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Augmenting and Reducing
Auditory Brain Stem Responses
Among Individuals

presented by

Geoffrey Kwabla Pilot Amedofu

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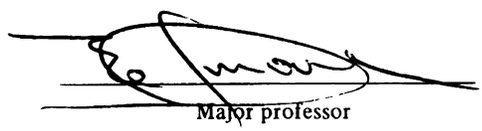
of the requirements for

M.A. degree in Audiology & Speech Sciences



Major professor

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AUGMENTING AND REDUCING AUDITORY BRAIN STEM EVOKED RESPONSES AMONG INDIVIDUALS

by

Geoffrey Kwabla Pilot Amedofu

A THESIS

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ABSTRACT

AUBMENTING AND REDUCING BRAIN STEM EVOKED RESPONSES AMONG INDIVIDUALS

By

Geoffrey Kwabla Pilot Amedofu

Among several acoustic variables which affect the amplitude and latency of ABR, intensity has been shown to have the greatest influence, in that there is a direct relationship between intensity and amplitude, and an inverse relationship between intensity and latency. While this is a widely held concept, the question is whether these observations are applicable to all hearing subjects.

Eleven audiometrically normal subjects with hearing no greater than 10 dB nHL were used. The intesnity levels were 60, 70, 80, and 90 dB nHL. The electrode placement was C_z -M_2 with forehead serving as the ground. Filtered clicks at 2000 and 4000 Hz were presented to the right ear of each subject at a repetition rate of 10/sec.

We demonstrated that subjects differed in their response to filtered clicks at different intensity levels. As regards amplitude, normal hearing subjects can be classified as strong or moderate augmenters, as well as reducers.

To my parents, Amegan (bereaved) and Kesevi,

my wife, Doris, and children, Manwuli and Sitsophe.

ACKNOWLEDGEMENTS

I wish to express my deepest appreciation to Dr. Ernest J. Moore, my major professor and chairman of my guidance committee, for his advice, guidance and encouragement during the planning, execution and writing of this study.

Special thanks go to other members of my guidance committee, Dr. Leo V. Deal, my academic advisor, and Dr. Linda L. Smith for their interest, direction and academic stimulation.

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I also extend my sincere gratitude to my parents, Amegah (bereaved) and Kesevi, brothers and sisters, wife, Doris, and children Mawuli and Sitsophe, who have painfully endured my involuntary absence.

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CHAPTER I

BACKGROUND AND PURPOSE

Auditory brain-stem audiometry has assumed a prominent and optimistic role as an objective method of assessing hearing sensitivity in the difficult-to-test population. These far-field potentials represent electrical events originating in the primary afferent auditory pathway during its course from the inner ear to the brain. They were first recorded in animals (Jewett, 1970) and then in human subjects (Sohmer and Feinmesser, 1967; Jewett, Romano and Williston, 1970; Moore 1971).

The anatomical origins of the auditory brain-stem response (ABR) often named with Roman numerals, have been demonstrated in both animals and human subjects (Bullock, Grinnell and Ikezone et al., 1968; Jewett, 1970; Lev and Sohmer, 1972; Jewett et al., 1970; Sohmer and Feinmesser, 1970). Previous data revealed that wave I originates from the VIIIth nerve, wave II from the cochlear nucleus, wave III from the superior olivary complex, wave IV from the lateral lemniscus, wave V from inferior colliculus, wave VI from medial geniculate body and finally wave VII from the auditory cortex (Jewett, 1970; Lev and Sohmer, 1972; Starr and Hamilton, 1976; Buchwald and Huang, 1975). Current literature on the anatomical origins of the waves has been given by Hashimoto, Ishiyama and Yoshimoto (1981) who noted that wave I is generated within the most distal portion of the auditory nerve, wave II from the most proximal part of the auditory nerve and the pons, waves III and IV from the pons, wave V from lateral lemniscus to the inferior colliculus and wave VI from the medial geniculate body. Hashimoto

et al. (1981) also found that . . . intracranially recorded brain-stem auditory evoked potential showed very rapid changes in amplitude within the brain stem. Noting that there were only slight changes in the more rostral regions, their amplitude gradients varied in the different components. They also demonstrated minor, but nevertheless, systematic shifts in latency with distance from the potential sources thus, reflecting a significant overlap of separate potentials.

Within a clinical setting, the ABR has become a vital adjunct to the clinical test battery of the otologist, audiologist, neurologist , neurosurgeon and pediatrician who are collectively involved in determining hearing sensitivity, site of lesion, central nervous system intergrity, pathology and maturation (Moore, 1983). It can be used to determine middle ear hearing loss (Popelka 1981) and sensorineural hearing loss (Jerger and Mauldin 1978), and to evaluate pseudohypacusis (Kavanaugh and Beardsley 1979). It has been shown to be of value for localization of brain-stem lesions (Starr and Achor, 1975; Stockard and Rossiter, 1977; Hashimoto, Ishiyama and Tozuka, 1979) and is now routinely applied for objective screening in neonates and children who cannot be tested with behavioural methods (Sohmer and Feinmesser, 1973; Hecox and Galambos 1975). Moore (1983) reported that

> brain-stems lesions cause a selective absence or alteration of one or more of the components and that patients with brain-stem damage due to various types of tumors, demyelinating diseases, diminished brain-stem circulation and brain death, show either absence of certain components or prolonged latency and reduced amplitude of response components (Moore, 1983, p. 13).

The wave-forms are elicited by click-like transients which are generated by driving an earphone with rapid rise-time, brief duration electrical pulse. Several acoustic variables, notably intensity, frequency and time influence the amplitude and latency of the ABR. Among these, intensity has been shown to have a direct influence on amplitude and latency. Various investigators (Romano and Williston, 1970; Rossie, solero and Pira, 1982; Moore 1971) have shown that as the intensity of the stimulus is increased, the amplitude of the ABR increases while latency decreases. While this has been demonstrated in a number of individuals, the question is whether the pattern is applicable to all subjects. It would appear that the amplitude relationship to intensity that we observe is not always true. Thus in this study, the relationship between amplitude and latency to clicks presented at high intensity levels was explored. It is always important to separate amplitude and latency in an investigation of this kind; since ABR latencies and amplitudes have been shown to reflect interactive but different physiologic processes which can covary or which may concurrently change (Stephens and Thorton, 1976; Robinson and Rudge, 1977). This brings into focus the idea of augmenting and reducing evoked potential measurements.

The idea of augmenting/reducing evoked potentials is not new. It was originally reported by Petre (1960) and further elaborated by Buchsbaum and Silverman (1968). Subjects in whom evoked potential amplitude increased as a function of stimulus intensity were classified as augmenters, while those whose amplitude remain constant or diminish as stimulus intensity is increased were classified as reducers. A thorough review of the results of the literature revealed that there has been no investigation conducted as

to augmenters/reducers for ABR data. Thus, this experimental investigation was designed to answer the following null hypotheses about the ABR:

(i) The amplitude of waves I, III and V of the ABR do not increase as the intensity of the stimulus is increased.

(ii) The latency of waves I, III and V of the ABR do not decrease as the intensity of the stimulus is increased.

(iii) The magnitude of the increase in amplitude as the intensity of the stimulus is increased is not the same for all individuals.

(iv) Audiometric test frequency has no effect on the amplitude of waves I, III and V of the ABR as intensity is increased.

(v) Audiometric test frequency has no effect on the latency of waves I, III and V of the ABR as intensity is increased.

CHAPTER TWO

REVIEW OF LITERATURE

The ideal stimulus for evoking the ABR has been a subject of great interest among several investigators. This interest was engendered by a desire on the part of clinicians to include the ABR test in the clinical armamentarium. For these reasons, it is necessary that the stimulus used in ABR has enough frequency specificity and dynamic range, and must be presented rapidly to conserve time. In addition, a sufficient number of responses must be averaged in order to obtain identifiable responses. The aim is to have a signal that is abrupt enough to synchronize primary auditory nerve units, yet long enough to maintain frequency specificity. Since it is difficult to have an abrupt signal in time and also have frequency specificity, a compromise is reached by the use of tone pips (Davis 1976); filtered clicks (Naunton and Zerlin, 1976), and short tone bursts (Moore 1971). But the most widely used stimulus for eliciting the ABR has been either acoustic clicks or tone pips. Thus, filtered clicks will be used in this investigation and are justified in the following sections.

Stimulus Parameters

There is a great deal of literature on certain stimulus parameters associated with biophysical electrical potentials other than the ABR (Rubin, 1967; Davis, 1976; Moore, 1971). These topics will not be discussed in this review. Specifically, this study will examine the

stimulus parameters most relevant to the ABR, namely time, frequency and intensity.

Hecox, Squires and Galambos (1976) studied the effects of stimulus duration on the latency and amplitude of wave V of the ABR using white noise bursts (specified in the article as 20-20,000 Hz, although it is well known that the band width was limited by the characteristics of the earphones). The recordings were taken from the vertex referenced to the right mastoid using a repetition rate of 16/ssec with varying duration (0.5, 2, 5, 20 and 30 msec) and rise-decay times of from 0-10 msec. They found that when the duration of the stimulus is increased, the latency of the ABR increases while the amplitude of the response decreases.

Moore (1983) also explored the effects of duration and inter stimulus interval (ISI) on the ABR. Frequency (4,000 Hz), intensity (90 dB SPL), rise-decay time (1.0) msec, the latency of all components increases while amplitude decreases. As it is known that as repetition rate is held constant, the ISI will decrease if duration is increased, and that as the ISI is decreased the various ABR waves become less distinct and show an increase in latency and a decrease in amplitude.

Schalafman (1977) investigated the effects of short tone bursts with various rise-decay times (0.01, 0.1, 0.25, 0.5, 1.0, 2 and 5) and durations (5.0 and 10 msec) on waves I through VI of the ABR. A 4,000 Hz tone burst was presented to the right ear of ten normal subjects at 90 dB SL using a rate of 9.2/sec. She found that as rise-decay time is increased, latency increases, while amplitude decreases. The two durations had no significant effect on the amplitude and latency of the ABR, due perhaps to the fact that the ISI was not appreciably decreased.

Hyde, Stephens and Thorton (1976) presented acoustic clicks at 60 dB SL. They found that as stimulus rates increased from 12/sec to 50/sec, there was a significant increase in the latency of all the waves except V.

Gerling and Hieber (1983) collected normative data on forty eight subjects to determine the effects of increasing stimulus rates on ABR. They found that increasing the stimulus rate from 10/sec to 90/sec with intensity held constant, resulted in a significant increase in the latency of wave V and an overall decrease in the amplitude of the ABR.

Picton, Stapells and Campbell (1981) explored the effects of frequency on the ABR using the "derived response technique." With this technique, ABRs are obtained to clicks presented in high-pass masking noise at different cut-off frequencies. Subtraction of the ABR response to clicks obtained at high-pass noise at a higher cut-off frequency results in a derived response to the frequency between the two cut-off settings (Picton et al. 1981). Clicks were presented in a high-pass filtered noise at -10, -20, -30, -40, -50 and 60 dB of attenuation at a repetition rate of 50/sec, and at frequencies from 500 Hz through 8,000 Hz. They found that waves I-IV are very small in amplitude. At the 8,000 Hz frequency band, wave V is most recognizable below 30 dB. For the other frequency bands, however, wave V is recognizable down to 10 dB, its latency increasing with decreasing frequency and with decreasing intensity.

Moore (1983) used short tone bursts at -10, -20, -30 and -40 dB of attenuation at various frequencies from 500 Hz through 8,000 Hz to elicit waves I-V of the ABR. Each tone burst was presented at 9.2/sec repetition rate, at a rise-decay time of 1.0 msec and a duration of 3.0 msec. He observed that for wave I, at the lowest intensity level (-40 dB of

attenuation) latency values do show a difference in time of occurence, while at the highest intensity level (-10 dB of attenuation), the shift in latency as a function of intensity is not observed. Separation in latency between 1000 Hz and 4000 Hz was observed regardless of the intensity level used. A decrease in intensity also shows a corresponding increase in latency for both 1000 Hz and 4000 Hz. He also found that as intensity is increased, the amplitude for wave V increased at 1000 Hz and at 4000 Hz.

Kodera, Yamane and Suzuki (1977) employed tone pips at -20, -30, -40 and -50 dB SL of attenuation, at three frequencies (500, 1000 and 2000 Hz), at a rate of 10/sec and with 5 msec rise-decay times. They found that the amplitude of the ABR decreased as the stimulus intensity is decreased. The latency of the ABR increased as stimulus intensity decreased and as the tone pip frequency decreased. This occurs because as stimulus intensity is decreased the more apical part of the cochlear is more selectively stimulated, and presumably, contributes to the major part of the ABR. As such, this will cause an increase in latency because of the longer travel time along the cochlear partition (Kodera, et al., 1977).

Jewett, Romano and Williston (1970) studied the relationship between ABR and stimulus intensity. They used a 0.1 msec electrical click. They found an inverse relationship between stimulus intensity and latency. As stimulus intensity is increased, they also observed that the amplitude of the ABR increased.

Picton, Stapells and Campbell (1981) investigated the effects of stimulus intensity on the ABR. They employed clicks at -40, -50, -60 and 70 dB of attenuation and at a rate of 11/sec. They found that as the intensity of the click is increased, the latency of the ABR decreased while

the amplitude increased.

Stockard, Westmoreland, and Corfits (1979) used rare-faction clicks presented at -30, -40, -50, -60 and -70 dB SL at rates of 10/sec and 70/sec. They found that the peak latencies of ABR increased with decreasing stimulus intensity. These reports closely parallel similar demonstrations by several other researchers (e.g. Moore, 1971; Lev and Sohmer, 1972; Hecox and Galambos, 1974; Kodera et al., 1977; Moore, 1983), who confirmed that increases in signal intensity produce systematic and highly stable decreases in ABR latency and increases in ABR amplitude.

