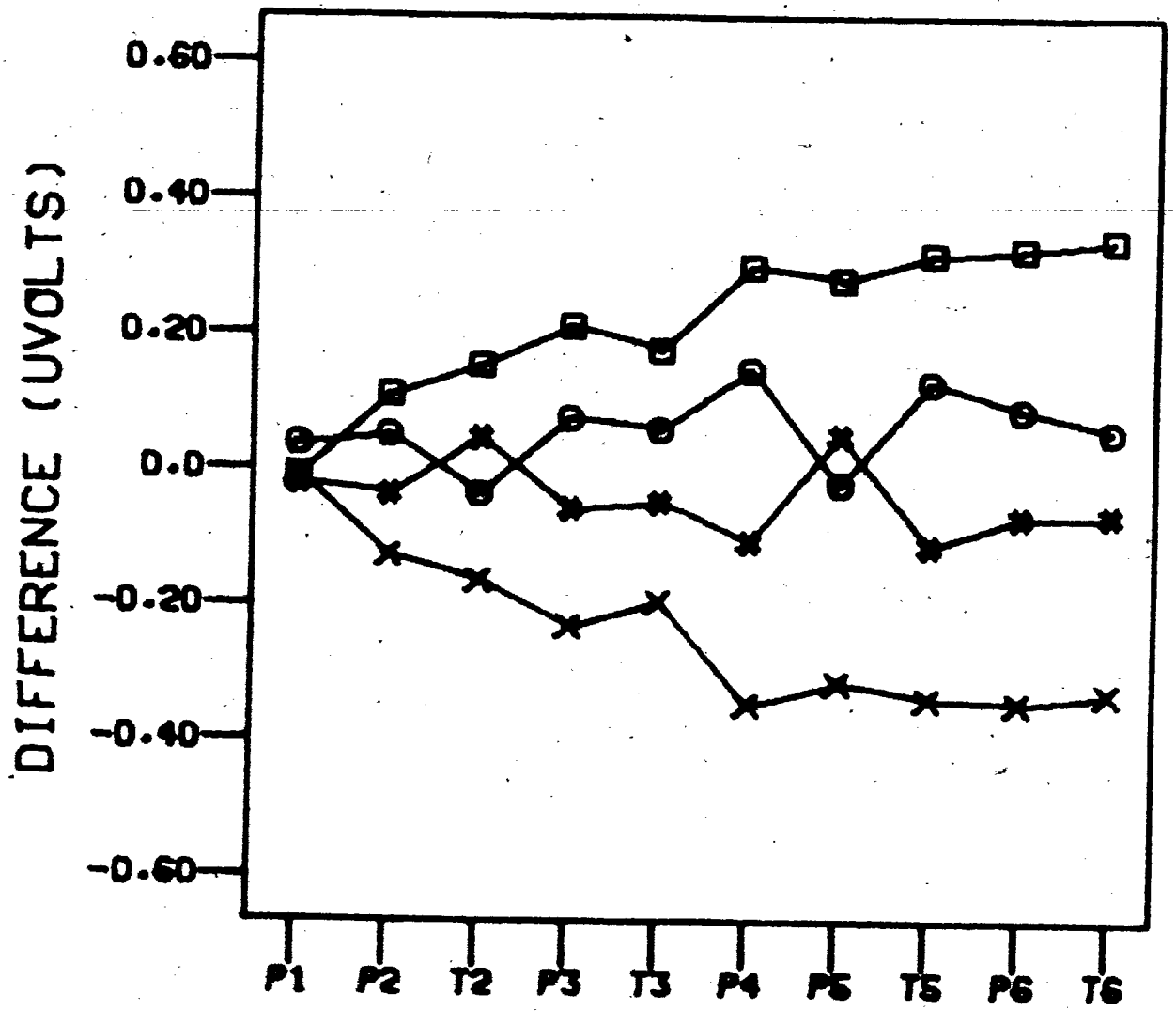
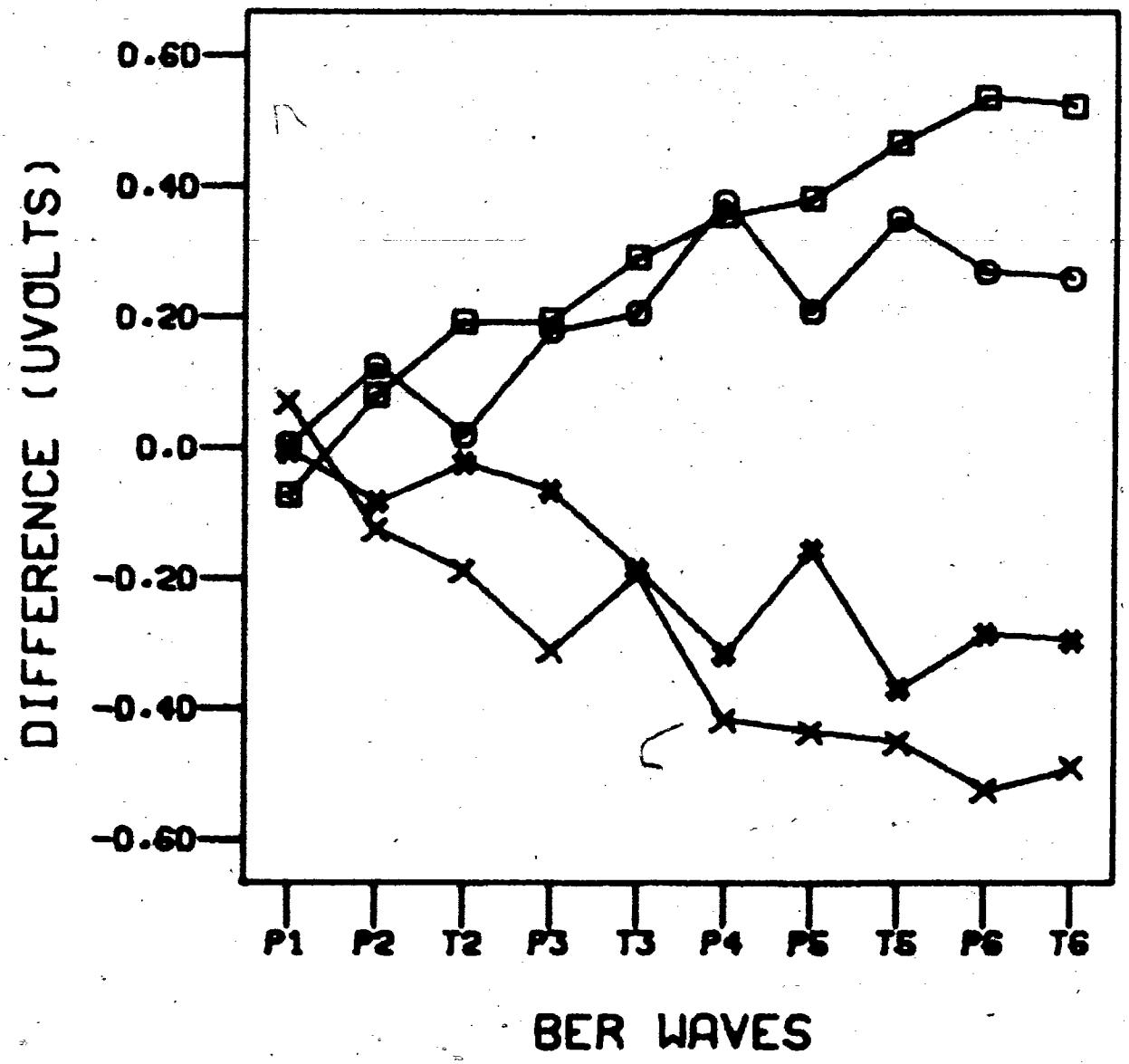


FIGURE 38
ZERO BASELINE AMPLITUDES - VN



general trend for both derivations, with VN showing a larger difference than VM, similar to that discussed above. What this implies (necessarily tentative until supported by replication) is that a basic difference exists in the coding and transmission of auditory signals at initial CAS nuclei, or possibly the cochlea, between the right and left ears. One must be careful not to extrapolate beyond the data since the differences are small. It is also important to consider why they have been shown in this study and not others. The possible reasons are three-fold: a) this is the first study that set out to investigate the binaural properties of R and C stimuli. All past studies have either used monaural stimulation, or a comparison between monaural and binaural stimulation; b) amplitude determination of the peaks is a very heterogeneous process across studies (as discussed in the introduction). The author is not presently aware of any other study that used T1 as an arbitrary zero voltage point (Picton and Hillyard, 1974, used a midpoint between T1 and T2 but did not test for right/left differences). Although it is difficult to see how this would result in the present finding, further experimentation comparing different amplitude determining procedures is indicated; and, c) this study uses a unique method to plot minor amplitude differences across peaks and troughs, resulting in a waveform in which the peaks and troughs can be easily compared for relative amplitude changes.

It is difficult to speculate on the meaning of these differences since anatomical, neurochemical and depth electrophysical research have either assumed bilateral CAS equality or have simply not looked for differences. It is probable that all CNS bilateral systems are divergent at some level, however, findings that suggest a divergence in the same direction across subjects points to fundamental differences and requires further study. The function (if any) and cause of this right/left difference remains obscure.

The peak-to-trough amplitude functions show clearly (whether the right or left ear is held constant) that a change from an R to a C stimulus results in smaller relative amplitudes, similar to the zero baseline data. Again, this suggests a basic coding difference in at least P2, P3 and P4 generator sources. What is of interest is the approximate coincidence of all four conditions at T3/P3; it may be that this is evidence that the finding of SOC differential phase coding (Galambos et al., 1959) is also represented in the BER. One would have to assume that the pool of neurons in the two MSO nuclei result in binaural potential waveforms that tend to cancel when recorded at the vertex. In addition, a difference is shown between T2/P3 and P3/T3 measures, again giving support to the contention that BER troughs do represent auditory information processing.

The phase latency effects for raw latency do not show up for derived latency. This suggests, if one assumes that derived latencies represent only neural CAS processing (i.e., higher nuclei in the CAS), that these significances are a reflection of end organ or eighth nerve processing, and not additional information processing at these nuclei. In essence the lack of latency differences support the expectation of no difference between RC and CR. However, the lack of RR/CC differences would suggest that the latency effects described in the literature either are cancelled by binaural potential fields, or the particular sample of subjects did not fall into the population of individuals that show phase effects (Ornitz and Walter, 1975, 1976).

D. Delta I and Delta T

The Delta I and Delta T waveforms suggest a number of possibilities:

a) since Delta I did not result in observable latency differences between BI and MON, it is probable that the known latency shifts for decreases in stimulus intensity are bilaterally equal within the CAS. With the particular averaging procedure used, these would then cancel;
b) since Delta I did result overall in a more positive waveform for MON, with the specific peak-to-trough larger

differences for BI at P3/T3, P4/T3 and P5/T5, it is possible that i) the slow wave component reported in the literature is more a function of monaural than of binaural stimulation and ii) the finding (and expectation) of lowered amplitudes for binaurally active nuclei (as they are represented in the literature) does not hold when the stimulus intensities are not equal. A possible hypothesis is that, if we assume monaural clicks of 65 dB result in the activation of more than half of a common left/right neuronal population, it is clear that the algebraic summation of two evoked monaural BER waves will result in amplitudes in excess of that evoked by binaural stimulation. If the parameters are then changed so one of the clicks has an intensity considerably below the other such that the algebraic summation of the two results in a BER amplitude less than the maximum possible, it is probable that binaural stimulation with these different intensities will result in greater BER amplitudes. This would occur simply because a greater number of coincident impulses would impinge on specific nuclei during binaural stimulation, thus resulting in greater numbers of post-synaptic neurons reaching threshold;

c) since Delta T resulted in decreased amplitudes at P3/T3 and P5/T5 for BI, additional support is given to the original hypothesis, that the generators of these potentials (i.e., SOC and IC) are involved in binaural processing.

These two paradigms should be prime candidates for future research.