Augmenting/Reducing Measurements

The idea of augmenting/reducing measurement was first described by Petre (1960), based on the notion that there is a tendency for some persons to reduce the intensity of a perception subjectively after they have been stimulated by a more intense perception (Kinesthetic Figural After Effects [KFA]). He blind-folded his subjects and asked them to feel with their right hand the width of a test object, a standard block of smooth wood. The subjects were then asked to feel a long tapered bar of similar wood and to determine the place on the bar where it seemed just as wide as the test block. Finally, the subjects were given a wider test block to rub with their right hand as before at a constant rate for 90 sec. After 90 sec of rubbing they again equated the original test block to the perceived equivalent width on the tapered bar, determining four equivalences. The rubbing was increased, first to 90 sec and then to 120 sec. Petre (1960) called those who reduced the size of the block subjectively after stimulation, reducers, and those who tend to enlarge the size of the block

subjectively were called augmenters.

Buchsbaum and Silverman (1968) developed a measure of augmenting/reducing that used signal averaging techniques to obtain an average visual evoked-potential measure. They employed four stimulus intensities ranging in brightness from 32-980 lumen seconds. A total of 120 stimulus presentations were summed. The flashes were also presented in blocks of ten at the same intensity. The intervals between the flashes and the blocks were 1.0 and 3.0 sec respectively. An X-Y plotter was used to record the curves for measurement. The electrocephalogram (EEG) was recorded from silver disk electrodes between vertex and right ear, with the left ear serving as ground. They classified individuals whose evoked potential amplitudes increased as a function of stimulus intensity as augmenters, while those in whom amplitudes do not increase or even diminish as reducers.

Braden, Haier and Space (1983) employed a system for obtaining evoked-potential augmenting/reducing measurements using the Apple II microcomputer. They used four different intensity levels of light flashes of 500 sec duration. The light flashes were repeated 256 times at one flash per sec, with 64 trials per intensity level. The EEG was recorded from the vertex referenced to the right ear. As before, individuals whose evoked-potential amplitudes increased as a function of stimulus intensity were classified as augmenters, while those whose amplitudes reduced were classified as reducers.

As was noted above, several of these studies investigated stimulus parameters which affect ABRs. It has been shown that as that intensity of the stimulus is increased, the amplitude of the ABR increases while latency

decreases. There is an apparent void in the results of the literature on whether the intensity relationship to amplitude that we observe is applicable to all subjects. Indeed, it has not been determined whether all ABR waves adhere to a latency increase or decrease, and amplitude increase-decrease relationship as function of intensity. Studies using kinesthetic figural after effects and visually evoked potential revealed that individuals differ in their response to different intensities, in that there are individuals who are augmenters, as well as individuals who are reducers. Thus, there have been no investigations conducted as to augmenters-reducers for ABR data. Another area that has not been fully explored is the relationship of augmenting-reducing potential as a function of audiometric frequencies and intensities. There is the need to conduct such an investigation, thus, the impetus for the present study.

CHAPTER III

INSTRUMENTATION AND PROCEDURE

Stimulus Generation System

The basic experimental apparatus employed in the presentation and control of the filtered clicks is depicted by the block diagram in figure III-1. The specific components were the following:

One power source (Madsen 2250)

One stimulus generator (Madsen 2250)

One attenuator (Madsen 2250)

Two earphones (Magnetically shielded, MSH 87)

One timer/counter (Madsen 2250)

One oscilloscope (Madsen 2250)

Two stimulus frequencies at 2000 and 4000 Hz were routed to the right ear of each subject. Stimulus generation was monitored by the LED (light emitting diode) section of the instrumentation.

Electrophysiologic Recording System

The experimental equipment employed in the recording of electrophysiologic activity is also shown in figure III-1. The specific components were the following:

(1) three gold disc electrodes (Grass ESH)

(2) one pre-amplifier (Madsen 2250) and a post-amplifier (Madsen 2250)

(3) one external filter (Krohnite, 3550)

(4) a signal averager (Madsen 2250)

(5) one monitor oscilloscope (Textronix VM-78)

(6) one X-Y plotter (Madsen 2250, ME 7010B).

Each of the electrodes was connected to the preamplifier which amplify the very small electrophysiologic signal from the subject. The amplified activity was filtered using a band pass of 20-3000 Hz and the gain was set at "Auto". This setting resulted in the gain varying between 136 dB to 100 dB HTL depending on on-going eletrophysiologic signals. The eletrophysiologic activity were then routed to the post-amplifier, the averager and summed 1024 times using a sweep time of 10 msec, 250 data points and a dwell time of 100 microseconds. The monitor-oscilloscope was used to display the 2000 and 4000 Hz signals that were delivered to the earphone. The summed responses were printed in an analog form on the X-Y recorder.

Callibration of Earphone

Figure III-2 shows the block diagram of the equipment used in the calibration of the earphones, using pure tones across the frequency range in octaves from 250-8000 Hz. As a first step, the microphone was calibrated using a pistophone. Secondly, a pure tone generator was connected to the external output of the Madsen ERA 2250. The output from the ERA was routed to the earphone (MSH 87) and coupled to the KEMAR.

Using a pure tone with an arbitrary intensity level of 70 dB generated through the ERA, the equivalent intensity level in dB SPL was noted on the meter. Any correction factor was observed by turning the calibration key, operating the attenuator and then watching the meter to compare the various

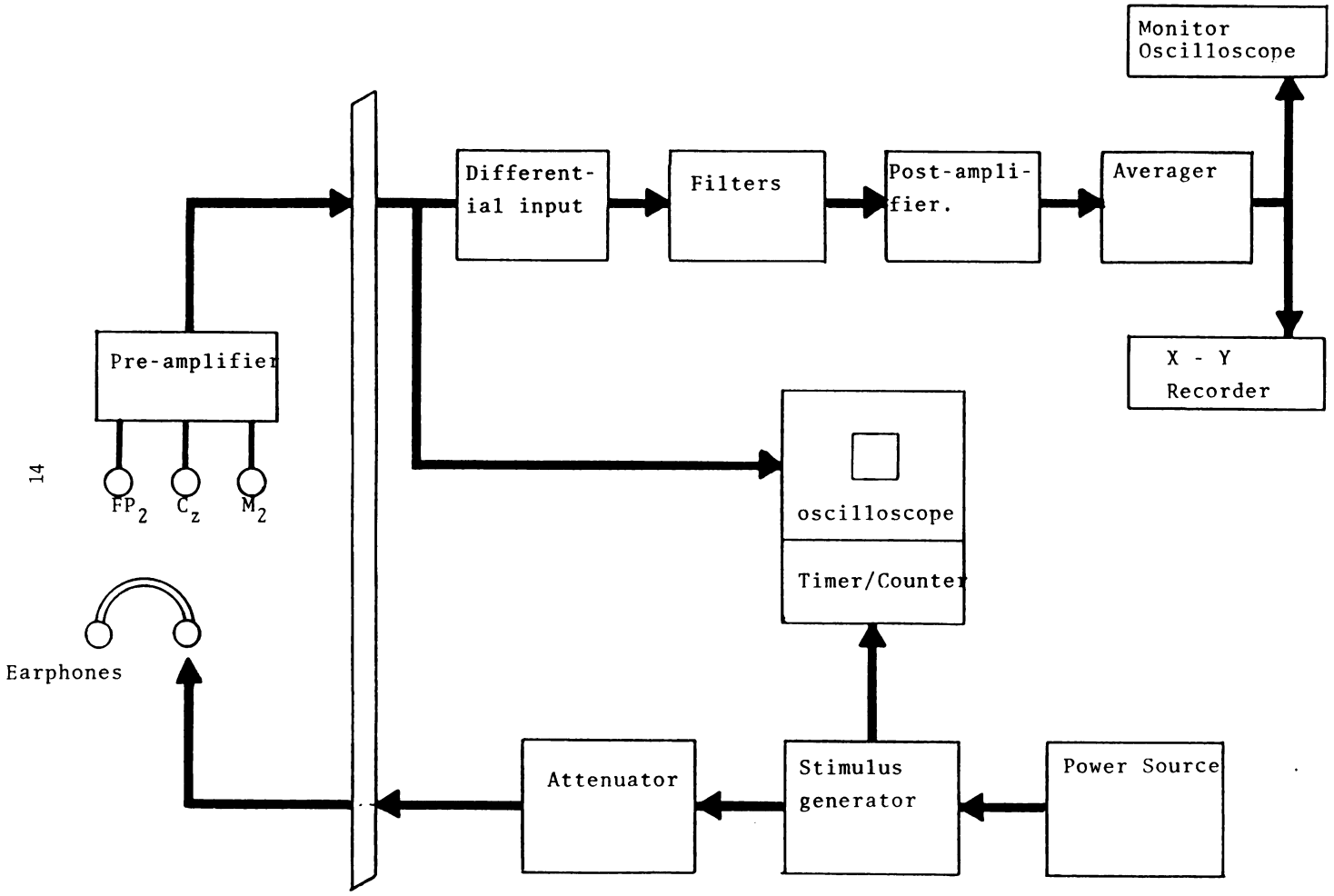


Figure 111-1 Instrumentation.

levels on the meter with the input generated at 70 dB.

To calibrate the output intensity, the calibration key was turned to the position of "Cal". The aim was to obtain "0" dB HTL with a 4000 Hz filtered click. After selecting the respective positions of the output switch, the frequency (2000 or 4000 Hz), and intensity level was adjusted to the zero dB level. Once the system was calibrated, the headset was then placed on the right ear of the subject. The averaging procedure was started using a 10/sec repetition rate. While the series of stimuli was presented to the subject, the intensity level was adjusted to subjective "0" dB level. The attenuator was operated in 1 dB steps during this procedure.

Procedure

Eleven audiometrically normal subjects with hearing no greater than 10 dB HTL (re: ANSI 1969) at frequencies from 250 through 8000 Hz were used. The subjects consisted of eleven women in the age range of 18-25 years. For experimental purposes only one ear of each subject was used.

As a first step in the experiment, audiometric threshholds were determined for the subjects using the modified Hughson Westlake procedure (Jerger and Carhart, 1959). The lowest intensity level at which the subject responded at two out of three presentations was defined as threshhold for tone bursts.

Three electrodes were attached to the subject, active on the Vertex (Cz), ground at the center of the forehead (FPz) and reference to the right mastoid (M_2). The skin over the area to which the electrodes were attached was cleaned with acetone and abraded with a solution (omniprep) to improve

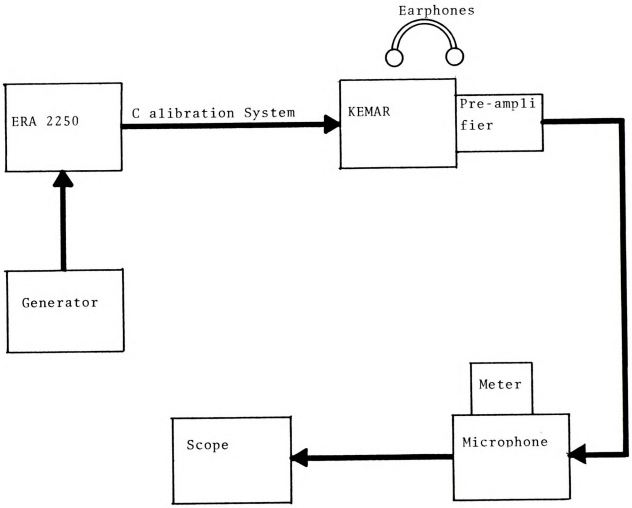

Earphones
ERA 2250
C alibration System
KEMAR
Pre-amplifier
Generator
Meter
Scope
Microphone

Figure 111-2

bioelectric conductivity. The electrode paste was placed into the disc electrodes and inserted into the "cup" within the clip electrode. The dis electrodes were applied to the vertex mastoid and the forehead with a gauze pad soaked in collodion and dried with cool compressed air from a compressed air machine. After the electrode was secured in place, a conductivity gel was inserted into the electrode cup by means of a blunt tipped syringe, through the perforation in the electrode cup, in order to increase conductivity. The electrodes were further secured with small pieces of surgical tape.

The subjects were seated in a reclining chair inside an acoustically and magnetically shielded room. Room lights remained off; and the subjects were told to relax, remain steady, keep their eyes closed, and, if possible, to sleep. A quiescent state was desirable since it promoted a quieter physiologic background and reduced ongoing physiologic noise levels (Moore, 1971).

Filtered clicks at frequencies 2000 and 4000 Hz were presented to the right ear of each subject at a 10/sec repetition rate. It was not appropriate to use frequencies lower than 2000 Hz, since such frequencies tend to elicit responses that are consistently smaller than those obtained for higher frequencies (Zerlin and Naunton, 1976). Picton et al., (1981) also revealed that at frequencies lower than 2000 Hz, the ABR showed only a slow broad wave V. The intensity of the tone burst was varied between 60 and 90 dB in 10 dB steps. The electrophysiologic activity was monitored visually to check for test artifacts; the artifact rejection circuit was utilized.

Data reduction and statistical analysis

ABR latencies were measured from the onset of the filtered clicks at the earphone to the most prominent peak of waves I, III and V. Peak-to-peak amplitude measurements were made from the first positive peak to the next negative trough of waves I, III or V. The mean, standard deviation, variance, slope and interceps were computed for amplitude and latency of waves I, III, and V across subjects for the six intensity levels and the three frequencies for waves I, III and V. The F-test was used to determine whether the means of the waves demonstrated a significant difference at the various intensity levels.

CHAPTER IV

RESULTS

This study employed a 2 X 4 factorial design, and it sought to determine latency and amplitude of the ABR response at two frequencies of 2000 and 4000 Hz and at four intensity levels of 60, 70, 80 and 90 dB nHL. Specifically, the study was designed to answer the following null hypotheses:

(1) The amplitude of waves I, III and V of the ABR do not increase in a systematic manner as the intensity of the stimulus is increased.

(2) the latency of waves I, III and V of the ABR do not decrease in a systematic manner as the intensity of the stimulus is increased.

(3) The magnitude of the increase in amplitude as the intensity of the stimulus is increased is not the same for all individuals.

(4) Audiometric test frequency has no effect on the amplitude of waves I, III and V of the ABR as intensity is increased.

(5) Audiometric test frequency has no effect on the latency of waves I, III and V of the ABR as intensity is increased.