SUMMARY

This study set out to determine the extent of CAS binaural processing representation in the BER. Clear evidence for binaural coding at an early and a later BER generator source was shown. In addition, support was provided for the following:

a) a reiteration of the BER active nature of mastoid "reference" sites;

b) the mimicking of dipole configurations by P2;

c) a larger overall vertex/non-cephalic waveform, as compared with a vertex/mastoid waveform, suggesting that all BER waves are homophasic at the vertex and mastoid;

d) active CAS processing represented in some BER troughs;

e) BER representation of a dual CAS information processing system -- one involving peripheral coding, and a second involving central decoding and encoding for localization;

f) phase-coding as an end-organ process;

g) a right/left difference in the coding and transmission of information concerning phase;

h) possibility of bilaterally equal BER intensity/latency functions; and,

i) possibility that the BER "slow wave" is a monaural phenomenon.

One final note: the most pressing need in BER research at present is a comprehensive study of the effects of all combinations of stimulus and recording parameters. Without knowledge about the extent of alteration these produce in recorded BERs, comparisons across studies is difficult, and conclusions reached in specific research must be tentative at best. It is apparent that if one is to use the BER in clinical settings, comparative waveforms from normal and pathological groups must be developed within the particular setting and not from the literature.

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APPENDIX A

DIRECTIONS FOR PEAK AND TROUGH DETERMINATION

The following is the procedure used by the author and the raters for determining peaks and troughs. An average waveform (composite) of all subjects across all conditions, and an average waveform of each subject across all conditions, were used. It is interesting to note that five out of the six raters failed to use (c) under "Procedure to be used" below.

Your task will be to determine the six peaks (P) and five troughs (T) as illustrated on your master composite. The task requires two steps: a) to determine the peaks and troughs on the subject composites from the master composite; and, b) to determine the peaks and troughs on the raw waveforms using the appropriate subject composite.

Whenever you cannot determine the peak or trough after comparing the subject composite or the other waveforms from the particular subject, refer to the rules listed below. Once you have determined the point at which a peak or trough occurs, draw a fine vertical line through that point. Occationally neither the composite nor referring to other waveforms will give you the clue you are looking for. In these cases, use the rules and search for any change in the waveform that is MOST CONSISTENT with the composite and other waveforms.

Procedure to be used:

a) form a gestalt -- that is, get a feeling for where the peaks and troughs are for the two subject waveforms.

b) make a gross determination of where you think the peak or trough is and draw an arrow to that peak or trough.

c) go through the waveforms with the appropriate composite to ensure that the peak or trough picked is correct and consistent.

d) check a final time, this time making a fine vertical line though the point where you think the peak or trough is.

When you make the final determination of where the line should be drawn, it is important to pay strict attention to the rules, such as whether the leading or following corner of a peak or trough should be chosen.

For changes in slope, use a ruler to determine the most horizontal segment. Draw a fine line along that segment and, referring to the rules, pick the most leading or following point (see Figure 39, a).

Definition: a change of slope is considered a change in the descending or ascending limbs of the waveform that approximate a peak or trough but does not actually form one (see Figure 39, b).

RULES

1) If a peak is more like a plateau than a point:
   a) Peak One (P1) -- if one corner is higher than the other, choose the corner most consistent with the subject composite. If the composite appears midway between the corners and reference to other waveforms by this subject does not delineate a particular corner, choose the corner closest to trough one (see Figure 39, c).
   b) Peaks Two, Three and Four (P2, P3, and P4) -- choose the highest level immediately following the preceeding trough (see Figure 39, d).
   c) Peaks Five and Six (P5 and P6) -- choose the highest level immediately preceeding the following trough (see Figure 39, e).
2) If a peak has a rounded, rather than a sharp point: -- choose the highest point closest to the appropriate trough.
3) If a trough is more like a plateau than a point:
   a) Trough Five (T5) -- choose the lowest point closest to P5
   b) Trough Six (T6) -- choose the lowest point closest to P6 (see Figure 39, f).
   c) all other troughs -- choose the lowest point closest to the preceeding peak.

4) Peak One (P1) -- if a peak appears more than 200 usec from the subject composite, and there is a change of slope that is more consistent with the composite, choose the change of slope (see Figure 39, g).

5) Whenever a change of slope occurs on a descending or ascending limb, rather than a distinct peak or trough:

   a) for ascending changes of slope (peaks) -- the peak is considered the first point at which the slope is closest to a horizontal position (see Figure 39, h).

   b) for ascending changes of slope (troughs) -- the trough is considered the last point at which the slope is closest to a horizontal position (see Figure 39, i).

   c) for descending changes of slope (peaks) -- the peak is considered tha last point at which the slope is closest to a horizontal position (see Figure 39, j).

   d) for descending changes of slope (troughs) -- the trough is considered the first point at which the slope is closest to a horizontal position (see Figure 39, k).

6) Whenever three peaks (or peaks and changes of slope) occur in the P4-P5 range:

   a) if only one peak appears in this range but a change of slope occurs on both sides (and these are consistent with the subject composite), choose the changes of slope and disregard the peak (see Figure 39, l).

b) if two peaks and an ascending change of slope occurs, if the change of slope is more consistent with P4 of the subject composite, choose the change of slope as P4 and disregard the following peak

c) if three equal peaks occur and the first has been determined P4, the middle peak and related troughs will be disregarded (see Figure 39, m).

7) If a change of slope occurs after P5 and after the descending limb has reached the level of T3 or lower, the change of slope will be considered T5 (see Figure 39, n).

8) If two or more peaks occur between T5 and T6, the peak immediately preceeding T6 will be considered P6 (see Figure 39, o).

9) If P6 approximates a plateau but with one corner slightly higher than the other, choose the corner closest to T6 (see Figure 39, p).

10) If the descending limb immediately preceeding T6 has a clearly discernable change of slope, this change will be considered P6 (see Figure 39, q).

As a guide, use the following schematic to help determine whether the leading or following corner of a peak or trough should be chosen (see Figure 39, r).

FIGURE 39
ILLUSTRATIONS FOR
PEAK DETERMINATING PROCEDURE

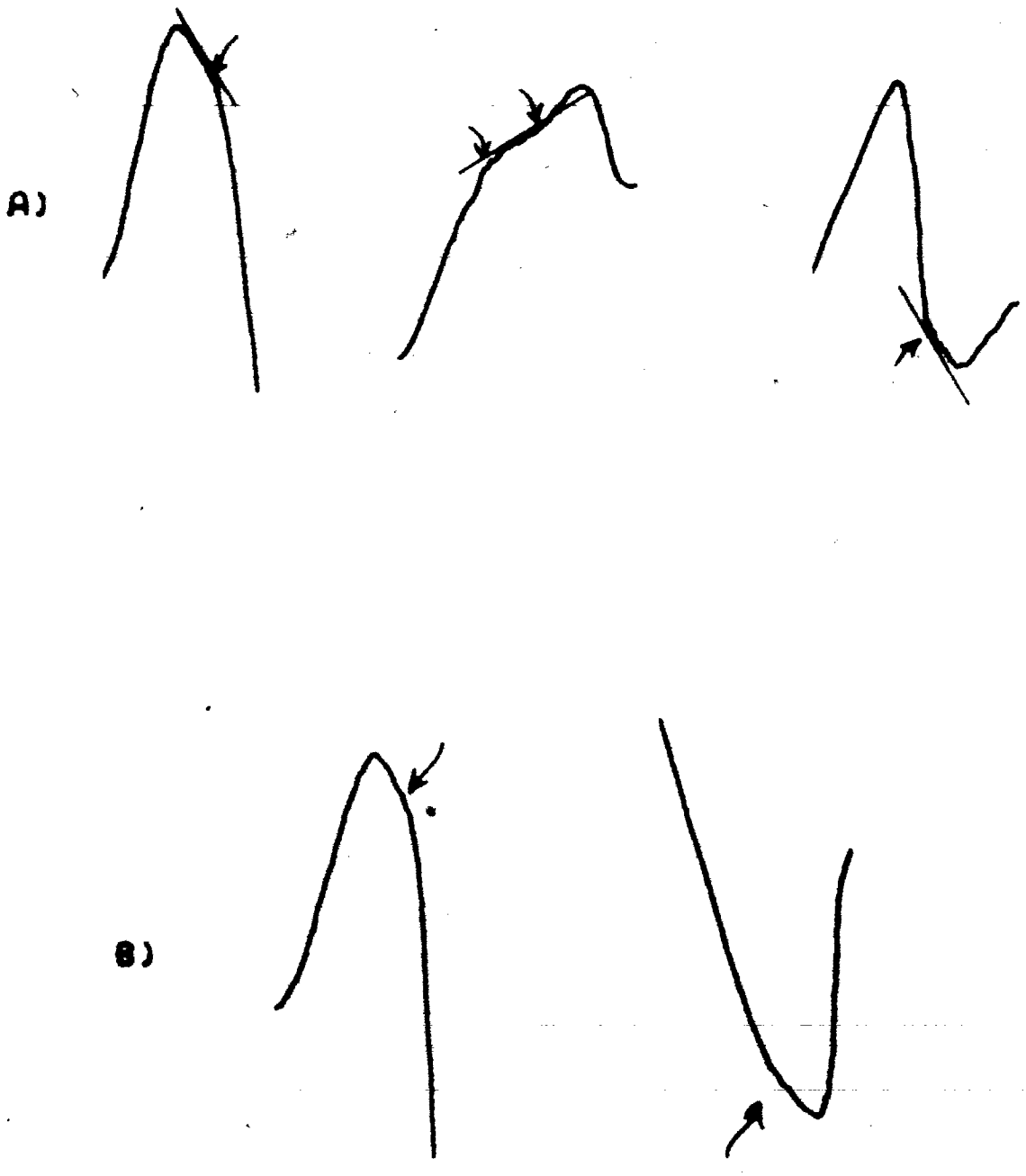
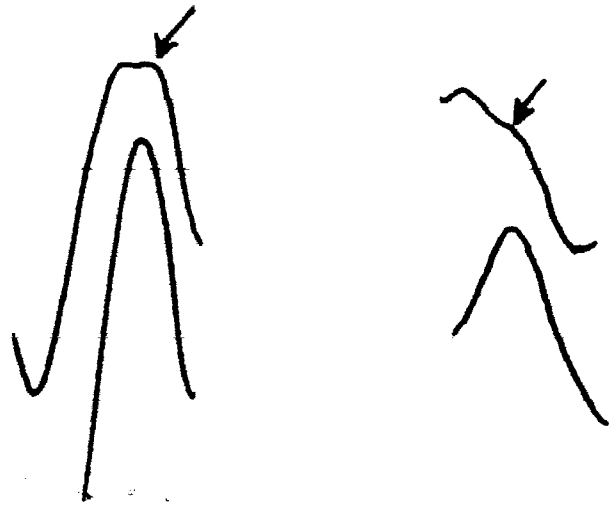


FIGURE 39 CONTINUED

C)



D)

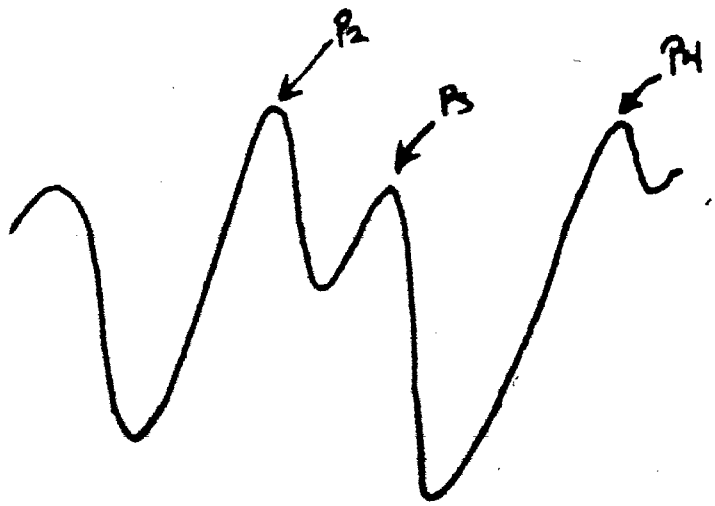
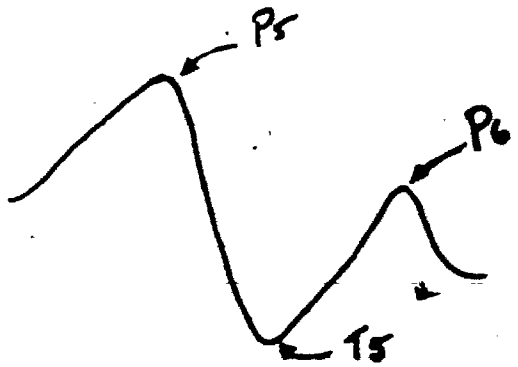