Eleven audiometrically normal, female subjects in the age range of 18-25 years and with hearing no greater than 10 dB were used in the study. They were chosen from a total of fifty-seven subjects so as to constitute a

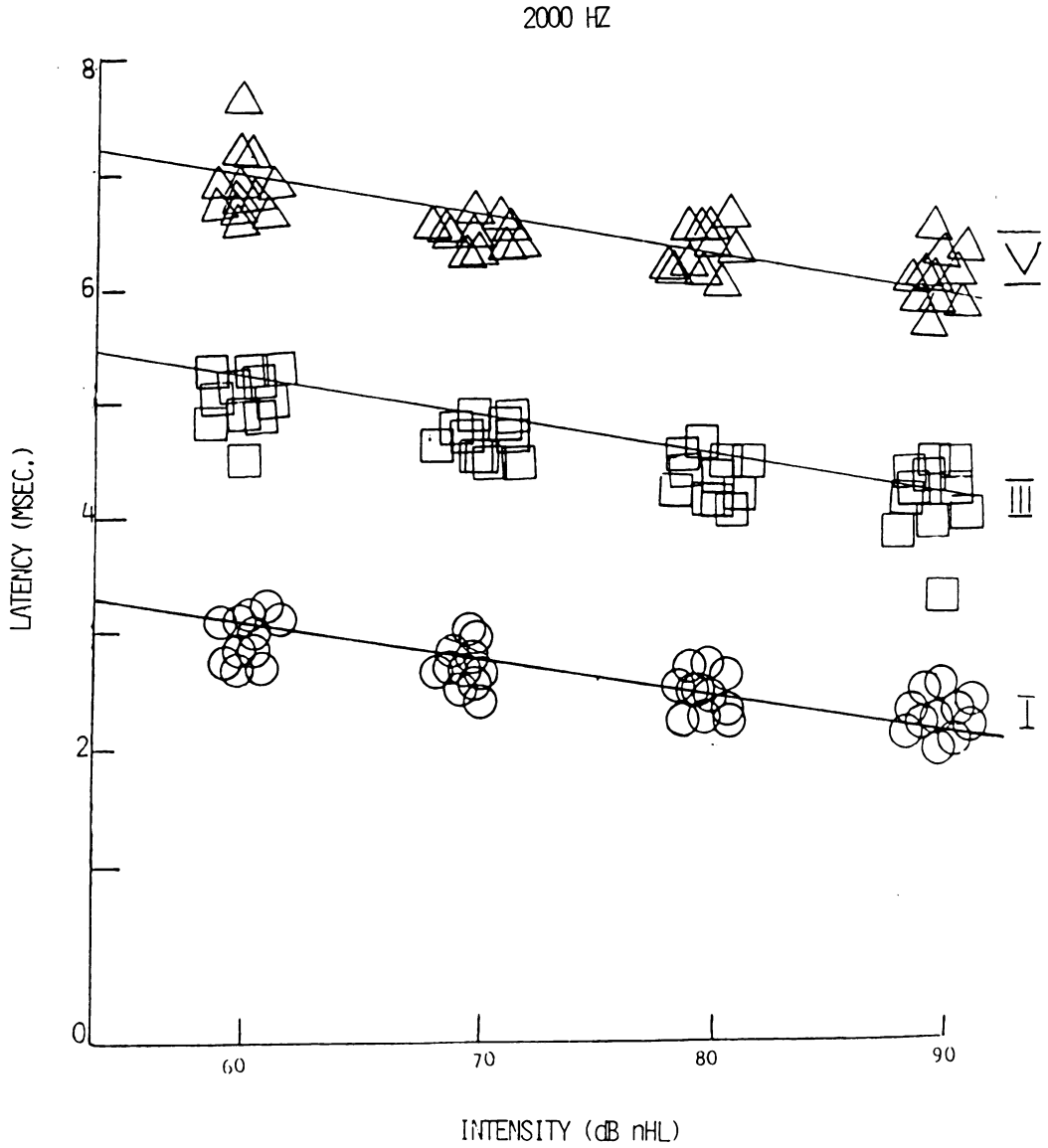
final sample. The electrode placement was, active on the vertex (C_2), ground at the center of the forehead (FP_2) and reference to the right ear (M_2). Filtered clicks at the frequencies of 2000 and 4000 Hz were presented to the right ear at a repetition rate of 10/sec. The typical responses obtained were a series of positive-negative waves occurring within the first 10 msec after stimulus onset. The waves obtained from all eleven subjects exhibited the same morphological characteristic as reported by earlier investigators (Jewett and Williston, 1971; Moore, 1971; Sohmer and Feinmesser, 1973). The analog wave forms for the eleven subjects can be found in Appendices A1 and A2, and the numerical values are listed in Appendices B1 and B2.

We see in Appendix C1 the latency input-output function for waves I, III and V at 2000 Hz. The salient features of these illustrations are that all three waves revealed a systematic decrease in latency as a function of stimulus intensity for a majority (N=9) of the subjects. The exceptions noted were for subjects PW and LB in which the latency remained constant at several intensity levels. This was the case for subject LB at 2000 Hz for waves I and V at intensity levels of 70 and 80 dB for waves I and V HL; and for subject PW for wave V at 2000 Hz at intensity levels of 60 and 70 dB, and at 80 and 90 dB. On the other hand, one can observe that functions in the illustrations (see Appendix CI) are parallel for nine of the eleven subjects. This, in effect, is an indication that latency was, indeed, decreasing as a function of stimulus intensity for a majority of the subjects. The one-way analysis of variance was calculated for waves I, III and V to determine whether the various stimulus intensity levels have any significant effect on the dependent variable of latency. It turns out that

the latencies of waves I, III and V are dependent on the various stimulus intensity levels. For instance, the one-way analysis of variance for the group data revealed that there were statistically significant differences between the mean values for latency. For wave I, it was found to be [F(2.84) = 28.38 > p 05]. We know that the null hypothesis states that no difference will be found between the means compared. If the null hypothesis is correct, we would find this large difference (see Appendix G1) between the sample means only once in twenty experiments. Since we have found this large difference, it is quite probable that the null hypothesis of no difference between the sample means is false. Therefore, we would reject the null hypothesis and conclude that the difference between the sample means reflected a true difference between the sample means.

Scatter diagrams were plotted to show how the independent variable of intensity and the dependent variable of latency are related. As can be seen in figure IV-I, intensity levels, or the independent variable, is plotted on the horizontal axis, while latency, the dependent variable, is plotted on the vertical axis. We know that the relationship between intensity and latency is an inverse function since increases in intensity tend to be associated with decreases in latency. Within this frame of reference, then, linear regression lines were drawn for the mean latencies of waves I, III and V using the method of least squares so as to provide an adequate representation of the average relationship between intensity and corresponding latency (see figure IV-I).

Figure IV-1. Individual data points for latency as a function of stimulus intensity for waves I, III and V at 2000 Hz. The method of least squares was used to construct the line of "best fit". Intercepts and slopes can be found in Appendix FI.


2000 HZ
8
6
4
2
0
LATENCY (MSEC.)
60
70
80
90
INTENSITY (dB nHL)
V
III
I

It is quite obvious that the lines that describe the relationship between intensity and latency are a decreasing accelerating function.

Correlation coefficients were calculated using the formula:

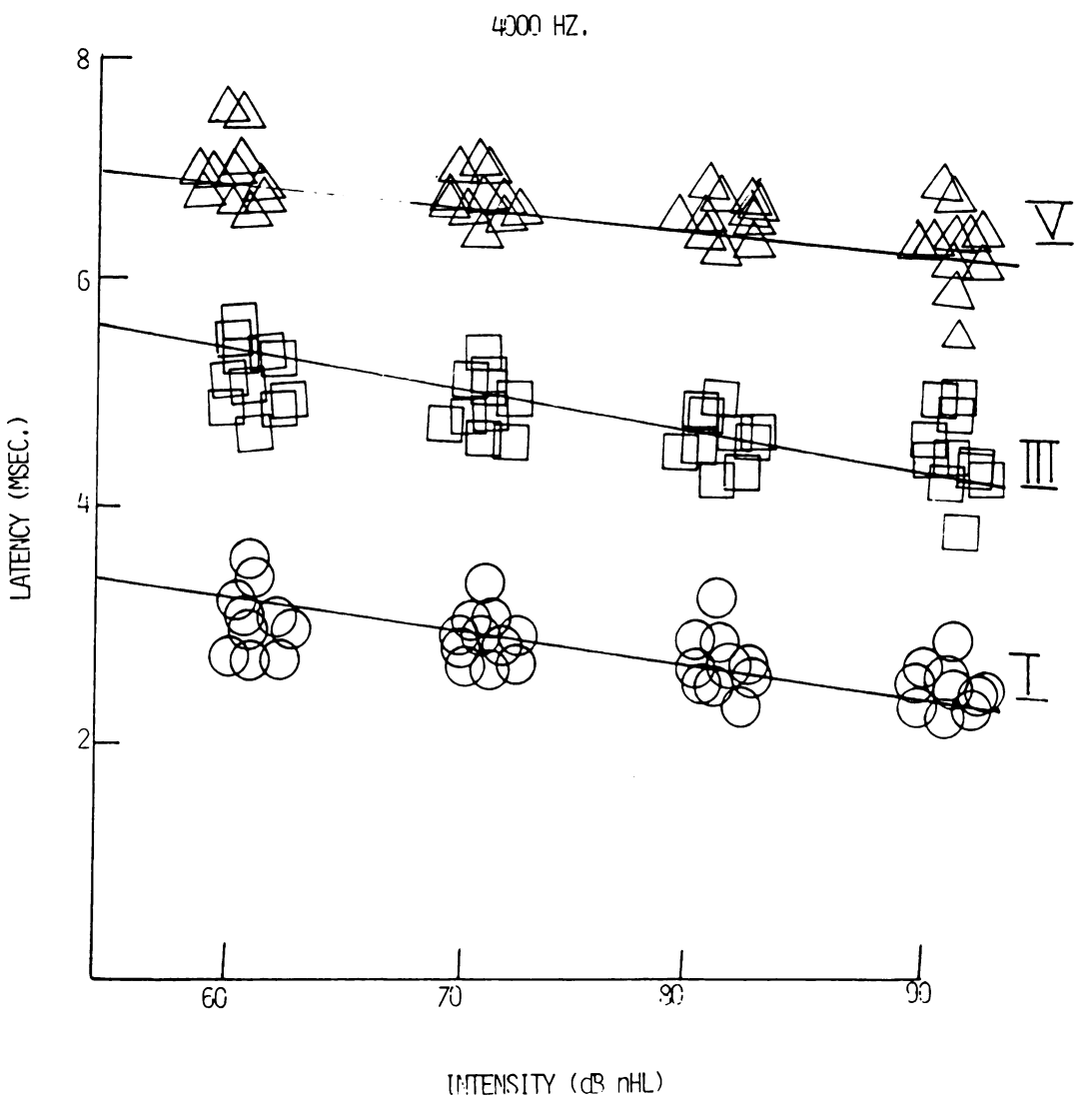
$$r = \frac{n\ xy\ -\ zxy}{\sqrt{n\ x^2\ -\ (x)^2\quad n\ y^2\ -\ (y)^2}}$$

The strength of the relationship was measured between intensity and latency values so that we could determine whether predictions could be made for latency, given a particular intensity level (see Appendix FI). Thus, the correlation coefficient measures the "degree of fit" of latency data points to the straight line function of each wave.

The correlation values for the latency of waves I, III and V showed a negative correlation coefficient. The correlation values for the waves were: wave I -.60, wave III -.84 and wave V -.45. The negative correlation values indicated that there is an indirect relationship between the latency of the ABR and stimulus intensity in that as the intensity of the stimulus is increased, latency decreases. The -.84 value found for wave III is indicative of a strong negative correlation between the stimulus intensity and latency.

Latency measure for 4000 Hz are given in Appendix C2. Individual input-output latency functions at intensity levels from 60 to 90 dB are given. We also see in figure IV-2 the scatter diagram and the linear regression lines for waves I, III and V. Here again we note that waves I, III and V showed a gradual decrease in latency as a function of stimulus intensity. The notable exception was subject PW in whom it was observed that the latency of wave I remained constant as a function of stimulus

Figure IV-2. Individual data points for latency as a function of stimulus intensity for waves I, III and V at 4000 Hz. The method of least squares was used to draw the lines of "best fit". Intercepts and slopes can be found in Appendix FI.


4000 HZ.
8
6
4
2
LATENCY (MSEC.)
V
III
I
60
70
90
99
INTENSITY (dB nHL)

intensity at the intensity levels of 80 and 90 dB NHL (see Appendix C2).

As it turned out, the correlation coefficient values of latency for waves I, III and V showed a negative correlation coefficient. The correlation values for the waves were as follows: wave I -.37, wave III -.32 and wave v -.77. The -.77 value found for wave V is indicative of a strong negative correlation between stimulus intensity and latency.

The statistical results showed that the latencies of waves I, III and V are dependent on the various intensity levels. For instance, the ANOVA results for the group data revealed that there was a significant difference between the mean values for latency. For wave I, it was found to be [$F(2.84) = 21.38 > .05$] (see Appendix G2). Thus, the null hypothesis of no difference between the sample means is rejected for all the subjects.