FIGURE 39 CONTINUED

E)



F)

G)

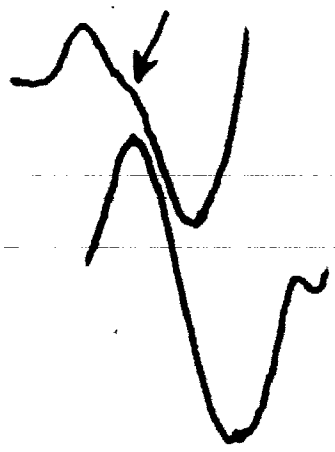
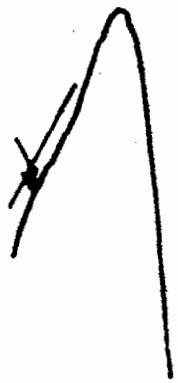


FIGURE 39 CONTINUED

H)



I)

J)

K)

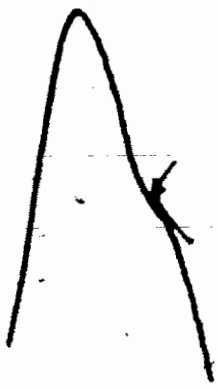


FIGURE 39 CONTINUED

L)

M)

N)

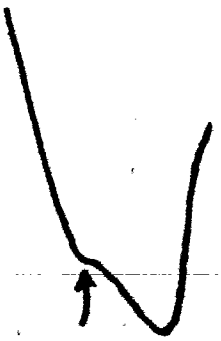
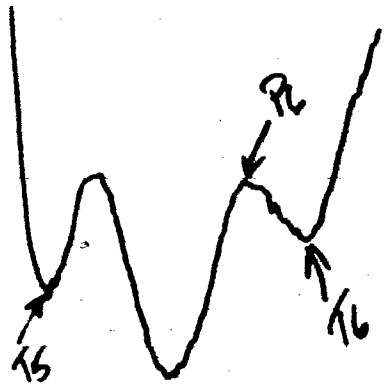


FIGURE 39 CONTINUED

O)



P)

Q)

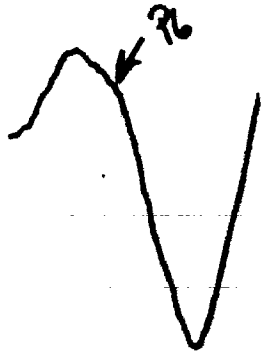
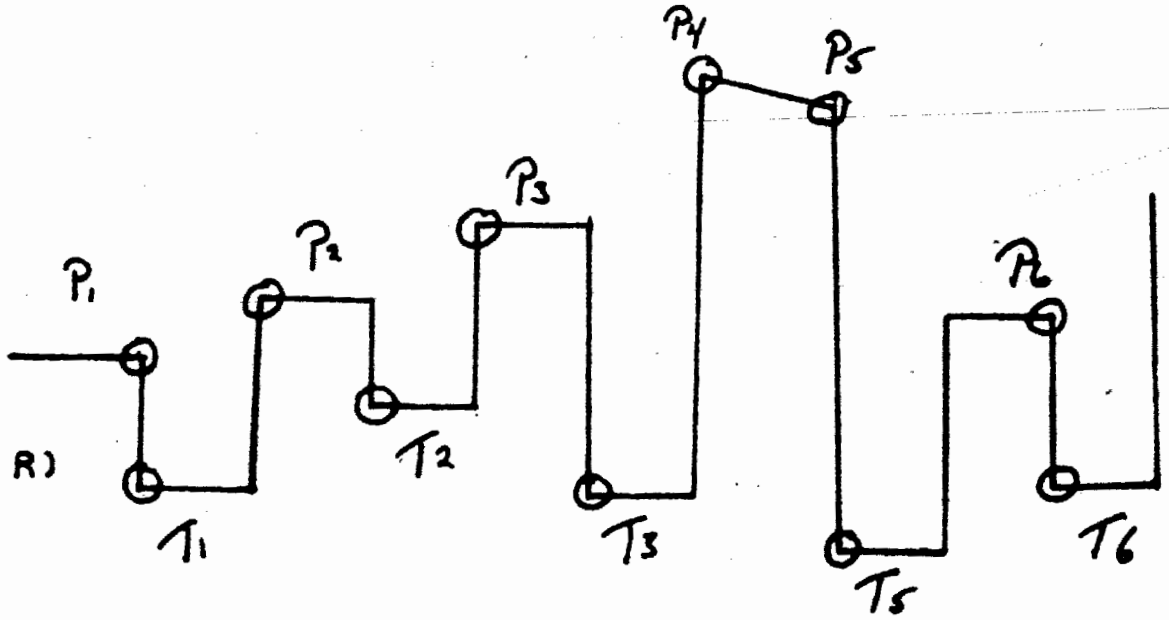


FIGURE 39 CONTINUED



APPENDIX B

ANOVA TABLES FOR RAW LATENCY DATA

For all following tables:
A = DERIVATION
B = BI/MON
C = PHASE

P1 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.05	0.07	0.788
Error	19	0.66		
B	1	1.65	7.14	0.015
Error	19	0.23		
AxB	1	0.01	0.01	0.928
Error	19	0.37		
C	3	0.14	0.37	0.776
Error	57	0.39		
AxC	3	0.62	2.93	0.041
Error	57	0.21		
BxC	3	0.11	0.31	0.815
Error	57	0.36		
AxBxC	3	0.13	0.92	0.439
Error	57	0.27		

T1 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	2.77	4.23	0.054
Error	19	0.65		
B	1	0.88	1.92	0.182
Error	19	0.46		
AxB	1	0.06	0.17	0.683
Error	19	0.33		
C	3	3.18	4.15	0.010
Error	57	0.77		
AxC	3	0.23	1.28	2.921
Error	57	0.18		
BxC	3	0.28	1.01	0.394
Error	57	0.28		
AxBxC	3	0.13	0.85	0.471
Error	57	0.15		

P2 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	11.44	44.30	< 0.001
Error	19	0.26		
B	1	0.31	0.69	0.417
Error	19	0.45		
AxB	1	0.49	1.73	0.204
Error	19	0.28		
C	3	1.07	2.00	0.125
Error	57	0.54		
AxC	3	0.10	0.83	0.484
Error	57	0.12		
BxC	3	0.26	2.31	0.086
Error	57	0.11		
AxBxC	3	0.36	0.47	0.702
Error	57	0.12		

T2 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	12.11	43.09	< 0.001
Error	19	0.28		
B	1	0.03	0.06	0.814
Error	19	0.58		
AxB	1	1.21	6.31	0.021
Error	19	0.19		
C	3	0.76	0.65	0.588
Error	57	1.17		
AxC	3	0.18	1.30	0.284
Error	57	0.14		
BxC	3	0.34	1.11	0.352
Error	57	0.31		
AxBxC	3	0.04	0.17	0.916
Error	57	0.21		

P3 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	3.10	21.18	< 0.000
Error	19	0.13		
B	1	0.53	0.70	0.414
Error	19	0.76		
AxB	1	0.20	1.95	0.179
Error	19	0.10		
C	3	0.85	1.74	0.170
Error	57	0.49		
AxC	3	0.03	0.30	0.826
Error	57	0.10		
BxC	3	0.53	2.48	0.070
Error	57	0.21		
AxBxC	3	0.22	2.42	0.075
Error	57	0.09		

T3 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	1.91	4.55	0.046
Error	19	0.42		
B	1	0.24	0.73	0.403
Error	19	0.33		
AxB	1	0.40	1.15	0.298
Error	19	0.34		
C	3	1.64	1.67	0.184
Error	57	0.98		
AxC	3	0.31	1.69	0.178
Error	57	0.18		
BxC	3	0.75	2.22	0.096
Error	57	0.34		
AxBxC	3	0.21	1.31	0.279
Error	57	0.16		

P4 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	1.25	4.89	0.041
Error	17	0.26		
B	1	0.42	0.65	0.432
Error	17	0.65		
AxB	1	0.07	0.72	0.408
Error	17	0.10		
C	3	1.58	3.50	0.022
Error	51	0.45		
AxC	3	0.46	4.15	0.010
Error	51	0.11		
BxC	3	0.08	0.33	0.803
Error	51	0.24		
AxBxC	3	0.02	0.15	0.927
Error	51	0.13		

P5 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.01	0.02	0.896
Error	18	0.30		
B	1	8.47	12.03	0.003
Error	18	0.70		
AxB	1	0.07	0.33	0.575
Error	18	0.23		
C	3	0.31	0.84	0.479
Error	54	0.37		
AxC	3	0.01	0.09	0.964
Error	54	0.11		
BxC	3	0.02	0.11	0.956
Error	54	0.18		
AxBxC	3	0.42	3.21	0.030
Error	54	0.13		

T5 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	3.25	11.75	0.003
Error	19	0.28		
B	1	16.09	14.29	0.001
Error	19	1.13		
AxB	1	0.01	0.02	0.887
Error	19	0.46		
C	3	2.48	4.17	0.010
Error	57	0.60		
AxC	3	0.05	0.43	0.731
Error	57	0.12		
BxC	3	0.29	0.90	0.448
Error	57	0.32		
AxBxC	3	0.12	1.05	0.379
Error	57	0.11		

P6 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.05	0.17	0.684
Error	18	0.27		
B	1	6.15	3.30	0.086
Error	18	1.87		
AxB	1	0.15	0.48	0.496
Error	18	0.31		
C	3	4.62	6.19	0.001
Error	54	0.75		
AxC	3	0.06	0.21	0.888
Error	54	0.30		
BxC	3	0.41	0.82	0.486
Error	54	0.49		
AxBxC	3	0.18	0.79	0.506
Error	54	0.23		

T6 RAW LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.00	0.01	0.928
Error	18	0.39		
B	1	0.01	0.01	0.918
Error	18	0.68		
AxB	1	0.84	1.72	0.207
Error	18	0.49		
C	3	1.96	1.54	0.214
Error	54	1.27		
AxC	3	0.05	0.16	0.925
Error	54	0.30		
BxC	3	0.26	0.39	0.760
Error	54	0.68		
AxBxC	3	0.10	0.33	0.805
Error	54	0.30		

APPENDIX C

ANOVA TABLES FOR DERIVED LATENCY DATA

For all following tables:
A = DERIVATION
B = BI/MON
C = PHASE

P2 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	2.95	3.57	0.074
Error	19	0.83		
B	1	0.14	0.29	0.598
Error	19	0.50		
AxB	1	0.88	1.42	0.247
Error	19	0.62		
C	3	2.13	2.33	0.084
Error	57	0.92		
AxC	3	0.53	2.07	0.114
Error	57	0.25		
BxC	3	0.25	0.78	0.511
Error	57	0.32		
AxBxC	3	0.29	1.40	0.252
Error	57	0.21		

T2 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	3.30	3.91	0.063
Error	19	0.84		
B	1	1.25	2.78	0.112
Error	19	0.45		
AxB	1	0.75	1.71	0.207
Error	19	0.44		
C	3	1.47	2.02	0.121
Error	57	0.73		
AxC	3	0.00	0.01	0.998
Error	57	0.31		
BxC	3	0.02	0.03	0.991
Error	57	0.47		
AxBxC	3	0.10	0.29	0.833
Error	57	0.34		

P3 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.01	0.02	0.897
Error	19	0.56		
B	1	2.77	4.39	0.050
Error	19	0.63		
AxB	1	0.04	0.11	0.743
Error	19	0.40		
C	3	6.96	8.78	< 0.000
Error	57	0.79		
AxC	3	0.36	1.33	0.274
Error	57	0.27		
BxC	3	0.33	0.82	0.489
Error	57	0.40		
AxBxC	3	0.59	2.95	0.040
Error	57	0.20		

T3. DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.08	0.04	0.835
Error	19	1.75		
B	1	0.20	0.41	0.530
Error	19	0.49		
AxB	1	0.75	0.85	0.367
Error	19	0.88		
C	3	0.53	0.84	0.477
Error	57	0.63		
AxC	3	0.64	2.18	0.101
Error	57	0.29		
BxC	3	0.13	0.30	0.829
Error	57	0.43		
AxBxC	3	0.49	1361	0.264
Error	57	0.36		

P4 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.40	0.37	0.549
Error	17	1.07		
B	1	3.17	3.55	0.077
Error	17	0.90		
AxB	1	0.21	0.61	0.445
Error	17	0.34		
C	3	2.34	1.84	0.152
Error	51	1.27		
AxC	3	0.87	2.65	0.059
Error	51	0.33		
BxC	3	0.43	1.47	0.233
Error	51	0.29		
AxBxC	3	0.18	0.54	0.657
Error	51	0.34		

P5 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	3.26	7.20	0.015
Error	18	0.45		
B	1	15.43	17.11	0.001
Error	18	0.90		
AxB	1	0.01	0.01	0.910
Error	18	0.56		
C	3	3.42	2.69	0.055
Error	54	1.27		
AxC	3	0.17	0.74	0.533
Error	54	0.25		
BxC	3	0.18	0.39	0.759
Error	54	0.47		
AxBxC	3	0.67	2.07	0.115
Error	54	0.57		

T5 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	0.02	0.03	0.861
Error	19	0.62		
B	1	24.48	13.21	0.002
Error	19	1.85		
AxB	1	0.02	0.03	0.863
Error	19	0.64		
C	3	0.75	0.60	0.618
Error	57	1.25		
AxC	3	0.36	1.22	0.310
Error	57	0.29		
BxC	3	1.10	1.73	0.172
Error	57	0.63		
AxBxC	3	0.49	1.82	0.154
Error	57	0.27		

P6 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	4.20	5.04	0.038
Error	18	0.83		
B	1	12.34	5.23	0.035
Error	18	2.36		
AxB	1	0.92	1.66	0.213
Error	18	0.55		
C	3	2.72	1.60	0.201
Error	54	1.70		
AxC	3	0.27	0.64	0.590
Error	54	0.42		
BxC	3	1.12	1.38	0.260
Error	54	0.82		
AxBxC	3	0.16	0.93	0.433
Error	54	0.17		

T6 DERIVED LATENCIES

Source	df	Mean Square	F	Probability
A	1	3.58	4.60	0.0446
Error	18	0.78		
B	1	1.25	0.97	0.338
Error	18	1.29		
AxB	1	2.22	2.65	0.121
Error	18	0.84		
C	3	4.67	1.97	0.129
Error	54	2.37		
AxC	3	0.12	0.28	0.837
Error	54	0.41		
BxC	3	0.03	0.04	0.991
Error	54	0.80		
AxBxC	3	0.44	1.41	0.250
Error	54	0.31		

APPENDIX D

ANOVA TABLES FOR ZERO BASELINE DATA

For all following tables:
A = DERIVATION
B = BI/MON
C = PHASE

P1 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	72.20	2.40	0.138
Error	19	30.09		
B	1	88.20	7.75	0.012
Error	19	11.37		
AxB	1	13.20	0.90	0.354
Error	19	14.63		
C	3	51.38	1.31	0.279
Error	57	39.11		
AxC	3	47.30	4.85	0.005
Error	57	9.76		
BxC	3	30.90	1.58	0.204
Error	57	19.53		
AxBxC	3	5.11	0.77	0.516
Error	57	6.64		

P2 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	3028.87	67.49	< 0.001
Error	19	44.88		
B	1	620.22	7.53	0.013
Error	19	82.36		
AxB	1	10.69	0.36	0.557
Error	19	29.93		
C	3	593.60	8.43	< 0.001
Error	57	70.39		
AxC	3	32.82	1.38	0.259
Error	57	23.84		
BxC	3	25.61	1.37	0.262
Error	57	18.72		
AxBxC	3	15.41	0.95	0.420
Error	57	16.14		

T2 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	1597.58	28.36	< 0.001
Error	19	56.32		
B	1	189.11	3.40	0.081
Error	19	55.63		
AxB	1	4.05	0.14	0.710
Error	19	28.47		
C	3	1023.11	6.47	0.001
Error	57	151.72		
AxC	3	42.05	1.12	0.348
Error	57	37.51		
BxC	3	17.98	0.67	0.573
Error	57	26.76		
AxBxC	3	20.99	1.65	0.189
Error	57	12.76		

P3 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	2743.65	44.57	< 0.001
Error	19	61.56		
B	1	613.27	6.33	0.021
Error	19	96.86		
AxB	1	23.65	0.62	0.442
Error	19	38.42		
C	3	2569.31	7.97	< 0.001
Error	57	322.56		
AxC	3	209.59	3.35	0.025
Error	57	62.50		
BxC	3	42.59	1.29	0.285
Error	57	32.90		
AxBxC	3	63.87	2.67	0.056
Error	57	23.92		

T3 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	507.53	9.23	0.007
Error	19	54.97		
B	1	332.11	4.34	0.051
Error	19	76.52		
AxB	1	27.61	0.70	0.414
Error	19	39.57		
C	1	2491.62	6.18	0.001
Error	57	403.25		
AxC	3	286.88	3.39	0.024
Error	57	84.54		
BxC	3	84.81	1.63	0.193
Error	57	52.12		
AxBxC	3	50.89	3.49	0.021
Error	57	14.60		

P4 - ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	4012.56	37.28	< 0.001
Error	17	107.63		
B	1	1192.35	12.48	0.003
Error	17	95.52		
AxB	1	108.78	3.68	0.072
Error	17	29.54		
C	3	5489.61	9.72	< 0.001
Error	51	564.52		
AxC	3	395.70	3.79	0.016
Error	51	104.43		
BxC	3	182.21	4.01	0.012
Error	51	45.41		
AxBxC	3	74.75	4.26	0.009
Error	51	17.56		

P5 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	3009.50	78.30	< 0.001
Error	18	38.44		
B	1	1443.77	11.55	0.003
Error	18	125.01		
AxB	1	151.35	5.13	0.036
Error	18	29.52		
C	3	4098.69	8.08	< 0.001
Error	54	507.35		
AxC	3	413.90	3.67	0.018
Error	54	112.74		
BxC	3	20.03	0.54	0.659
Error	54	37.29		
AxBxC	3	49.87	2.37	0.0881
Error	54	21.06		

T5 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	3292.82	37.78	< 0.001
Error	19	87.16		
B	1	131.97	0.75	0.397
Error	19	175.65		
AxB	1	11.44	0.27	0.612
Error	19	42.97		
C	3	7027.93	9.27	< 0.001
Error	57	757.81		
AxC	3	602.04	4.53	0.006
Error	57	132.97		
BxC	3	36.99	0.83	0.485
Error	57	44.82		
AxBxC	3	24.02	1.22	0.309
Error	57	19.61		

P6 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	351.09	11.10	0.004
Error	18	31.62		
B	1	11.18	0.06	0.812
Error	18	190.89		
AxB	1	18.16	0.47	0.501
Error	18	38.44		
C	3	6931.04	10.35	< 0.001
Error	54	669.47		
AxC	3	618.65	4.72	0.005
Error	54	130.98		
BxC	3	321.14	4.10	0.011
Error	54	78.41		
AxBxC	3	24.17	0.99	0.403
Error	54	24.34		

T6 ZERO BASELINE AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	934.50	23.08	< 0.001
Error	18	40.49		
B	1	423.95	2.64	0.122
Error	18	160.85		
AxB	1	70.12	1.86	0.189
Error	18	37.68		
C	3	6512.44	11.50	< 0.001
Error	54	566.49		
AxC	3	609.24	4.61	0.006
Error	54	132.17		
BxC	3	47.49	1.10	0.359
Error	54	43.31		
AxBxC	3	29.96	0.55	0.651
Error	54	18.21		

APPENDIX E

ANOVA TABLES FOR PEAK-TO-TROUGH DATA

For all following tables:
A = DERIVATION
B = BI/MON
C = PHASE

T1 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	68.45	2.30	0.146
Error	19	29.78		
B	1	92.45	8.35	0.009
Error	19	11.07		
AxB	1	11.63	0.81	0.3879
Error	19	14.32		
C	3	50.61	1.32	0.276
Error	57	38.23		
AxC	3	46.89	4.76	0.005
Error	57	9.84		
BxC	3	28.91	1.49	0.226
Error	57	19.35		
AxBxC	3	4.87	0.71	0.552
Error	57	6.89		

P2 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	3053.54	68.23	< 0.001
Error	19	44.11		
B	1	609.13	7.56	0.013
Error	19	80.62		
AxB	1	9.28	0.33	0.578
Error	19	28.43		
C	3	599.82	8.51	< 0.001
Error	57	70.45		
AxC	3	32.13	1.41	0.249
Error	57	22.79		
BxC	3	25.81	1.42	0.246
Error	57	18.15		
AxBxC	3	15.42	1.02	0.389
Error	57	15.05		

T2 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	397.16	19.00	< 0.001
Error	19	20.91		
B	1	43.88	2.32	0.144
Error	19	18.90		
AxB	1	13.81	1.48	0.238
Error	19	9.31		
C	3	293.16	5.06	0.004
Error	57	57.90		
AxC	3	0.32	0.02	0.996
Error	57	15.12		
BxC	3	49.42	2.57	0.063
Error	57	19.23		
AxBxC	3	6.78	0.60	0.618
Error	57	11.31		

P3 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	257.40	15.29	0.001
Error	19	16.83		
B	1	56.11	3.34	0.084
Error	19	16.81		
AxB	1	2.11	0.44	0.514
Error	19	4.77		
C	3	755.50	12.63	< 0.001
Error	57	59.82		
AxC	3	38.13	2.95	0.040
Error	57	12.93		
BxC	3	42.13	2.39	0.0678
Error	57	17.60		
AxBxC	3	17.03	2.14	0.105
Error	57	7.97		

T3 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	871.20	27.03	< 0.001
Error	19	32.23		
B	1	23.65	0.49	0.494
Error	19	48.66		
AxB	1	0.00	0.00	0.991
Error	19	20.29		
C	3	18.24	0.28	0.842
Error	57	66.08		
AxC	3	63.36	4.34	0.008
Error	57	14.61		
BxC	3	29.84	0.91	0.440
Error	57	32.65		
AxBxC	3	19.83	2.41	0.0676
Error	57	8.22		

P4 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	1820.06	37.10	< 0.001
Error	17	49.05		
B	1	262.59	6.57	0.020
Error	17	39.95		
AxB	1	49.17	1.64	0.218
Error	17	30.06		
C	3	1099.90	8.55	< 0.001
Error	51	128.59		
AxC	3	77.79	5.06	0.004
Error	51	15.36		
BxC	3	39.75	1.51	0.223
Error	51	26.33		
AxBxC	3	15.48	1.92	0.138
Error	51	8.05		

T5 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	12546.72	100.58	< 0.001
Error	18	124.74		
B	1	941.53	10.55	0.004
Error	18	89.25		
AxB	1	147.84	5.26	0.034
Error	18	28.11		
C	3	641.81	4.71	0.005
Error	54	134.63		
AxC	3	12.16	0.82	0.489
Error	54	14.85		
BxC	3	11.08	0.36	0.783
Error	54	30.93		
AxBxC	3	33.07	2.26	0.091
Error	54	14.60		

P6 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	1802.78	38.54	< 0.001
Error	18	46.78		
B	1	4.93	0.04	0.836
Error	18	111.86		
AxB	1	19.15	0.60	0.449
Error	18	31.98		
C	3	151.97	1.29	0.288
Error	54	118.00		
AxC	3	52.67	4.97	0.004
Error	54	10.59		
BxC	3	406.25	8.28	< 0.001
Error	54	49.09		
AxBxC	3	10.91	0.66	0.581
Error	54	16.54		

T6 PEAK-TO-TROUGH AMPLITUDES

Source	df	Mean Square	F	Probability
A	1	162.56	7.90	0.012
Error	18	20.57		
B	1	210.06	6.00	0.025
Error	18	35.02		
AxB	1	140.00	23.15	< 0.001
Error	18	6.05		
C	3	14.91	0.25	0.863
Error	54	60.30		
AxC	3	5.54	0.78	0.509
Error	54	7.07		
BxC	3	102.33	3.04	0.037
Error	54	33.71		
AxBxC	3	5.18	1.04	0.380
Error	54	4.96		

APPENDIX F

NEWMAN-KEULS STUDENTIZED TANGE TESTS FOR ZERO BASELINE DATA

For the following tables:
RR = Rarefaction right; Rarefaction left
RC = Rarefaction right; Condensation left
CR = Condensation right; Rarefaction left
CC = Consensation right; Condensation left

PEAK TWO ZERO BASELINE PHASE DATA

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 8796.48	RR = 9092.00	RC = 9097.04	Shortest Significant Ranges
CR = 8662.00	134.48	430.00**	435.04**	R2 = 214.62
CC = 8796.48		295.52*	300.56*	R3 = 258.15
RR = 9092.00			5.04	R4 = 284.41

Note: df = 57; n per group = 80; MSe = 70.39

* = $p < .05$; ** = $p < .01$.