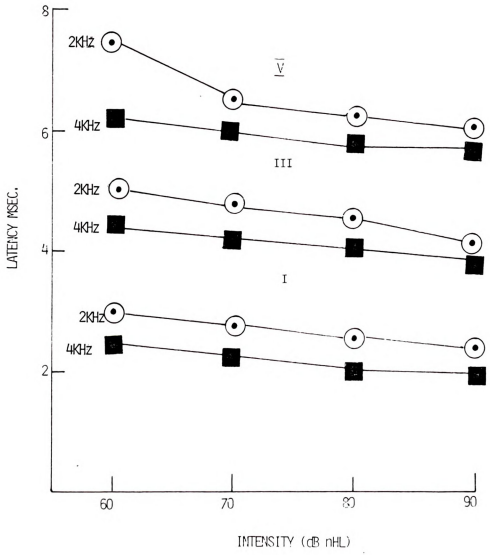
In another vein, we observe in Figure IV-3 the mean input-output latency function for waves I, III and V at 2000 and 4000 Hz. The salient features of this illustration are that as the intensity of the stimulus is increased, the latency of the evoked response decreases for the two frequencies. However, the magnitude of the decrease in latency was greater at 2000 Hz than the magnitude of the decrease at 4000 Hz (see Appendices B1 and B2). We also see that the range of differences between the latencies for wave 1 at 2000 and 4000 Hz were 0.7 to 0.5 msec when the stimulus intensity was increased from 60-90 dB nHL. It is also noted that the corresponding range of differences was greater for waves III and V (see appendix I). Observe also that while the slopes for the two frequencies were the same for wave I, they were different for waves III and V. Thus, the slope for wave I was .02 for both frequencies, but for wave III the slopes were -.04 and .03 at 2000 and 4000 Hz respectively. The slope for

wave V were .04 and .02 at 2000 and 4000 Hz respectively

Appendices J1 and J2 display the interpeak latency values and function for waves I, III and V respectively. We see in Appendix J2 that as intensity is increased I-III, I-V and III-V interpeak latencies increase slightly at the two frequencies. It is also noted that the change in interpeak latency was not linear. In certain definite regions the IPL remained constant or even reduced as a function of stimulus intensity. We also see that the IPL that involve wave I were smaller at 2000 Hz than the IPL at 4000 Hz.

We see in Appendix E1 the input-output amplitude function for waves I, III and V at 2000 Hz. Figure IV-4 through VI-6 depict the scatter diagrams and the linear regression lines for waves I, III and V. On the abcissa of the scatter diagram are plotted the intensity values, and on the ordinate the amplitude values. The logical conclusion from this representation is that each point on the graph is indicative of an individual subjects ABR response. In Figure IV-4 are two linear regression lines, A and B. Line A shows a perfect correlation since every 10 dB increases in intensity is accompanied by 100 (units) of increment in amplitude. The correlation coefficient for this imaginary line is I.0 because once we know the intensity level we can predict perfectly the value of the evoked responses. Line A is an idealized representation of the real world, in that in actual life situations we usually have situations in which the relationship is not perfect. Line B (a positive correlation of .21), on the other hand, represents real data. It reveals that with every 10 dB increase in intensity, we cannot predict the amplitude of the response on the vertical axis perfectly but rather, the score given by the line of "best fit" will

Figure IV-3. Mean input-output latency functions for waves I, III and V at 2000 and 4000 Hz.


8
6
4
2
LATENCY MSEC.
2KHz
4KHz
V
III
2KHz
4KHz
I
2KHz
4KHz
60
70
80
90
INTENSITY (dB nHL)

yield a fairly stable prediction. We also note that the line of "best fit" for waves III and V showed a weak positive correlation of .40 and .25, respectively (see Figure IV-5 and IV-6). Thus, the positive correlation coefficient showed that, in general, as the intensity of the stimulus increases the amplitude of waves I, III and V increase.

The exceptions were subject LB and MO in whom it was observed that he amplitude of waves I, III and V did not increase as a function of stimulus intensity. This was noted for subject LB at the intensity levels of 60 to 90 dB nHL for waves I and V, and at the intensity levels of 60, 70 and 80 dB nHL for wave III. The same observations were also noted for subject MO for waves I, III and V.

We also see that the amplitude of waves I, III and V of seven of the subjects increased above average or maximally as a function of stimulus intensity. Again, it is observed that the amplitude of subjects PW and SN increase below average and on the average or minimally as a function of stimulus intensity. Those whose amplitude increase maximally are classified as strong augmenters, while PW and SN are classified as moderate augmenters (see Appendix D3). The one-way and two-way analysis of variance was calculated for the mean amplitude of waves I, III and V to determine whether the various stimulus intensity levels have any significant effect on amplitude. The results showed that the difference between the sample means was statistically significant for all subjects. For example, the statistical differences between the mean values for the amplitude of wave I was [F(2.84) = II.74 > .05] (see Appendices Hl and H3). Thus for all of the subjects grouped together, amplitude increases as a function of stimulus intensity.

Figure IV-4. Individual data points for amplitude as a function of stimulus intensity for wave I at 2000 Hz. The method of least squares was used to draw the lines of "best fit." Intercepts and slopes can be found in Appendix F2. Line A is an imaginary regression line. Line B is the line of "best fit" for the individual data points.

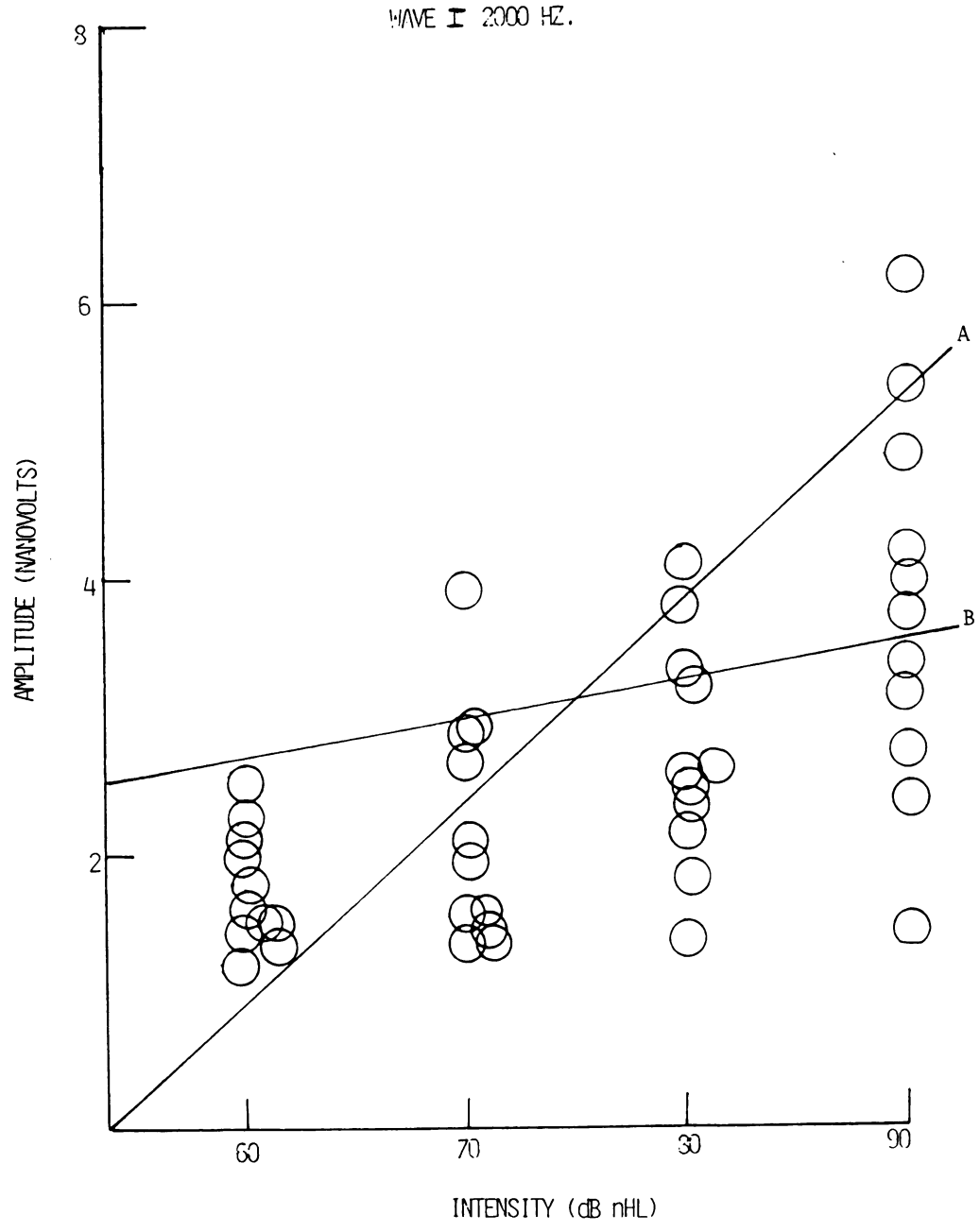

WAVE I 2000 HZ.
8
6
4
2
AMPLITUDE (NANOVOLTS)
A
B
60
70
30
90
INTENSITY (dB nHL)

Figure IV-5. Individual data points for amplitude as a function of stimulus intensity for waves III at 2000 Hz. The method of least squares was used to draw the lines of "best fit" Intercepts and sloped can be found in Appendix F2. Line A is an imaginary linear regression line. Line B is the line of "best fit" for the individual data points.

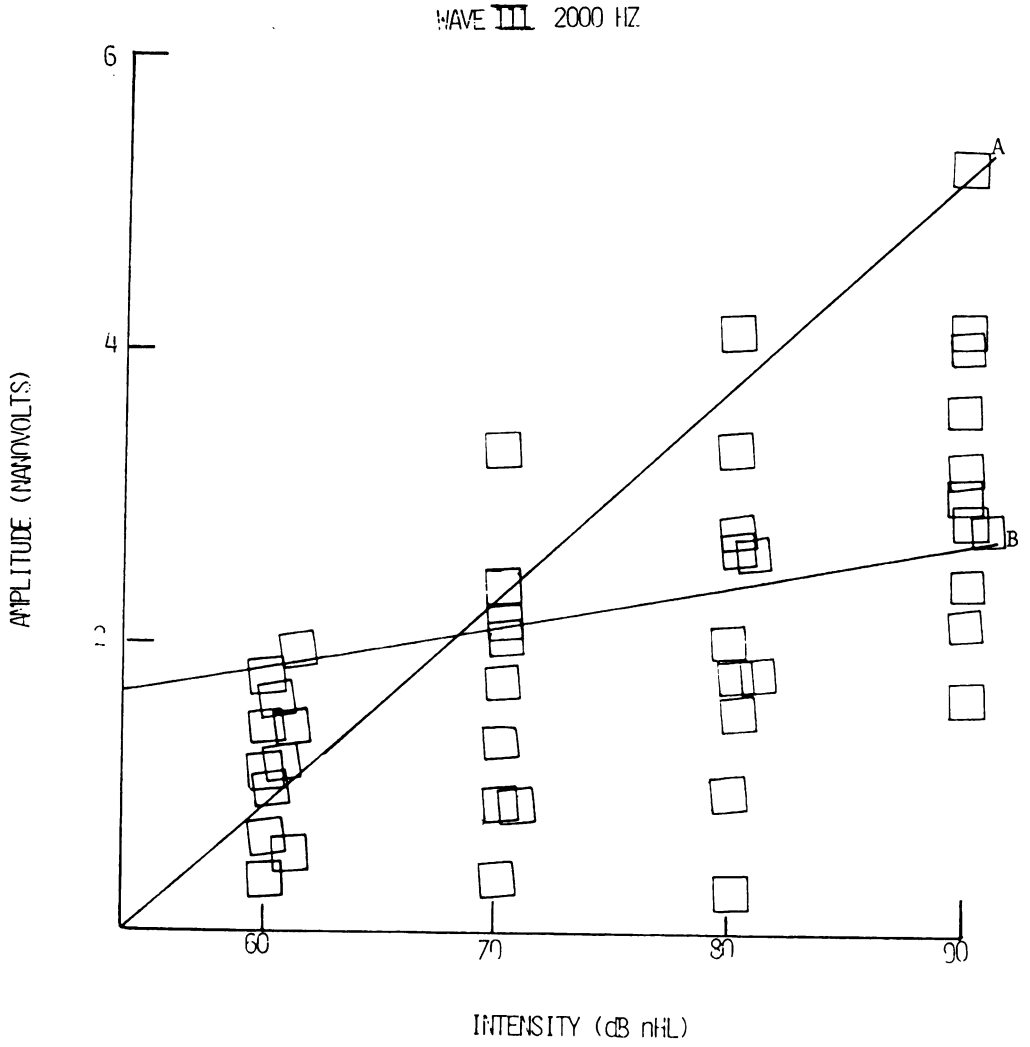
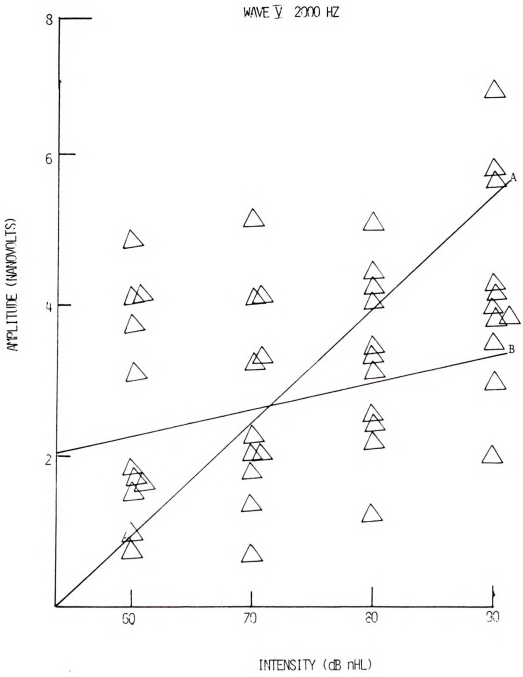

WAVE III 2000 HZ
6
4
AMPLITUDE (NANOVOLTS)
A
B
60
70
80
90
INTENSITY (dB nHL)

Figure IV-6. Individual data points for amplitude as a function of stimulus intensity for wave V at 2000 Hz. The method of least squares was used to draw the lines or "best fit." Intercepts and slopes can be found in Appendix F. Line A is an imaginary linear regression line. Line B is the line of "best fit" for the individual data points.


WAVE V 2000 HZ
8
6
4
2
AMPLITUDE (NANOVOLTS)
A
B
50
70
80
90
INTENSITY (dB nHL)

Viewing the input-output amplitude function for the individual data (see Appendix E2) and the scatter diagram in Figures IV-7 through IV-9, we note that, in general, as the intensity of the 4000 Hz stimulus increases, the amplitude of waves I, III and V increases. The notable exceptions were, as usual, subjects LB and MO for whom it was observed that the amplitude of the waves did not increase as a function of stimulus intensity. This was noted for subject LB for all three waves for the intensity levels of 60 and 80 dB. An exception was noted for waves I and III where the amplitude increases as a function of stimulus intensity at the 90 dB level. The same observations were noted for subject MO for all three waves.