TROUGH TWO ZERO BASELINE PHASE DATA

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	RR = 8186.96	CC = 8228.48	RC = 8551.52	Shortest Significant Ranges
CR = 7852.00	334.96*	376.48*	699.52**	R2 = 315.09
RR = 8186.96		41.52	364.56	R3 = 378.99
CC = 8228.48			323.04	R4 = 417.55

Note: df = 57; n per group = 80; MSe = 151.72

* = $p < .05$; ** = $p < .01$.

PEAK THREE ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 8861.52	RR = 9439.52	RC = 9589.52	Shortest Significant Ranges
CR = 8642.48	219.04	797.04**	947.04**	R2 = 459.43
CC = 8861.52		578.00*	728.00**	R3 = 552.60
RR = 9439.52			150.00	R4 = 608.82

Note: df = 57; n per group = 80; MSe = 322.56

* = $p < .05$; ** = $p < .01$.

TROUGH THREE ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 7372.00	RR = 7869.04	RC = 8073.52	Shortest Significant Ranges
CR = 7100.48	271.52	768.56**	973.04**	R2 = 519.07
CC = 7372.00		497.04	701.52*	R3 = 617.86
RR = 7869.04			204.48	R4 = 680.72

Note: df = 57; n per group = 80; MSe = 403.25

* = $p < .05$; ** = $p < .01$.

PEAK FOUR ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 9073.92	RR = 10017.76	RC = 10151.36	Shortest Significant Ranges
CR = 8731.12	342.80	1286.64**	1420.24**	R2 = 607.82
CC = 9073.92		943.84*	1077.44**	R3 = 731.09
RR = 10017.76			133.60	R4 = 805.47

Note: df = 51; n per group = 80; MSe = 564.59

* = p < .05; ** = p < .01.

PEAK FIVE ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 9160.56	RR = 9412.64	RC = 9926.88	Shortest Significant Ranges
CR = 8513.12	647.44*	899.52**	1413.76**	R2 = 576.19
CC = 9160.56		252.08	766.32*	R3 = 693.04
RR = 9412.64			512.24	R4 = 763.55

Note: df = 54; n per group = 80; MSe = 507.35

* = $p < .05$; ** = $p < .01$.

TROUGH FIVE ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 5406.00	RR = 6352.48	RC = 6673.04	Shortest Significant Ranges
CR = 5099.04	306.96	1253.44**	1574.00**	R2 = 704.19
CC = 5406.00		946.48*	1267.04**	R3 = 847.00
RR = 6352.48			320.56	R4 = 933.18

Note: df = 57; n per group = 80; MSe = 757.81

* = $p < .05$; ** = $p < .01$.

PEAK SIX ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 7019.12	RR = 7732.64	RC = 8234.72	Shortest Significant Ranges
CR = 6504.72	514.40	1227.92**	1730.00**	R2 = 661.88
CC = 7019.12		713.52	1215.60**	R3 = 796.11
RR = 7732.64			502.08	R4 = 877.11

Note: df = 54; n per group = 80; MSe = 669.76

* = p < .05; ** = p < .01.

TROUGH SIX ZERO BASELINE AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 6226.32	RR = 6901.60	RC = 7445.76	Shortest Significant Ranges
CR = 5763.12	463.20	1138.48**	1682.64**	R2 = 608.85
CC = 6226.32		675.28	1219.44**	R3 = 732.32
RR = 6901.60			544.16	R4 = 806.83

Note: df = 54; n per group = 80; MSe = 566.49

* = $p < .05$; ** = $p < .01$.

APPENDIX G

NEWMAN-KEULS STUDENTIZED RANGE TESTS FOR PEAK-TO-TROUGH DATA

For the following tables:
RR = Rarefaction right; Rarefaction left
RC = Rarefaction right; Condensation left
CR = Condensation right; Rarefaction left
CC = Consensation right; Condensation left

PEAK TWO PEAK-TO-TROUGH AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 1596.80	RR = 1892.00	RC = 1900.80	Shortest Significant Ranges
CR = 1461.60	135.20	430.40**	439.20**	R2 = 258.25
CC = 1596.80		295.20*	304.00**	R3 = 258.25
RR = 1892.00			8.80	R4 = 284.53

Note: df = 57; n per group = 80; MSe = 70.45

* = $p < .05$; ** = $p < .01$.

PEAK THREE PEAK-TO-TROUGH AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 792.00	RC = 1038.40	RR = 1252.80	Shortest Significant Ranges
CR = 790.40	1.60	248.00*	462.40**	R2 = 197.85
CC = 792.00		246.40*	460.80**	R3 = 237.97
RC = 1038.40			214.40	R4 = 262.18

Note: df = 57; n per group = 80; MSe = 59.82

* = $p < .05$; ** = $p < .01$.

PEAK FOUR PEAK-TO-TROUGH AMPLITUDES

Newman-Keuls Studentized Range Test for PHASE groups.

Sums	CC = 1642.40	RC = 2080.00	RR = 2229.60	Shortest Significant Ranges
CR = 1606.40	36.00	473.60**	623.20**	R2 = 290.08
CC = 1642.40		437.60*	587.20**	R3 = 348.90
RC = 2080.00			146.60	R4 = 384.40

Note: df = 51; n per group = 80; MSe = 128.59

* = p < .05; ** = p < .01.

APPENDIX H

FREQUENCY HISTOGRAMS FOR RAW LATENCY AND ZERO BASELINE DATA

P1 LATENCIES

MSEC	5 10 15 20 25 30 35 40 +----+----+----+----+----+----+----+----+	FREQUENCY INT.	CUM.
1.3860	+	0	0
1.4040	+	0	0
1.4220	+	0	0
1.4400	+X	1	1
1.4580	+	0	1
1.4760	+X	1	2
1.4940	+	0	2
1.5120	+X	1	3
1.5300	+	0	3
1.5480	+XX	2	5
1.5660	+	0	5
1.5840	+XXXXXXXXX	9	14
1.6020	+XXXXXXXXXXXXXXX	15	29
1.6200	+	0	29
1.6380	+XXXXXXXXXX	10	39
1.6560	+	0	39
1.6740	+XXXXXXXXXXXXXXXXXXXXXXXXXXXX	28	67
1.6920	+	0	67
1.7100	+XXXXXXXXXXXXXXXXXX	18	85
1.7280	+	0	85
1.7460	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXX	29	114
1.7640	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	32	146
1.7820	+	0	146
1.8000	+XXXXXXXXXXXXXXXXXXX	19	165
1.8180	+	0	165
1.8360	+XXXXXXXXXXXXXXXXXXXXXXX	23	188
1.8540	+	0	188
1.8720	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX*	45	233
1.8900	+	0	233
1.9080	+XXXXXXXXXXXXXXXXXXXXXXXXXX	26	259
1.9260	+XXXXXXXXXXXX	12	271
1.9440	+	0	271
1.9620	+XXXXXXXXXXXXX	13	284
1.9800	+XXXXXXXXX	9	293
1.9980	+	0	293
2.0160	+	0	293
2.0340	+XXXXXXXXXXXXX	13	306
2.0520	+XXXXXXXX	8	314
2.0700	+	0	314
2.0880	+XXX	3	317
2.1060	+	0	317
2.1240	+X	1	318
2.1420	+X	1	319
2.1600	+	0	319
2.1780	+	0	319
2.1960	+X	1	320

T1 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
1.8900	+	0	0
1.9080	+	0	0
1.9260	+X	1	1
1.9440	+	0	1
1.9620	+X	1	2
1.9800	+X	1	3
1.9980	+	0	3
2.0160	+	0	3
2.0340	+	0	3
2.0520	+	0	3
2.0700	+	0	3
2.0880	+XXXX	4	7
2.1060	+	0	7
2.1240	+XXXXXXXXXX	10	17
2.1420	+XXXXXXXXXXXX	12	29
2.1600	+	0	29
2.1780	+	0	29
2.1960	+XXXXXXXXXXXXXXXXXXXXXX	23	52
2.2140	+XXXXXXXXXXXXXXXXXXXXXXXX	25	77
2.2320	+	0	77
2.2500	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	34	111
2.2680	+	0	111
2.2860	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	34	145
2.3040	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	38	183
2.3220	+	0	183
2.3400	+	0	183
2.3580	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	33	216
2.3760	+XXXXXXXXXXXXXXXXXXXX	22	238
2.3940	+	0	238
2.4120	+XXXXXXXXXXXXX	13	251
2.4300	+	0	251
2.4480	+XXXXXXXXXXXXXX	15	266
2.4660	+XXXXXXX	7	273
2.4840	+	0	273
2.5020	+XXXXXXX	7	280
2.5200	+	0	280
2.5380	+XXXXXXXXX	9	289
2.5560	+	0	289
2.5740	+XXXXXXXXXXXXXXX	16	305
2.5920	+XXXXXXXX	8	313
2.6100	+	0	313
2.6280	+XXX	3	316
2.6460	+	0	316
2.6640	+X	1	317
2.6820	+	0	317
2.7000	+XXX	3	320

P2 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
2.7000	+XXX	3	3
2.7150	+	0	3
2.7300	+XXXXXXXXXXX	12	15
2.7450	+	0	15
2.7600	+XXXXXXXXXXXXXXXX	17	32
2.7750	+	0	32
2.7900	+XXXXXXXXXXXXXXXX	17	49
2.8050	+	0	49
2.8200	+XXXXXXXXXXXXXXXXXXXXXXXXXXXX	30	79
2.8350	•	0	79
2.8500	+	0	79
2.8650	+XXXXXXXXXXXXXXXXXXXXXXXXXXXX	31	110
2.8800	+	0	110
2.8950	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX*	49	159
2.9100	+XXXXXXXXXXXXXXXXXX	19	178
2.9250	+	0	178
2.9400	+XXXXXXXXXXXXXXXXXXXXXXXXX	26	204
2.9550	+	0	204
2.9700	+	0	204
2.9850	+XXXXXXXXXXXXXXXXXXXXXXXXX	27	231
3.0000	+	0	231
3.0150	+XXXXXXXXXX	10	241
3.0300	+	0	241
3.0450	+XXXXXXXXXXXXXXX	16	257
3.0600	+	0	257
3.0750	+XXXXXXXXXXXXX	14	271
3.0900	+	0	271
3.1050	+XXXXXXX	7	278
3.1200	+	0	278
3.1350	+	0	278
3.1500	+XXXXX	5	283
3.1650	+	0	283
3.1800	+XXXXXXX	7	290
3.1950	+	0	290
3.2100	+XXXXXXXXXX	10	300
3.2250	+	0	300
3.2400	+XXXXXX	6	306
3.2550	+	0	306
3.2700	+XXXXXX	6	312
3.2850	+	0	312
3.3000	+	0	312
3.3150	+XXXX	4	316
3.3300	+X	1	317
3.3450	•	0	317
3.3600	+X	1	318
3.3750	+	0	318
3.3900	+	0	318
3.4050	+X	1	319
3.4200	+	0	319
3.4350	+X	1	320

T2 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
2.9000	+	0	0
2.9250	+	0	0
2.9500	+XX	2	2
2.9750	+	0	2
3.0000	+X	1	3
3.0250	+X	1	4
3.0500	+X	1	5
3.0750	+XXX	3	8
3.1000	+	0	8
3.1250	+XXXXXXXX	8	16
3.1500	+XXXXXXXXXXXXXXXX	16	32
3.1750	+XXXXXXXXXXXXXX	14	46
3.2000	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	34	80
3.2250	+	0	80
3.2500	+XXXXXXXXXXXXXXXXXXXXXXXXX	27	107
3.2750	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	36	143
3.3000	+	0	143
3.3250	+XXXXXXXXXXXXXXXXXXXX	21	164
3.3500	+XXXXXXXXXXXXXXXXXXXXXXXXXX	27	191
3.3750	+XXXXXXXXXXXXXXXXXXXXXXXXXX	27	218
3.4000	+XXXXXXXXXXXXXXXX	16	234
3.4250	+XXXXXXXXXXXX	12	246
3.4500	+	0	246
3.4750	+XXXXXXXXXXXXXXXX	16	262
3.5000	+XXXXXXXXXX	11	273
3.5250	+XXXXXXXXX	9	282
3.5500	+	0	282
3.5750	+XXXXX	5	287
3.6000	+XXXXXXX	7	294
3.6250	+XXXXXXXX	8	302
3.6500	+XXXXXX	6	308
3.6750	+	0	308
3.7000	+XXX	3	311
3.7250	+	0	311
3.7500	+X	1	312
3.7750	+	0	312
3.8000	+XXX	3	315
3.8250	+XX	2	317
3.8500	+	0	317
3.8750	+XX	2	319
3.9000	+	0	319
3.9250	+	0	319
3.9500	+	0	319
3.9750	+	0	319
4.0000	+	0	319
4.0250	+	0	319
4.0500	+X	1	320

P3 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
3.3800	+	0	0
3.4000	+X	1	1
3.4200	+	0	1
3.4400	+	0	1
3.4600	+	0	1
3.4800	+XXXX	4	5
3.5000	+XXX	3	8
3.5200	+	0	8
3.5400	+XXXXXX	6	14
3.5600	+XXXXXXXXXXXXXXXXXX	19	33
3.5800	+XXXXXXXXXXXXXX	15	48
3.6000	+	0	48
3.6200	+XXXXXXXXXXXXXX	15	63
3.6400	+	0	63
3.6600	+XXXXXXXXXXXXXX	15	78
3.6800	+	0	78
3.7000	+XXXXXXXXXXXXXXXXXX	19	97
3.7200	+XXXXXXXXXXXXXXXXXXXXXXXX	26	123
3.7400	+XXXXXXXXXXXXXXXXXXXXXXXXXX	28	151
3.7600	+	0	151
3.7800	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	39	190
3.8000	+	0	190
3.8200	+XXXXXXXXXXXXXXXXXX	19	209
3.8400	+	0	209
3.8600	+XXXXXXXXXXXXXXXXXXXXXXXX	26	235
3.8800	+XXXXXXXXXXXXXX	15	250
3.9000	+XXXXXXXXXXXXXXXXXX	19	269
3.9200	+	0	269
3.9400	+XXXXXXX	7	276
3.9600	+	0	276
3.9800	+XXXXXXXXXX	10	286
4.0000	+XXXXXXXXXXXX	13	299
4.0200	+	0	299
4.0400	+XXXXX	5	304
4.0600	+	0	304
4.0800	+XXXX	4	308
4.1000	+	0	308
4.1200	+XXX	3	311
4.1400	+XX	2	313
4.1600	+	0	313
4.1800	+	0	313
4.2000	+X	1	314
4.2200	+	0	314
4.2400	+XXX	3	317
4.2600	+	0	317
4.2800	+X	1	318
4.3000	+XX	2	320

T3 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
3.9000	+	0	0
3.9250	+	0	0
3.9500	+	0	0
3.9750	+X	1	1
4.0000	+XXXXXX	6	7
4.0250	+	0	7
4.0500	+XXX	3	10
4.0750	+XXXXXXX	7	17
4.1000	+	0	17
4.1250	+XXXXXXXXXXXX	12	29
4.1500	+XXXXXXXXXXXXXXXXXXXXXXXXXX	27	56
4.1750	+XXXXXXXXXXXXX	13	69
4.2000	+XXXXXXXXXXXXXXXXXXXXXXXXX	26	95
4.2250	+XXXXXXXXXXXXXXXXXXXXXX	22	117
4.2500	+	0	117
4.2750	+XXXXXXXXXXXXXXXXXXXXXXXXXXXX	29	146
4.3000	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXX	31	177
4.3250	+XXXXXXXXXXXXXXXXX	18	195
4.3500	+	0	195
4.3750	+XXXXXXXXXXXXXXXXXXXXXXXXXXXX	30	225
4.4000	+XXXXXXXXXXXXXXXXXX	20	245
4.4250	+XXXXXXXXXX	11	256
4.4500	+XXXXXXXX	8	264
4.4750	+	0	264
4.5000	+XXXXXXXXXXX	12	276
4.5250	+XXXXXX	6	282
4.5500	+XXXXXXXXXXX	12	294
4.5750	+	0	294
4.6000	+X	1	295
4.6250	+XXXXX	5	300
4.6500	+XXXX	4	304
4.6750	+XX	2	306
4.7000	+X	1	307
4.7250	+	0	307
4.7500	+XXXX	4	311
4.7750	+XX	2	313
4.8000	+	0	313
4.8250	+X	1	314
4.8500	+X	1	315
4.8750	+XX	2	317
4.9000	+X	1	318
4.9250	+	0	318
4.9500	+X	1	319
4.9750	+	0	319
5.0000	+X	1	320

P4 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
4.6600	+	0	0
4.6800	+XX	2	2
4.7000	+XX	2	4
4.7200	+	0	4
4.7400	+XXXXXXXX	8	12
4.7600	+	0	12
4.7800	+XXXXXXXXX	9	21
4.8000	+	0	21
4.8200	+XXXXX	5	26
4.8400	+XXXXXXXXXX	10	36
4.8600	+XXXXXXXXXX	10	46
4.8800	+	0	46
4.9000	+XXXXXXXXXXXXXXXXXXXXXXXX	25	71
4.9200	+	0	71
4.9400	+XXXXXXXXXXXXXXXXXXXXXXXX	25	96
4.9600	+	0	96
4.9800	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	36	132
5.0000	+XXXXXXXXXXXXXXXXXX	20	152
5.0200	+	0	152
5.0400	+XXXXXXXXXXXXXXXXX	18	170
5.0600	+	0	170
5.0800	+XXXXXXXXXXXXXXXXX	18	188
5.1000	+XXXXXXXXXXXXX	14	202
5.1200	+XXXXXXXXXXXXX	14	216
5.1400	+	0	216
5.1600	+XXXXXXXXXXXXX	14	230
5.1800	+	0	230
5.2000	+XXXXXXXXXX	10	240
5.2200	+	0	240
5.2400	+XXXXX	5	245
5.2600	+XXXX	4	249
5.2800	+XXXXX	5	254
5.3000	+	0	254
5.3200	+X	1	255
5.3400	+	0	255
5.3600	+XXXXXX	6	261
5.3800	+	0	261
5.4000	+XXXXXXX	7	268
5.4200	+XXX	3	271
5.4400	+XXXXX	5	276
5.4600	+	0	276
5.4800	+XXXX	4	280
5.5000	+XXX	3	283
5.5200	+	0	283
5.5400	+X	1	284
5.5600	+	0	284
5.5800	+XXXX	4	288

P5 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
5.3200	X	1	1
5.3400		0	1
5.3600	XXXXX	5	6
5.3800		0	6
5.4000	XXXXXXXXX	9	15
5.4200	XXXXXXXXXXXXXXX	15	30
5.4400	XXXXXXXXXXXXXXX	15	45
5.4600		0	45
5.4800	XXXXXXXXXXXXXXXXXX	18	63
5.5000	XXXXXXXXXX	10	73
5.5200		0	73
5.5400	XXXXXXXXXXXXXXXXXXXXX	22	95
5.5600		0	95
5.5800	XXXXXXXXXXXXXXXXXXX	20	115
5.6000		0	115
5.6200	XXXXXXXXXX	10	125
5.6400	XXXXXXXXXXXX	13	138
5.6600	XXXXXXXX	8	146
5.6800		0	146
5.7000	XXXXXXXXXXXXXXXXXXXX	22	168
5.7200		0	168
5.7400	XXXXXXXXXXXXXXXXXX	19	187
5.7600		0	187
5.7800	XXXXXXXXXXXXXXXXXX	20	207
5.8000	XXXXXXXXXXXXXXXXXXXXX	23	230
5.8200	XXXXXXXXXXXXXXXXXXXXXXXX	26	256
5.8400		0	256
5.8600	XXXXXXX	7	263
5.8800		0	263
5.9000	XXXXXXXXXXXXXXXXX	18	281
5.9200		0	281
5.9400	XXXXXX	6	287
5.9600	XX	2	289
5.9800	XXX	3	292
6.0000		0	292
6.0200		0	292
6.0400	XX	2	294
6.0600	XXXX	4	298
6.0800	XXXX	4	302
6.1000		0	302
6.1200		0	302
6.1400		0	302
6.1600	X	1	303
6.1800		0	303
6.2000		0	303
6.2200		0	303
6.2400	X	1	304

T5 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
6.0400	+	0	0
6.0600	+XX	2	2
6.0800	+XXX	3	5
6.1000	+	0	5
6.1200	+XXXX	4	9
6.1400	+	0	9
6.1600	+XXXXXXXXXXX	12	21
6.1800	+	0	21
6.2000	+XXXXXXXXXXXXXX	15	36
6.2200	+XXXXXXXXXXXXXXXX	17	53
6.2400	+XXXXXXXXXXXXXXXXX	18	71
6.2600	+	0	71
6.2800	+XXXXXXXXXXXXXXXX	17	88
6.3000	+	0	88
6.3200	+XXXXXXXXXXXXXXXXXXXXXXXX	25	113
6.3400	+	0	113
6.3600	+XXXXXXXXXXXXXXXXXXXXXXXXX	27	140
6.3800	+XXXXXXXXXXXXXXXXX	19	159
6.4000	+XXXXXXXXXXXXXXXXX	20	179
6.4200	+	0	179
6.4400	+XXXXXXXXXXXXXXXXXXXXXXXXX	29	208
6.4600	+	0	208
6.4800	+XXXXXXXXXXXXXXXXXXX	22	230
6.5000	+XXXXXXXXX	9	239
6.5200	+	0	239
6.5400	+XXXXXXXXX	9	248
6.5600	+	0	248
6.5800	+XXXXXXXXX	10	258
6.6000	+XXXXXX	6	264
6.6200	+XXXXX	5	269
6.6400	+	0	269
6.6600	+XXXXXX	6	275
6.6800	+	0	275
6.7000	+XXXXXX	7	282
6.7200	+	0	282
6.7400	+XXXXXX	7	289
6.7600	+XXXXX	5	294
6.7800	+XXXXXXXXX	9	303
6.8000	+	0	303
6.8200	+XXXXXX	6	309
6.8400	+	0	309
6.8600	+X	1	310
6.8800	+	0	310
6.9000	+XX	2	312
6.9200	+XXXXX	5	317
6.9400	+XXX	3	320

P8 LATENCIES

MSEC	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
7.0500	+	0	0
7.0750	+	0	0
7.1000	+	0	0
7.1250	+	0	0
7.1500	+XXX	3	3
7.1750	+XX	2	5
7.2000	+XXXXXXXXX	9	14
7.2250	+	0	14
7.2500	+XXXXXX	6	20
7.2750	+XXXXXX	6	26
7.3000	+	0	26
7.3250	+XXXXXXXXXXXXXXXXXXXX	21	47
7.3500	+XXXXXXXXXXXXXX	14	61
7.3750	+XXXXXXXXXXXXXX	14	75
7.4000	+XXXXXXXXXXXXXXXXXXX	20	95
7.4250	+XXXXXXXXXXXXXX	14	109
7.4500	+	0	109
7.4750	+XXXXXXXXXXXXXXXXX	18	127
7.5000	+XXXXXXXXXXXXXXX	15	142
7.5250	+XXXXXXXXXXXXXXXXXXXXXXXXXXX	28	170
7.5500	+	0	170
7.5750	+XXXXXXXXXXXXXXXXXXXXX	22	192
7.6000	+XXXXXXX	7	199
7.6250	+XXXXXXXXXXXXXXX	15	214
7.6500	+XXXXXXXXXXXXXXXXX	18	232
7.6750	+	0	232
7.7000	+XXXXXXXXXXXXX	14	246
7.7250	+XXXXXXXXXX	10	256
7.7500	+XXXX	4	260
7.7750	+	0	260
7.8000	+XXXXXXX	7	267
7.8250	+XXXXXXX	7	274
7.8500	+XXXXXXXXX	9	283
7.8750	+XXXXXXX	7	290
7.9000	+XX	2	292
7.9250	+	0	292
7.9500	+X	1	293
7.9750	+XXXX	4	297
8.0000	+X	1	298
8.0250	+	0	298
8.0500	+XX	2	300
8.0750	+XX	2	302
8.1000	+	0	302
8.1250	+	0	302
8.1500	+XX	2	304
8.1750	+	0	304