The analysis of variance showed that the difference between the sample means was statistically significant for all subjects. For example, the statistical differences between the mean values for the amplitude for wave I was [$F(2.84) = 9.97 > .05$] (see Appendices H2 and H4).

Inspection of the mean data (see Appendix D) and the input-output amplitude functions for waves I, III and V (see Figure IV-10 through IV-13) revealed that the amplitude of the waves increase as the intensity of the stimulus is increased at 2000 and 4000 Hz. Observe, however, that the amplitude of the increase in amplitude was greater at 4000 Hz than the amplitude of the increase at 2000 Hz. It can also be observed that at high intensity levels the slope at 4000 Hz was greater than the slope at 2000 Hz for I and III. The slopes at 4000 Hz were as follows: I 6.5/dB, wave III 6.8/dB and at 2000 Hz it was found to be 6.3/dB for wave I and 5.7/dB for wave III.

Figure IV-7. Individual data points for amplitude as a function of stimulus intensity for wave I at 4000 Hz. The method of least squares was used to draw the line of "best fit." Intercepts and slopes can be found in Appendix F. Line A is an imaginary linear regression line. Line B is the line of "best fit" for the individual data points.

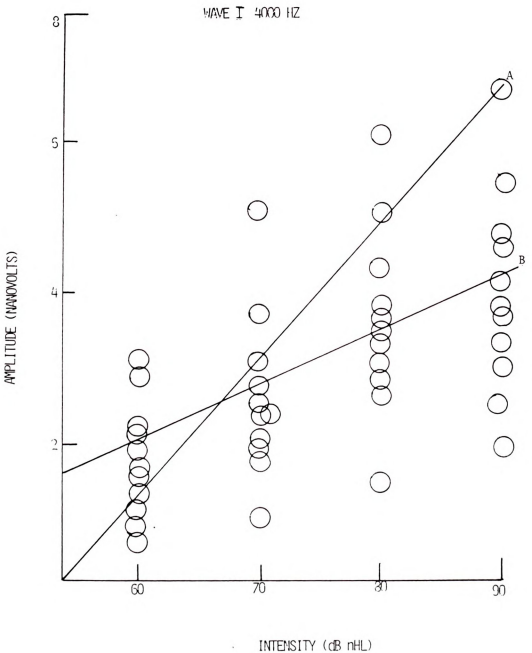

WAVE I 4000 HZ
8
6
4
2
A
B
60
70
80
90
AMPLITUDE (NANOVOLTS)
INTENSITY (dB nHL)

Figure IV-8. Individual data points for amplitude as a function of stimulus intensity for wave III at 4000 Hz. The method of least squares was used to draw the line of "best fit". Intercepts and slopes can be found in Appendix F2. Line A is an imaginary linear regression line. Line B is the line of "best fit" for the data points.

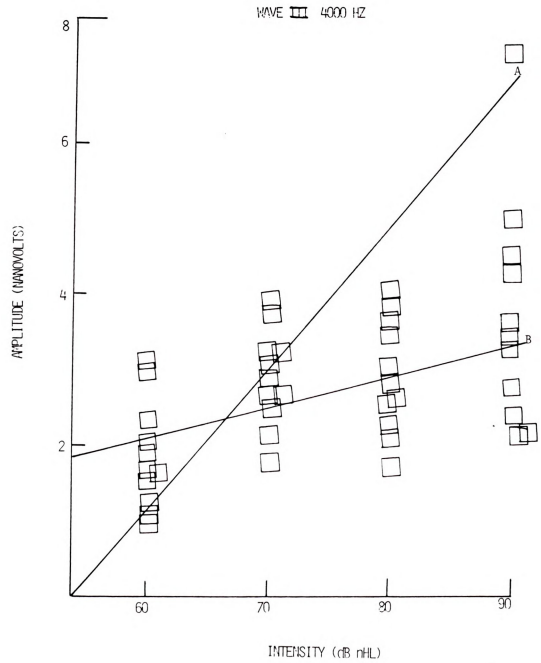
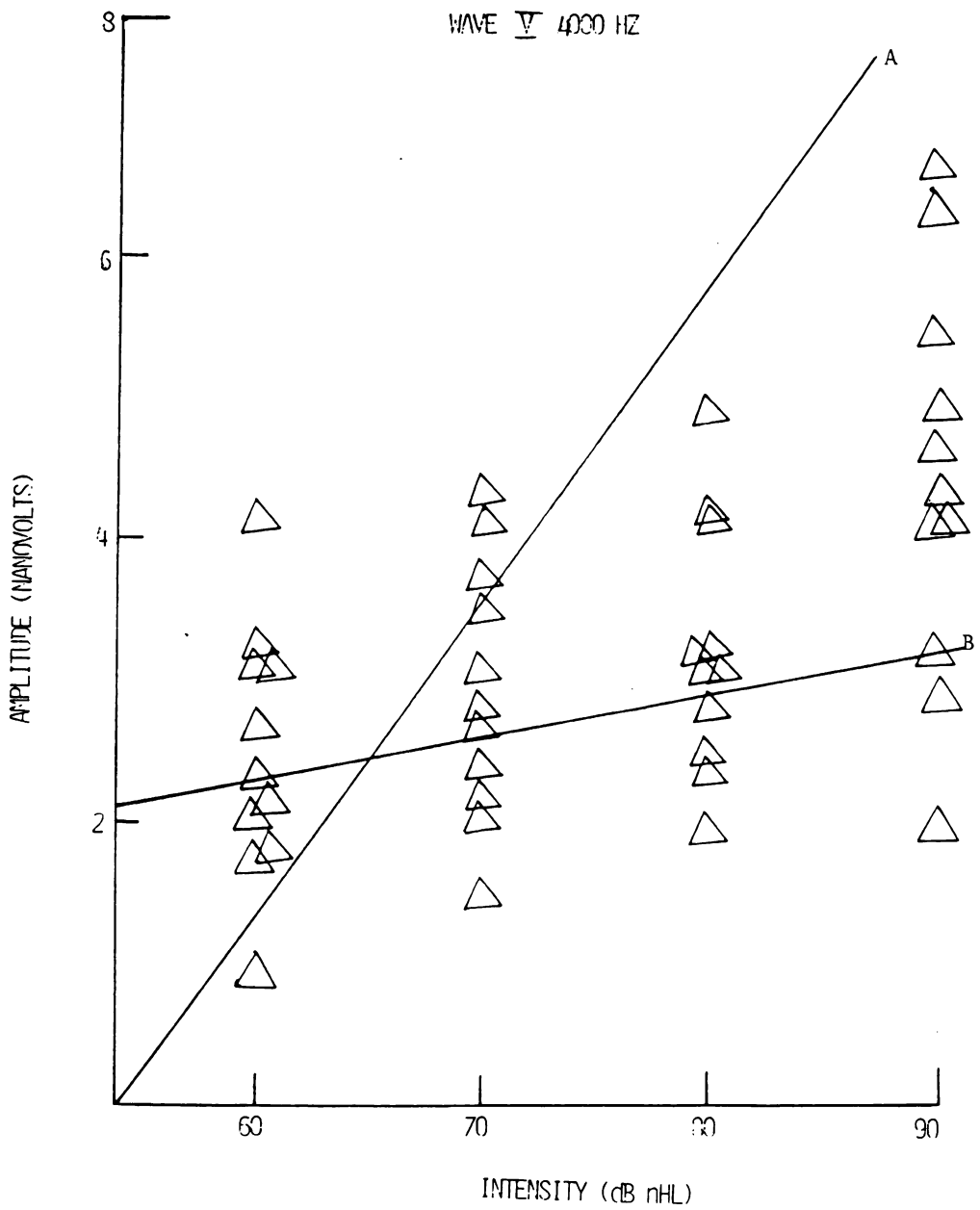

WAVE III 4000 HZ
8
6
4
2
A
B
60
70
80
90
AMPLITUDE (NANOVOLTS)
INTENSITY (dB nHL)

Figure IV-9. Individual data points for amplitude as a function of stimulus intensity for wave V at 4000 Hz. The method of least squares was used to draw the line of "best fit." Intercepts and slopes can be found in Appendix F2. Line A is an imaginary linear regression line. Line B is the line of "best fit" for the individual data points.


WAVE V 4000 HZ
8
6
4
2
A
B
60
70
80
90
AMPLITUDE (NANOVOLTS)
INTENSITY (dB nHL)

With regard to wave V, it was noted that the slope at 2000 Hz was greater than the slope at 4000 Hz. It was found to be 6.7/dB at 2000 Hz and 6.5/dB at 4000 Hz (see Appendix I).

We note that the ratio of the amplitude of wave I to that of wave V decreased with increasing stimulus intensity at 2000 and 4000 Hz (see Appendix K). It is also observed that the relative amplitude measure was greater at 4000 Hz than at 2000 Hz. Observe also that the ratios are greater than I.0 at both frequencies.

In summary, several studies have investigated stimulus parameters that affect ABR. The intensity of the stimulus has been studied and it was shown that as the intensity of the stimulus increases, the amplitude of the ABR increases; while latency decreases. While this has been a widely held concept, the question is whether this pattern is applicable to all subjects. It would appear that the amplitude relationship to intensity that was posited previously is not always true. The results of the literature on ABR revealed that there is an apparent void in the results as to whether the amplitude increase and latency-decrease as a function of stimulus intensity is applicable for all subjects. Indeed, it has not been determined whether all ABR waves adhere to the latency decrease, or the amplitude-increase relationship as a function of stimulus intensity. We also note that the effect of 2000 and 4000 Hz frequencies on waves I, III and V at high intensity levels have not been fully explored. The works of Petrie (1960), Buchsbaum and Silverman (1960) and Braden Haier and Space (1983) showed that there are individuals who are augmenters as well as individuals who are reducers.

Figure IV-10. Input-output functions for wave I at 2000 and 4000 Hz.

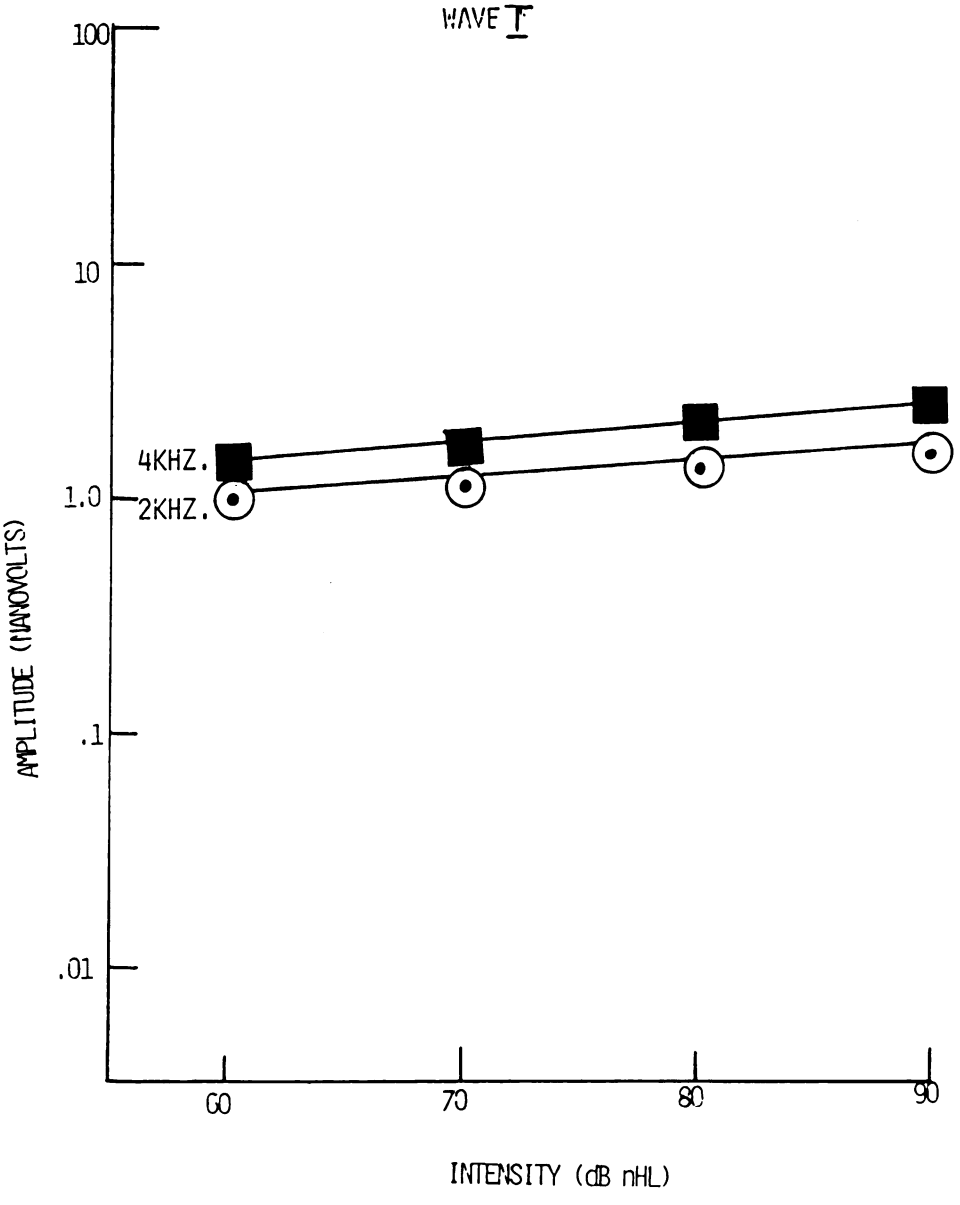

WAVE I
100
10
1.0
.1
.01
4KHZ.
2KHZ.
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)

Figure IV-11. Input-output amplitude functions for wave III at 2000 Hz and 4000 Hz.

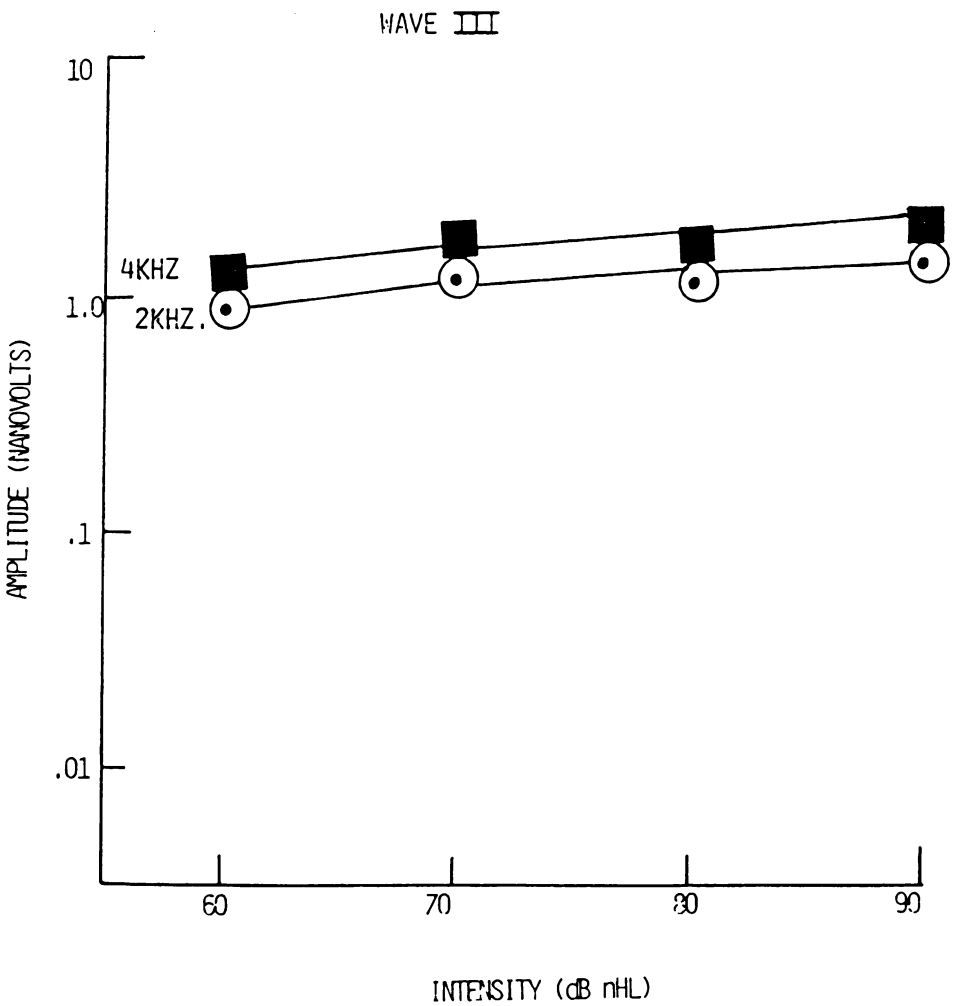
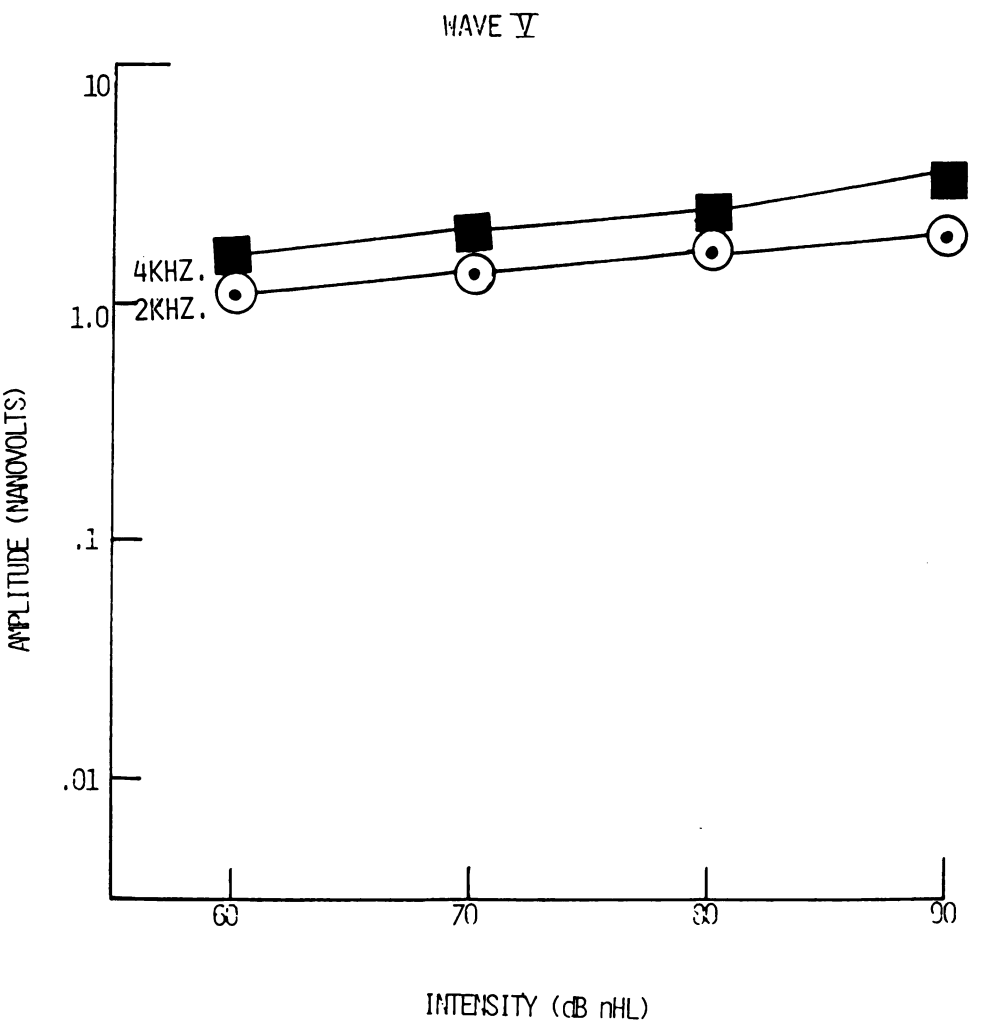

WAVE III
10
1.0
.1
.01
4KHZ
2KHZ.
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)

Figure IV-12. Input-output amplitude functions for wave V at 2000 and 4000 Hz.


WAVE V
10
1.0
.1
.01
4KHZ.
2KHZ.
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)

There have been no investigation conducted as to augmenters and reducers for ABR data. These fundamental observations formed the basis of this study. It can be observed from the results of this study that individuals differ in their response to filtered clicks at different intensities, in that there are individuals who could be classified as strong augmenters (N=7) as well as individuals who could be classified as reducers (N=2). The present study also demonstrate that audiometric frequencies at 2000 and 4000 Hz have an effect on waves I, III and V as the intensity of the acoustic stimulus is increased, a not too uncommon finding. Thus, it can be noted that only seven (64%) of the subjects could be classified as fitting the classical model of amplitude increase due to stimulus increase.

CHAPTER V

DISCUSSION

The results of the literature in the area of auditory brain stem evoked potentials has been shown to be valuable in assessing normal as well as abnormal auditory functioning. The amplitude and latency of the ABR have been widely investigated, and it has been demonstrated that as the intensity of a stimulus is increased, the amplitude of the ABR waves will increase, while latency decreases (Romano and Williston, 1970; Rossi, solero and Pira, 1982; Moore, 1971). It would appear, however, that the amplitude relationship to intensity that we have come to accept does not always occur in all clinical test situations.

This investigation was conducted to determine whether this pattern is applicable to all individuals, and whether all ABR waves adhere to the latency-decrease, and amplitude-increase relationship as a function of stimulus intensity. Secondly, it was the objective of this investigation to explore the relationship of augmenting-reducing potential as a function of two audiometric test frequencies and the components of the ABR as the intensity is altered.

We observed an inverse relationship between the intensity of the stimulus and latency, in that it was seen to decrease as a function of stimulus intensity. The exceptions noted were subjects PW and LB, where the latency of the waves remained constant as a function of stimulus intensity.

We also noted that latency was more systematic and less variable in both individual and group data. It can be seen in the scatter diagram for latency in figures IV-I and IV-2 that the points lie close to the line of "best fit". This showed that the relationship between intensity and latency is less variable. The logical sequel to this observation is that one can predict the value of latency quite accurately on the basis of the changing values of intensity.

In another vein, it was observed that ABR latencies decreased as the frequency of the filtered clicks were increased. We see that at lower intensity levels of 60 dB nHL there was a clear separation of the 2000 Hz and the 4000 Hz data. At high stimulus intensity levels (90 db nHL) however the difference between ABR latencies at both frequencies was slight (see Appendices B1 and B2 and figure IV-3). Why are the latency values of waves I, III and V greater in magnitude for lower frequency filtered clicks (2000 Hz) at low levels of intensity? We have no direct evidence to answer this question. However, a plausible speculation is that at high intensity levels of 90 dB nHL, a larger portion of the cochlea is being stimulated. We also know from the work of Davis (1976) that the basal part of the cochlea tends to evoke better synchronized discharges than its apical part. Therefore, at high stimulus intensity levels, the ABR evoked by 2000 Hz filtered click might have originated from a part of the cochlea more basalward than the portion most sensitive to 2000 Hz at lower intensity levels. With a decrease in stimulus intensity to 60 dB, however, the more apical part of the cochlea is more selectively stimulated and presumably plays a major part in evoking the ABR. This will cause an increase in latency because of the longer travel time along the cochlea partition.

We observed, also, that the interpeak latencies (IPL) that involve wave I were smaller at 2000 Hz than the IPL at 4000 Hz (see Appendix J). We do not have a definitive answer to this observation, but we can make certain speculative statements. There is some evidence from the work of Stockard, et al. (1979) that acoustic stimuli with lower frequency components which activate more apical portions of the cochlea produce smaller IPLs that involve wave I than do higher frequency acoustic stimuli that activate the basal cochlea.

It is also noted that a direct relationship exists between the amplitude of the ABR and stimulus intensity in that it did increase as a function of stimulus intensity (see Appendices E1 and E2). The exceptions noted were subjects MO and LB for whom the amplitude of the ABR remained constant, and, even decreased as a function of stimulus intensity.

Why is it that the amplitude of the ABR increases as the intensity of the stimulus is increased? The most cogent explanation is that the inner hair cells might respond to only sounds of high intensity. The work of Kiang et al. (1965) revealed that while the actual threshhold of the receptors is in doubt, there most definitely exist fibers in the auditory system that respond only to tones of high intensity. Pickles (1982) also reported that

> some nerve fibers did not saturate in a sharply defined manner, but went on increasing at high intensities. These fibers also tend to have high threshholds than others. The proportion of such fibers is only a few percent.

Can we then call subjects MO and LB reducers? The answer to this question is yes, in that it was observed that the amplitude of waves I, III and V

for these subjects did not increase as a function of stimulus intensity. Why is it that the amplitude of the ABR decreases as the intensity of the stimulus is increased? One factor to be considered in answering this question is the complex central descending auditory inhibitory system. We know from the work of Whitefield (1967) that the stimulation of the dorsal nucleus of the lateral lemniscus causes an inhibitory effect on the cochlear nucleus. We also know from the work of Chin, Killiam and Killiam (1965) that the stimulation of the recticular formation causes a decrease in the amplitude of evoked responses at the medial geniculate body. In addition, Livingstone (1958) reported that the reticular system seems to exert a tonic descending inhibitory influence on ascending activity both in the lemniscal pathways and its own ascending fibers. We do not know exactly why the decrease in amplitude as a function of stimulus intensity was observed for only two subjects. However, the limitations inherent in ABR audiometry as a diagnostic tool continued to be explored. For example, Worthington and Peters (1979) found that there was no recordable ABR for their four patients even though they have quantifiable hearing with no neurological involvement. It is likely that the complex central descending inhibitory auditory system explanation is not applicable to all subjects. It is our belief that the decrease in the amplitude of the ABR as a function of stimulus intensity reflects the activity of the central descending auditory system.

We saw in Figures IV-6 and IV-9, the scatter diagrams for wave V at 2000 Hz and 4000 Hz, respectively. We do know that the scatter diagram gives an indication of both the magnitude of the correlation coefficient and the sign of such a relationship. Fundamental to this idea is the fact

that before the correlation coefficient is computed, the data must meet two basic assumptions. The first of these assumptions is that the off-sets in the scatter diagram must fall on a straight line, the linear regression line. The second assumption is that the data must possess homoscedasticity, the variances of the off-sets must be equal. It can be seen that the scatter diagram meet the aforementioned assumptions, in that the off-sets in the two figures fall on the line of "best fit". In addition, the data for wave V possessed homoscedasticity, in that the variances or off-sets tended to be equal. Within this context, one is correct in supporting the view expressed by Stockard and Stockard (1983) that wave V is more stable and is, therefore, the most commonly used component in testing both newborns and adults, while using amplitude and peak latency criteria for otologic diagnosis.

It must be emphasized that even though there was constancy of input-output functions for a majority of the subjects, yet in certain definite regions, we see some amount of variability. A careful inspection of the individual data (see Appendices D1 and D2), revealed that variability was present, not only between subjects but, also, within subjects. However the variability was obliterated when we plotted the group data. These results confirmed the findings of Sohmer (1983) as well as several others who also found that the amplitude of the ABR show greater variability. The work of Starr and Achor (1975) and Row (1983), however, revealed that the variability of the amplitudes of ABR can be reduced by using the ratio of the amplitude of wave V to that of wave I. This ratio has been found to be greater than I.0 for normal subjects (see Appendix K).

The results of this investigation further demonstrated that the amplitude of wave V tended to saturate more quickly at 70 and 80 dB nHL at 4000 Hz than at 2000 Hz (see Figure IV-12). The reason for the saturation effect at 4000 Hz and the increasing amplitude at 2000 Hz is not fully understood. However, the work of Picton et al. (1981) suggests that there may be some increased syncronization of post-synaptic neural activity in the brain stem with increasing intensity of the lower frequency response; this effect not being available to high frequency responses which are already fully synchronized. It is to be noted that the responses at 4000 Hz are only slightly greater in amplitude than at 2000 Hz, and are not statistically different. Note that both do increase in amplitude as a function of increasing levels of the stimuli. This increase is perhaps not readily seen since this is a logarithmic scale. The logarithmic scale was used in order to partially compensate for the rapid increases in amplitude at the various stimulus intensity levels that are seen in linear plots.