T6 LATENCIES

MSEC.	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
7.3850	+	0	0
7.4200	+	0	0
7.4550	+X	1	1
7.4900	+XXX	3	4
7.5250	+XX	2	6
7.5600	+XXXXX	5	11
7.5950	+XXXX	4	15
7.6300	+XX	2	17
7.6650	+XXX	3	20
7.7000	+XXXXX	5	25
7.7350	+XXXXXXXXX	9	34
7.7700	+XXXXXXXX	8	42
7.8050	+XXXXXXXXXXXXXXXXX	17	59
7.8400	+XXXXXXXXXX	10	69
7.8750	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX*	49	118
7.9100	+XXXXXXXXXXXXXXXXXXX	19	137
7.9450	+XXXXXXXXXXXXXXXXXXXXXXXXX	25	162
7.9800	+XXXXXXXXXXXXX	13	175
8.0150	+XXXXXXXXXXXXXXXXXXXXXXX	23	198
8.0500	+XXXXXXXXXXXXXXXX	16	214
8.0850	+XXXXXXXXX	9	223
8.1200	+XXXXXXXXXXXXX	13	236
8.1550	+XXXXXXX	7	243
8.1900	+XXXXXXXXXXXX	12	255
8.2250	+XXXXX	5	260
8.2600	+	0	260
8.2950	+XXXXXXX	7	267
8.3300	+XXXXX	5	272
8.3650	+XXXX	4	276
8.4000	+XX	2	278
8.4350	+XX	2	280
8.4700	+XX	2	282
8.5050	+XXXX	4	286
8.5400	+XXXX	4	290
8.5750	+	0	290
8.6100	+X	1	291
8.6450	+	0	291
8.6800	+	0	291
8.7150	+X	1	292
8.7500	+XXX	3	295
8.7850	+XXX	3	298
8.8200	+	0	298
8.8550	+XXX	3	301
8.8900	+X	1	302
8.9250	+	0	302
8.9600	+X	1	303
8.9950	+X	1	304

P1 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
-.14000	+	0	0
-.10500	+	0	0
-.07000	+X	1	1
-.03500	+	0	1
0.0000	+X	1	2
.03500	+X	1	3
.07000	+XXXX	4	7
.10500	+XXXXX	5	12
.14000	+XXXXXXXXXXX	12	24
.17500	+XXXXXXXX	8	32
.21000	+XXXXXXXXXXXXXXXXXX	19	51
.24500	+XXXXXXXXXXXXXXXXXXXXXXXXXX	28	79
.28000	+XXXXXXXXXXXXXXXXXXXXXX	24	103
.31500	+XXXXXXXXXXX	12	115
.35000	+XXXXXXXXXXXXXXXXXXX	20	135
.38500	+XXXXXXXXX	9	144
.42000	+XXXXXXXXXXXXXXXXX	19	163
.45500	+XXXXXXXXXXX	12	175
.49000	+XXXXXXXXXX	11	186
.52500	+XXXXXXXXXXXX	13	199
.56000	+XXXX	4	203
.59500	+XXXXXXXXXXXXXX	15	218
.63000	+XXXXXXXXX	9	227
.66500	+XXXXXXXXXXXXX	14	241
.70000	+XXXXXXX	7	248
.73500	+XXXXXXXXX	9	257
.77000	+XXXXXXXXXX	11	268
.80500	+XXXXXXXXXX	11	279
.84000	+XXXXX	5	284
.87500	+	0	284
.91000	+XXXXXXXX	8	292
.94500	+XXXXX	5	297
.98000	+XXX	3	300
1.0150	+XXXX	4	304
1.0500	+XXXXXX	6	310
1.0850	+XX	2	312
1.1200	+X	1	313
1.1550	+	0	313
1.1900	+X	1	314
1.2250	+XXX	3	317
1.2600	+	0	317
1.2950	+	0	317
1.3300	+X	1	318
1.3650	+	0	318
1.4000	+XX	2	320

P2 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
-.66000	+X	1	1
-.60000	+	0	1
-.54000	+	0	1
-.48000	+X	1	2
-.42000	+	0	2
-.36000	+XX	2	4
-.30000	+	0	4
-.24000	+XXX	3	7
-.18000	+	0	7
-.12000	+X	1	8
-.06000	+XXXX	4	12
0.0000	+XXXXXXXXX	9	21
.06000	+XXXX	4	25
.12000	+XXXXXXXXXX	10	35
.18000	+XXXXXXXXXXXXXXXXXXXXXXX	24	59
.24000	+XXXXXXXXXXXXXXXXXXXXXX	23	82
.30000	+XXXXXXXXXXXXXXXXXXXXXXXX	25	107
.36000	+XXXXXXXXXXXXXXXXXXXX	22	129
.42000	+XXXXXXXXXXXXXXXXXXXXXXXXX	26	155
.48000	+XXXXXXXXXXXXXXXXXX	19	174
.54000	+XXXXXXXXXXXXXXXXXX	20	194
.60000	+XXXXXXXXXXXX	13	207
.66000	+XXXXXXXXXXXXXXXXX	18	225
.72000	+XXXXXXXXXXX	12	237
.78000	+XXXXXXXXXXXX	13	250
.84000	+XXXXXXXXXXXXXXXXXX	19	269
.90000	+XXXXXXX	7	276
.96000	+XXXXXXX	7	283
1.0200	+XX	2	285
1.0800	+XXXXX	5	290
1.1400	+XX	2	292
1.2000	+XXXXXXXX	8	300
1.2600	+XX	2	302
1.3200	+XXXXXXX	7	309
1.3800	+X	1	310
1.4400	+XX	2	312
1.5000	+	0	312
1.5600	+	0	312
1.6200	+XX	2	314
1.6800	+	0	314
1.7400	+XX	2	316
1.8000	+X	1	317
1.8600	+X	1	318
1.9200	+X	1	319
1.9800	+	0	319
2.0400	+	0	319
2.1000	+	0	319
2.1600	+X	1	320

T2 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
-.66000	+X	1	1
-.60000	+	0	1
-.54000	+	0	1
-.48000	+X	1	2
-.42000	+	0	2
-.36000	+XX	2	4
-.30000	+	0	4
-.24000	+XXX	3	7
-.18000	+	0	7
-.12000	+X	1	8
-.06000	+XXXX	4	12
0.0000	+XXXXXXXXX	9	21
.06000	+XXXX	4	25
.12000	+XXXXXXXXXX	10	35
.18000	+XXXXXXXXXXXXXXXXXXXXXX	24	59
.24000	+XXXXXXXXXXXXXXXXXXXXXX	23	82
.30000	+XXXXXXXXXXXXXXXXXXXXXXX	25	107
.36000	+XXXXXXXXXXXXXXXXXXXX	22	129
.42000	+XXXXXXXXXXXXXXXXXXXXXXXX	26	155
.48000	+XXXXXXXXXXXXXXXXXX	19	174
.54000	+XXXXXXXXXXXXXXXXXXX	20	194
.60000	+XXXXXXXXXXXX	13	207
.66000	+XXXXXXXXXXXXXXXX	18	225
.72000	+XXXXXXXXXXX	12	237
.78000	+XXXXXXXXXXXX	13	250
.84000	+XXXXXXXXXXXXXXXXXX	19	269
.90000	+XXXXXXX	7	276
.96000	+XXXXXXX	7	283
1.0200	+XX	2	285
1.0800	+XXXXX	5	290
1.1400	+XX	2	292
1.2000	+XXXXXXXX	8	300
1.2600	+XX	2	302
1.3200	+XXXXXXX	7	309
1.3800	+X	1	310
1.4400	+XX	2	312
1.5000	+	0	312
1.5600	+	0	312
1.6200	+XX	2	314
1.6800	+	0	314
1.7400	+XX	2	316
1.8000	+X	1	317
1.8600	+X	1	318
1.9200	+X	1	319
1.9800	+	0	319
2.0400	+	0	319
2.1000	+	0	319
2.1600	+X	1	320

P3 AMPLITUDES

UVOLTS

	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
-.06000	+X	1	1
0.0000	+X	1	2
.06000	+X	1	3
.12000	+XX	2	5
.18000	+XXXXX	5	10
.24000	+XX	2	12
.30000	+XXXXX	5	17
.36000	+XXXXXXXXXX	10	27
.42000	+XXXXXXXXXXXXXXXX	16	43
.48000	+XXXXXXXXXXXXXXXXXX	18	61
.54000	+XXXXXXXXXXXXX	14	75
.60000	+XXXXXXXXXXXXXXXXXX	19	94
.66000	+XXXXXXXXXXXXXXX	16	110
.72000	+XXXXXXXXXX	11	121
.78000	+XXXXXXXXXXXXXXXXXX	19	140
.84000	+XXXXXXXXXXXXXXXXXXXX	22	162
.90000	+XXXXXXXXX	10	172
.96000	+XXXXXXXXXXXXXXXXXXXX	22	194
1.0200	+XXXXXXXXX	10	204
1.0800	+XXXXXXXXX	9	213
1.1400	+XXXX	4	217
1.2000	+XXXXXXXXX	10	227
1.2600	+XXXXXX	6	233
1.3200	+XXXXXXXXXXXXXXX	16	249
1.3800	+XX	2	251
1.4400	+XXXXXX	6	257
1.5000	+XXXX	4	261
1.5600	+XXXX	4	265
1.6200	+XXXXXXXXX	9	274
1.6800	+XXXX	4	278
1.7400	+XXXXXX	6	284
1.8000	+XXXX	4	288
1.8600	+XXXXXX	6	294
1.9200	+	0	294
1.9800	+XXXX	4	298
2.0400	+XX	2	300
2.1000	+XX	2	302
2.1600	+XXX	3	305
2.2200	+X	1	306
2.2800	+XX	2	308
2.3400	+XXXX	4	312
2.4000	+XX	2	314
2.4600	+	0	314
2.5200	+	0	314
2.5800	+XXX	3	317
2.6400	+X	1	318
2.7000	+XX	2	320

T3 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
-1.4000	+X	1	1
-1.3300	+	0	1
-1.2600	+	0	1
-1.1900	+	0	1
-1.1200	+	0	1
-1.0500	+	0	1
-.98000	+X	1	2
-.91000	+X	1	3
-.84000	+X	1	4
-.77000	+	0	4
-.70000	+XX	2	6
-.63000	+XX	2	8
-.56000	+XXXXXXXXXX	10	18
-.49000	+X	1	19
-.42000	+XXXXXXX	7	26
-.35000	+XXXXXXX	7	33
-.28000	+XXXXXXXXXXX	12	45
-.21000	+XXXXXXXXX	9	54
-.14000	+XXXXXXXXXXXXXXXX	17	71
-.07000	+XXXXXXXXXXXXX	14	85
0.0000	+XXXXXXXXXXXXXXXXXXXXXXX	24	109
.07000	+XXXXXXXXXXXXXXXX	17	126
.14000	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXX	32	158
.21000	+XXXXXXXXXXXXXXXXXXXXXXX	24	182
.28000	+XXXXXXXXXXXXXXXXXXXXXXX	24	206
.35000	+XXXXXXXXXXXXXXXX	16	222
.42000	+XXXXXXXXXXXXXXXX	17	239
.49000	+XXXXXXXXXXXXXXXXXXXX	21	260
.56000	+XXXXXX	6	266
.63000	+XXXXXX	6	272
.70000	+XXXXXX	6	278
.77000	+XXXX	4	282
.84000	+XXXXXXXX	8	290
.91000	+XXXX	4	294
.98000	+X	1	295
1.0500	+X	1	296
1.1200	+XXX	3	299
1.1900	+X	1	300
1.2600	+XXX	3	303
1.3300	+XXXX	4	307
1.4000	+XXXX	4	311
1.4700	+XX	2	313
1.5400	+XXX	3	316
1.6100	+XX	2	318
1.6800	+	0	318
1.7500	+	0	318
1.8200	+	0	318
1.8900	+X	1	319
1.9600	+X	1	320