The limitations inherent in this investigation relate mainly to the subjects and the stimulus parameters that of necessity were either controlled or held constant for the purpose of this study. For instance the utilization of only female subjects may limit our generalization to other subjects. It is gratifying to note, however, that the results of this investigation are within reasonable bounds; for it was observed that about 80% of the subjects in this study can be classified as augmenters. Also, we have reason to believe that women have a greater tendency to be augmenters. Petrie (1960) has clearly demonstrated this in his investigation based on Figural Kinesthetic After-effects.

Secondly, the use of only two frequencies (2000 and 4000 Hz) may limit our generalization to other frequencies. However the work of Moore (1983) and Kodera et al. (1977) suggest that the findings in this investigation can be generalized up to 8000 Hz and perhaps to 1000 Hz, provided identifiable responses are obtained, and high intensities (greater than about 60 dB nHL) are avoided. Durrant (1983) reported that

> "relatively sharp tuning curves are obtained at intensities below 5-25 dB SL. But at intensities more than 5-15 dB SL, tuning greatly deteriorates and their tip is lost while their tails tend to become abnormal."

We also held repetition rate constant at 10/sec. There is evidence to suggest that a rate of 10/sec is within a reasonable margin of acceptability for obtaining ABR. We know from the work of others (Moore, 1971; Row, 1978; Don, Allen and Star, 1978; Moore, 1983) that increasing the repetition rate also increases the latency, but decreases the magnitude of the ABR waves. Such an effect is more pronounced for repetition rates greater than 10/sec even though a latency increase and an amplitude decrease do not go undetected at rates below 10/sec.

It is evident from the above findings that we can make reliable predictions to latency and amplitude under the conditions of this experiment. Thus, the latency of the ABR does decrease as the stimulus intensity is increased, and as the audiometric frequency is increased. Also, the amplitude of the ABR increase as the stimulus intensity is increased and at increasing audiometric test frequency. There is variability not only between subjects but also within subjects in the amplitude and latency of the ABR, but more so for the former.

Interestingly, we see from this variability a corpus of individuals who can be classified variously as strong augmenters (N=7) moderate augmenters (N=2) and reducers (N=2). In the light of these findings, it would seem that response variability should be an important consideration in the design of evoked potential studies and in clinical consideration in the design of evoked potential studies and in clinical application of experimental findings.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Since 1970 the ABR technique has emerged as a vital adjunct to the clinical test battery of the otologist, audiologist, neurologist, neurosurgeon and the pediatrician. The ABRs are elicited by click-like transients which are generated by driving an earphone with rapid rise time, brief duration electrical pulse.

We have learned that stimulus parameters such as time, frequency and intensity have an influence on the amplitude and latency of the ABR. Among these, intensity has been shown to have a direct influence on the amplitude and an indirect influence on the latency of the response. Thus, as the intensity of the stimulus is increased, the amplitude of the ABR increases, while latency decreases. While this notion has been widely accepted, the question is whether this pattern is applicable to all subjects. It would appear that the amplitude relationship to intensity that we observe is not always true. Studies using Figural Kinesthetic After-effects and visually evoked potentials revealed that there individuals who can be classified as augmenters that is those subjects in whom the amplitude of the ABR increased as function of stimulus intensity, as well as individuals who are reducers. Those whose amplitude of the ABR either decreased or remained constant as a function of stimulus intensity. However, there is no information in the results of the literature on the relationship or augmenting-reducing phenomenon and the components of the ABR as a function

of intensity. We also know from he work of Worthington and Peters (1979) that there are persons with normal hearing with no neurological evidence of brain stem dysfunction in whom the ABRs were either absent or showed threshholds inappropriate to their audiometric configurations. The absence of the ABR was attributed to lack of neural activity, block of nerve conduction, or synchrony and/or disruption of synchrony.

The present investigation was designed to determine whether the intensity relationship to amplitude and latency that we observe in the clinic and delineated by the results of the literature is applicable to all subjects, and whether there are individuals who are augmenters, as well as individuals who can be classified as reducers. Eleven audiometrically normal subjects with hearing no greater than 10 dB HTL (re: ANSI 1969) were used in the study to answer the following questions:

(1) The amplitude of waves I, III and V of ABR do not increase in a systematic manner as the intensity of the stimulus is increased.

(2) The latency of waves I, III and V of the ABR do not decrease in a systematic manner as the intensity of the stimulus is increased.

(3) The magnitude of the increase in amplitude as the intensity of the stimulus is increased is not the same for all individuals.

(4) Audiometric test frequency has no effect on the amplitude of waves I, III and V of the ABR as intensity is increased.

(5) Audiometric test frequency has no effect on the latency of waves I, III and V of the ABR as intensity is increased.

Filtered clicks at 2000 Hz and 4000 Hz were presented to the right ear of each subject at intensity levels of 60, 70, 80, and 90 dB nHL. The repetition rate was held constant at 10/sec, with an analysis time of 10

msec and an average of 1024 responses. The band pass of the filtered click was 20-3000 Hz, and a dwell time of 100 microseconds was used, utilizing 250 data points. The electrode placement was (C_2 - M_2) with the forehead (FP_2) serving as patient ground.

A total of fifty subjects were tested in a pilot study so as to eliminate independent and dependent variables having minimal influence on the resultant data or where the factorial design would have resulted in unwielding data sets. As expected, identifiable ABR responses were obtained from all eleven subjects. We also see a direct relationship in the group data between the amplitude of the ABR and stimulus intensity and an inverse relationship between intensity and latency. The results of the statistical analysis revealed that significant differences exist between the various intensity levels and amplitude for waves I, III and V of the ABR. Therefore, the null hypothesis that amplitude does not always increase as a function of stimulus intensity is rejected for all nine of the eleven subjects. Secondly, the null hypothesis that he latency of the ABR does not always decrease as a function of stimulus intensity is also rejected for all subjects except two. Audiometric test frequencies have also been shown to have effect on the amplitude and latency of the ABR as the intensity of the stimulus is increased. However, an inspection of the individual datum revealed that the results, while consistent, yet, a certain degree of variability exists, not only between the subjects but also within them; but more so for amplitude. From the variability data, however, we see a cohort of individuals who could be classified as strong augmenters (N=7), moderate augmenters (N=2) and reducers (N=2). Thus, it can be observed that only seven (64%) of the subjects could be classified

as fitting the classical model of amplitude increase due to stimulus increase.

Suggestions for Additional Research

In view of these results, the following recommendations are made as areas of additional investigation:

(1) Recording of ABR using electrode placements other than C_z-M_2, such as C_z-A_2 to determine whether electrode placement might be a factor in deriving the above conclusions.

(2) Studies of the design need to be extended to male subjects since only female subjects were used in the present study.

(3) Stimuli with frequencies other than 2000 and 4000 Hz should be used with the same or different intensity levels employed in this study.

(4) A study similar to the present investigation should be conducted using different repetition rates so as to determine whether changes in latency and amplitude values interact with intensity and frequency as a function of various repetition rates.

(5) The use of other forms of stimuli with no more frequency specificity such as tone bursts or tone pips in similar research is recommended.

(6) It may be worthwhile to conduct an investigation of this nature on a select group of patients with conductive hearing loss, sensorineural hearing loss retrocochlea, brain stem pathology and central auditory processing disorders.

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REFERENCES

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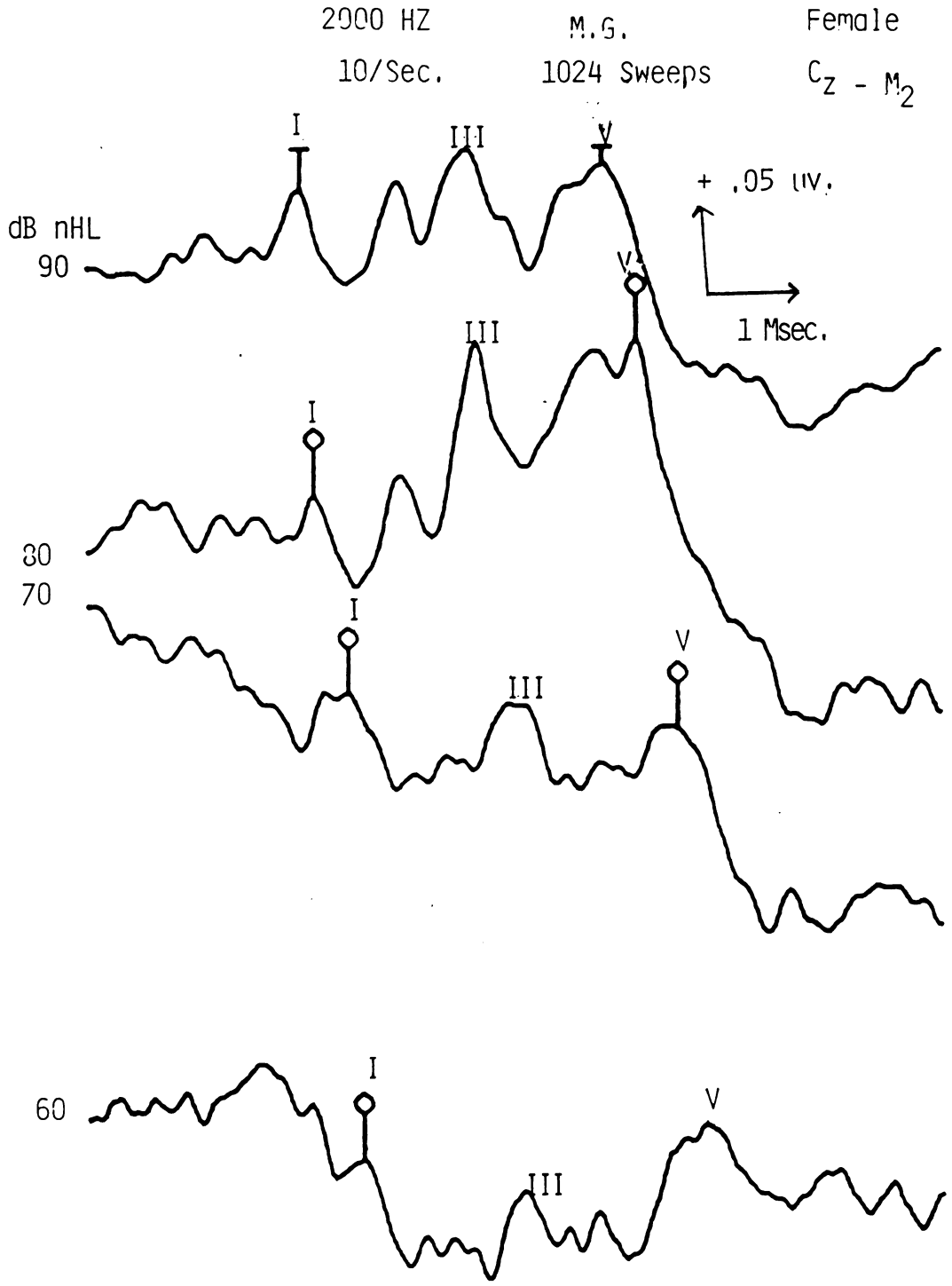
Appendix A1. Auditory brain-stem evoked responses of eleven subjects to filtered clicks at 2000 Hz presented at varying intensity levels of 60, 70, 80 and 90 dB nHL. Vertical calibration is .05 uv. Analysis time = 10 msec.