P4 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
-.80000	+	0	0
-.70000	+X	1	1
-.60000	+	0	1
-.50000	+	0	1
-.40000	+XX	2	3
-.30000	+	0	3
-.20000	+	0	3
-.10000	+XXXX	4	7
0.0000	+XXXXXXXXXX	10	17
.10000	+X	1	18
.20000	+XXXXXXXX	8	26
.30000	+XXX	3	29
.40000	+XXXXXXXXXXXXXXX	16	45
.50000	+XXXXXXXX	8	53
.60000	+XXXXXXXXXX	11	64
.70000	+XXXXXXXXXXXXXXXXXXXXXXXXX	26	90
.80000	+XXXXXXX	7	97
.90000	+XXXXXXXXXXXXXXXXXX	19	116
1.0000	+XXXXXXXXXXXXXXXXXXXXXXXXXXX	28	144
1.1000	+XXXXXXXXXX	11	155
1.2000	+XXXXXXXXXXXX	13	168
1.3000	+XXXXXXXXXXXX	13	181
1.4000	+XXXXXXXXXXXXXXXXXX	19	200
1.5000	+XXXXXX	6	206
1.6000	+XXXXXXXX	9	215
1.7000	+XXXXXXXXX	10	225
1.8000	+XXXXXX	7	232
1.9000	+XXXXXX	7	239
2.0000	+XXXXXXXXX	10	249
2.1000	+XXXXX	6	255
2.2000	+XXX	3	258
2.3000	+XX	2	260
2.4000	+XXX	4	264
2.5000	+XXXXX	6	270
2.6000	+XX	2	272
2.7000	+XXXXXXX	8	280
2.8000	+X	1	281
2.9000	+XXX	3	284
3.0000	+X	1	285
3.1000	+	0	285
3.2000	+X	1	286
3.3000	+	0	286
3.4000	+	0	286
3.5000	+	0	286
3.6000	+X	1	287
3.7000	+	0	287
3.8000	+X	1	288

PS AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
-.24000	+XXX	3	3
-.16000	+	0	3
-.08000	+XXX	3	6
0.0000	+XXXXX	5	11
.08000	+XXXXXXXXX	9	20
.16000	+X	1	21
.24000	+XXXXXXXXXXX	11	32
.32000	+XXXXXXXXXXXXXXXX	17	49
.40000	+XXXXXXXXXXXXXXXX	16	65
.48000	+XXXXXXXXXX	10	75
.56000	+XXXXXXXXX	9	84
.64000	+XXXXXXXXXXXXXXXXX	18	102
.72000	+XXXXXXXXXXX	12	114
.80000	+XXXXXXXXXXX	12	126
.88000	+XXXXXXXXXXXXXXXX	17	143
.96000	+XXXXXXXXXXXXXXXXXX	19	162
1.0400	+XXXXXXXXXXXX	13	175
1.1200	+XXXXXXXXXXXX	13	188
1.2000	+XXXXXXXXXX	11	199
1.2800	+XXXXXXXXXX	11	210
1.3600	+XXXXXXXXXXX	12	222
1.4400	+XXXXXXXXXXX	12	234
1.5200	+XXXXX	6	240
1.6000	+XXXXXX	7	247
1.6800	+XXXXXXXX	9	256
1.7600	+XXXX	4	260
1.8400	+XXXXX	6	266
1.9200	+X	1	267
2.0000	+XXX	3	270
2.0800	+X	1	271
2.1600	+XXXXX	6	277
2.2400	+XXXXX	6	283
2.3200	+XXXX	4	287
2.4000	+XXXX	4	291
2.4800	+X	1	292
2.5600	+	0	292
2.6400	+XX	2	294
2.7200	+X	1	295
2.8000	+XX	2	297
2.8800	+XX	2	299
2.9600	+X	1	300
3.0400	+X	1	301
3.1200	+	0	301
3.2000	+XX	2	303
3.2800	+	0	303
3.3600	+	0	303
3.4400	+	0	303
3.5200	+X	1	304

T5 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
-3.2000	+	0	0
-3.1000	+X	1	1
-3.0000	+	0	1
-2.9000	+	0	1
-2.8000	+	0	1
-2.7000	+XX	2	3
-2.6000	+X	1	4
-2.5000	+XX	2	6
-2.4000	+X	1	7
-2.3000	+XX	2	9
-2.2000	+X	1	10
-2.1000	+XXX	3	13
-2.0000	+XXXXX	5	18
-1.9000	+XX	2	20
-1.8000	+XXX	3	23
-1.7000	+XX	2	25
-1.6000	+XXXXXXXXX	9	34
-1.5000	+XXXXX	5	39
-1.4000	+XXXXXXXXXX	10	49
-1.3000	+X	1	50
-1.2000	+XXXXXXXXXX	10	60
-1.1000	+XXXXXXXXXXX	11	71
-1.0000	+XXXXXXXXXXX	11	82
-.90000	+XXXXXXXXXXXXXXXXX	17	99
-.80000	+XXXXXXXXXXXXXXXXXXXXXXXXXXX	27	126
-.70000	+XXXXXXXXXXXXXXXXXXXXX	22	148
-.60000	+XXXXXXXXXXXXXXXXXXXXXXXXXX	27	175
-.50000	+XXXXXXXXXXXXXXXXXXXXXXX	24	199
-.40000	+XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	33	232
-.30000	+XXXXXXXXXX	10	242
-.20000	+XXXXXXXXXXXXXXX	15	257
-.10000	+XXXXXXX	7	264
0.0000	+XXX	3	267
.10000	+XXXX	4	271
.20000	+XXXXXXXXX	9	280
.30000	+XXXX	4	284
.40000	+XXXX	4	288
.50000	+XXXXX	5	293
.60000	+XXXXXX	6	299
.70000	+XXXX	4	303
.80000	+XXXXX	5	308
.90000	+XXXXX	5	313
1.0000	+XXXX	4	317
1.1000	+XX	2	319
1.2000	+	0	319
1.3000	+	0	319
1.4000	+X	1	320

P6 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	FREQUENCY CUM.
-1.7000	+	0	0
-1.6000	+	0	0
-1.5000	+X	1	1
-1.4000	+X	1	2
-1.3000	+XX	2	4
-1.2000	+XXXX	4	8
-1.1000	+XXXXX	5	13
-1.0000	+XXX	3	16
-.90000	+XXX	3	19
-.80000	+XXXXXXXXXXXXX	14	33
-.70000	+XXXX	4	37
-.60000	+XXXXXX	6	43
-.50000	+XXXXXXX	7	50
-.40000	+XXXXXXXXXXXXX	14	64
-.30000	+XXXXXXXXXXX	12	76
-.20000	+XXXXXXXXXXXXXXXXXXXXXX	23	99
-.10000	+XXXXXXXXXXXXXXXXXX	19	118
0.0000	+XXXXXXXXXXXXXXXXXXXX	22	140
.10000	+XXXXXXXXXXXXXXXXXXX	21	161
.20000	+XXXXXXXXXXXXXXXXXXXXXXXXXX	29	190
.30000	+XXXXXXXXXXXXXXXXXX	19	209
.40000	+XXXXXXXXXXXXXXXX	18	227
.50000	+XXXXXXXXXXXXX	14	241
.60000	+XXXXXXXXXXX	12	253
.70000	+XXXXXXXXX	9	262
.80000	+XXXXX	5	267
.90000	+XXXXXXXXX	9	276
1.0000	+XXXX	4	280
1.1000	+XXX	3	283
1.2000	+XXXX	4	287
1.3000	+X	1	288
1.4000	+XX	2	290
1.5000	+	0	290
1.6000	+XX	2	292
1.7000	+XX	2	294
1.8000	+XXX	3	297
1.9000	+X	1	298
2.0000	+	0	298
2.1000	+	0	298
2.2000	+XXX	3	301
2.3000	+X	1	302
2.4000	+	0	302
2.5000	+X	1	303
2.6000	+	0	303
2.7000	+	0	303
2.8000	+X	1	304

T6 AMPLITUDES

UVOLTS	5 10 15 20 25 30 35 40	FREQUENCY INT.	CUM.
-2.0000	+	0	0
-1.9200	+X	1	1
-1.8400	+	0	1
-1.7600	+	0	1
-1.6800	+X	1	2
-1.6000	+XXXX	4	6
-1.5200	+	0	6
-1.4400	+XXXX	4	10
-1.3600	+XXX	3	13
-1.2800	+XXXXXXXXXX	10	23
-1.2000	+XXX	3	26
-1.1200	+XXXXXXX	7	33
-1.0400	+XXXXXXX	7	40
-.96000	+XXXX	4	44
-.88000	+XXXXXXXX	8	52
-.80000	+XXXXXXXXXXXXX	14	66
-.72000	+XXXXXXXXXXXXXXX	15	81
-.64000	+XXXXXXXXXXXXXX	15	96
-.56000	+XXXXXXXXXXXXXXX	16	112
-.48000	+XXXXXXXXXXXX	13	125
-.40000	+XXXXXXXXXXXX	13	138
-.32000	+XXXXXXXXXXXXXXXXXX	20	158
-.24000	+XXXXXXXXXXXXXX	15	173
-.16000	+XXXXXXXXXXXXXXXX	18	191
-.08000	+XXXXXXXXXXXXXXXXX	19	210
0.0000	+XXXXXXXXXXX	12	222
.08000	+XXXXXXXXXX	11	233
.16000	+XXXXXXX	8	241
.24000	+XXXXXXXX	8	249
.32000	+XXXXXXXXX	10	259
.40000	+XXXX	4	263
.48000	+XXX	3	266
.56000	+XXXXXX	7	273
.64000	+XXXXX	5	278
.72000	+XX	2	280
.80000	+XXXXX	5	285
.88000	+XXXXXX	6	291
.96000	+X	1	292
1.0400	+XXX	3	295
1.1200	+XXX	3	298
1.2000	+XX	2	300
1.2800	+XX	2	302
1.3600	+	0	302
1.4400	+	0	302
1.5200	+X	1	303
1.6000	+	0	303
1.6800	+X	1	304

APPENDIX I

ANOVA TABLE FOR MONAURAL P5 DATA

MONAURAL P5 DATA

Source	df	Mean Square	F	Probability
Sex		2.17	0.58	0.455
Error	18	3.72		
Ear	1	1.08	2.59	0.125
Ear x Sex	1	0.00	0.01	0.937
Error	18	0.42		

APPENDIX J

NEWMAN-KEULS STUDENTIZED TANGE TESTS FOR RAW LATENCY DATA P4 -- DERIVATION BY PHASE

For the following table:
RR = Rarefaction right; Rarefaction left
RC = Rarefaction right; Condensation left
CR = Condensation right; Rarefaction left
CC = Consensation right; Condensation left

PEAK FOUR DERIVATION X PHASE INTERACTION

Newman-Keuls Studentized Range Test for
DERIVATION X PHASE groups.

	VM-P1	VM-P3	VN-P1	VM-P4	VN-P3	VN-P2	VN-P4	Shortest Significant Ranges
VM-P1	5.00	10.64	14.68	14.68	16.24	19.04	19.36	R2 = 9.23
VN-P3		5.64	9.68	9.68	11.24	14.04	14.36	R3 = 8.89
VN-P1			4.04	4.04	5.60	8.40	8.72	R4 = 8.49
VM-P4				0.00	1.56	4.36	4.68	R5 = 7.96
VN-P3					1.56	4.36	4.68	R5 = 7.94
VN-P2						2.90	3.12	R6 = 7.23
VM-P2							0.32	R7 = 6.07

Groups = 40; df = 51; Error term = 0.11