2000 HZ
10/Sec.
M.G.
1024 Sweeps
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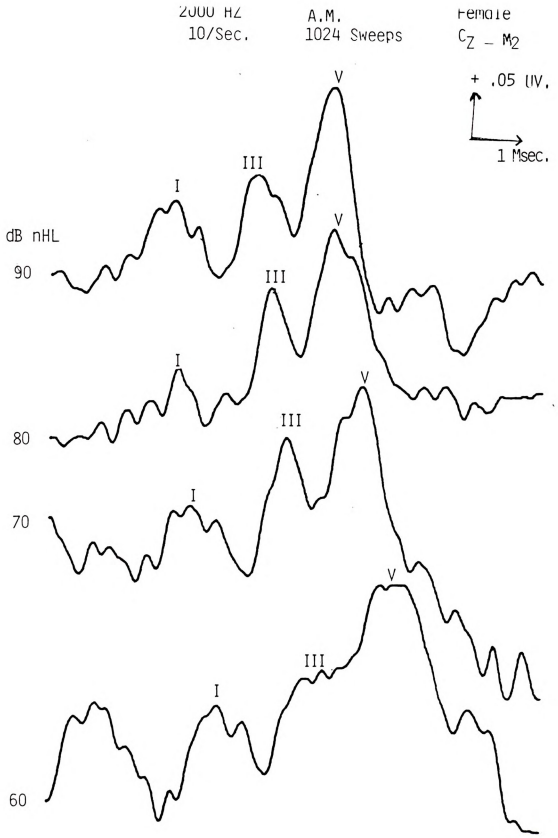
dB nHL
90
80
70
60
I
III
V
+ .05 uv.
1 Msec.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A.M.
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dB nHL
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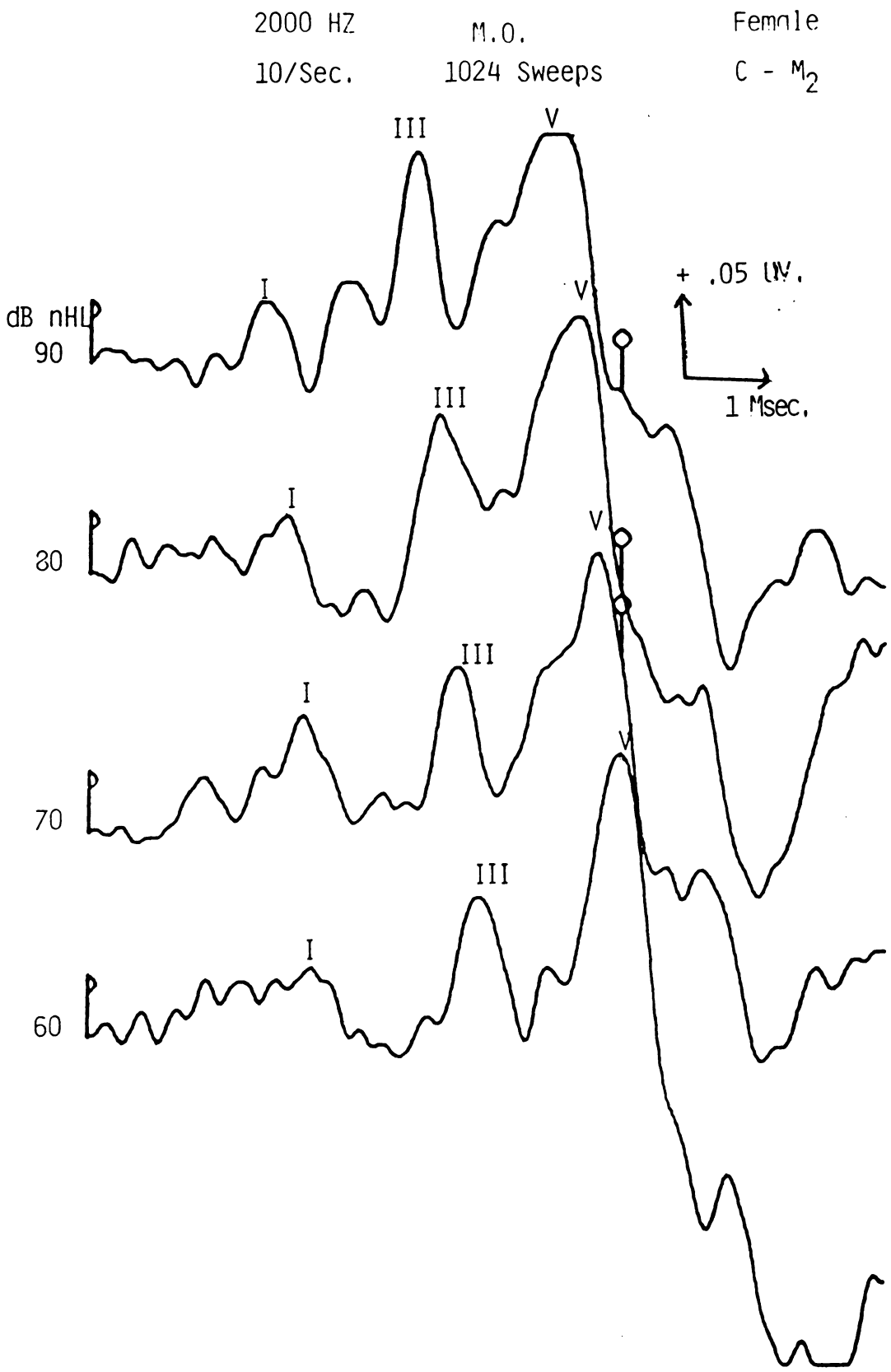
80
70
60
I
III
V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M.O.
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C - M2
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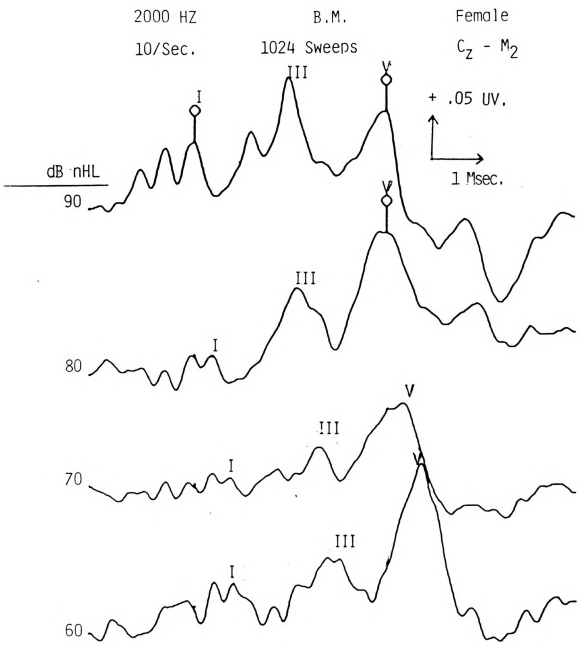
70
60
I
III
V
+ .05 UV.
1 Msec.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B.M.
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dB nHL
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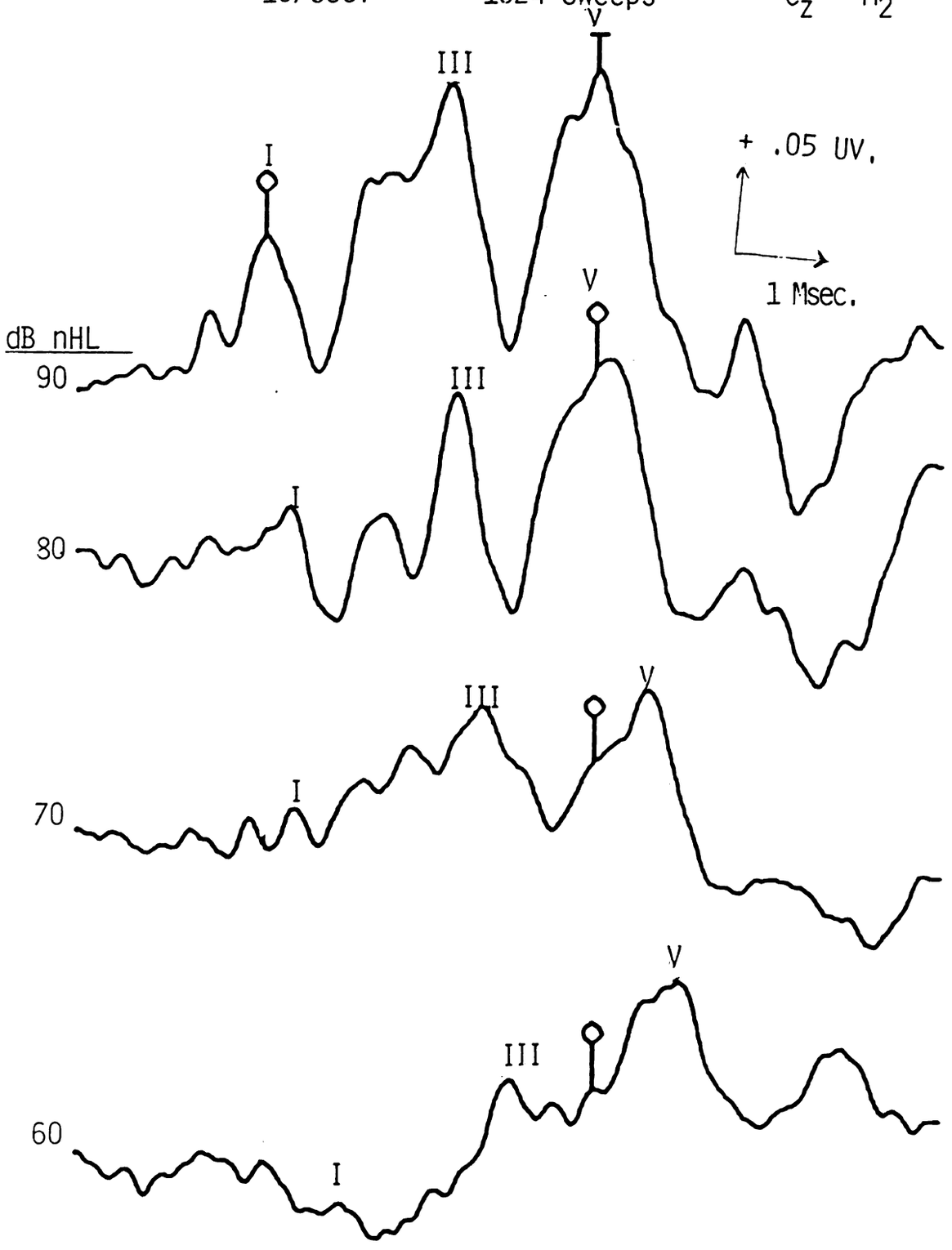
80
70
60
I
III
V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10/Sec.
L.B
1024 Sweeps
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C_z - M_2
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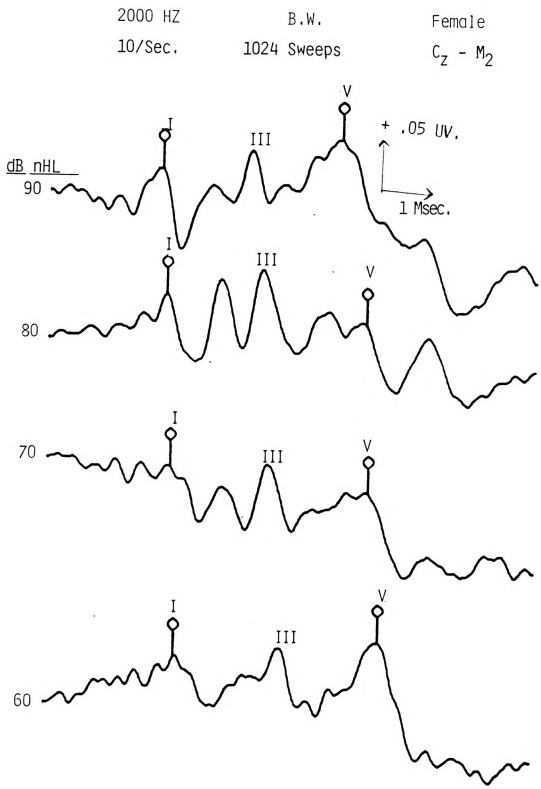
1 Msec.
dB nHL
90
80
70
60
I
III
V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10/Sec.
B.W.
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dB nHL
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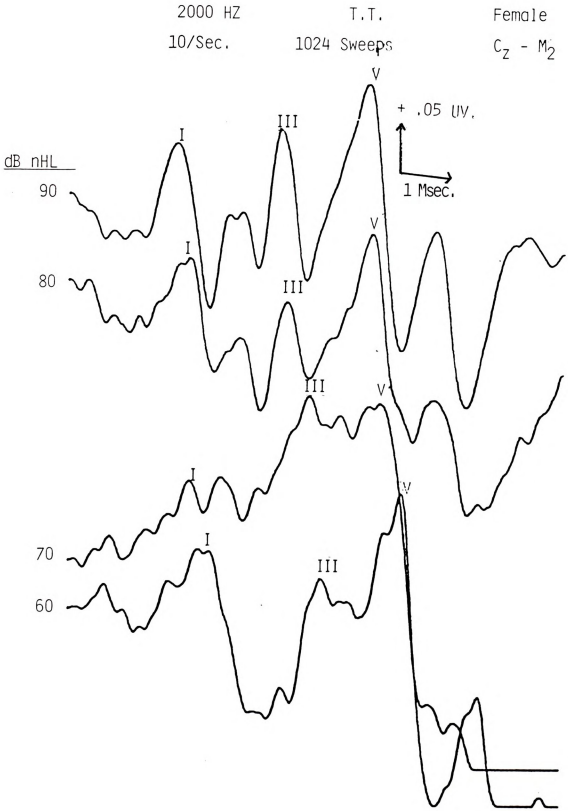
80
70
60
I
III
V
+ .05 UV.
1 Msec.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T.T.
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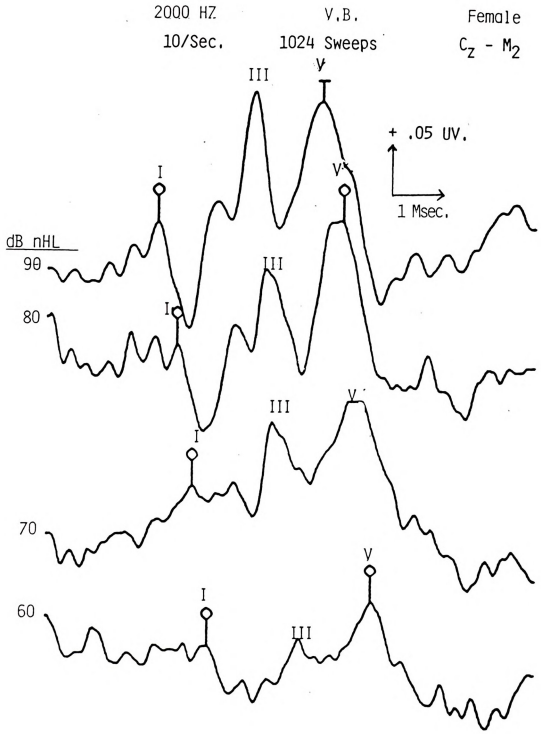
dB nHL
90
80
70
60
I
III
V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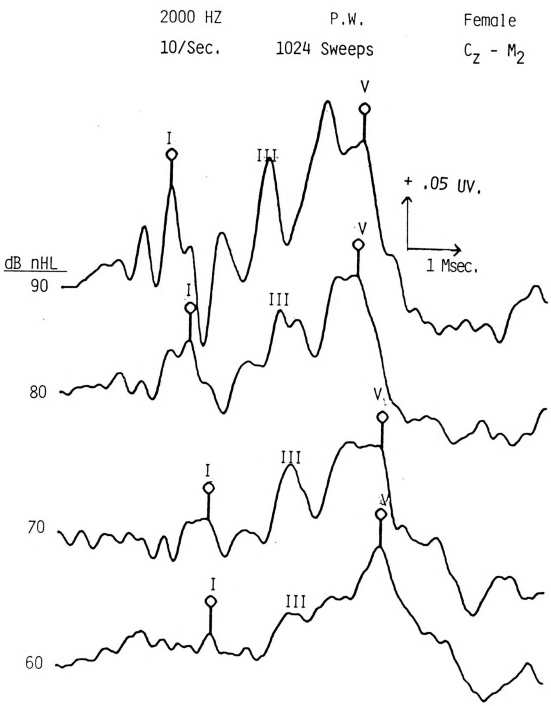
60
I
III
V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P.W.
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dB nHL
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80
70
60
I
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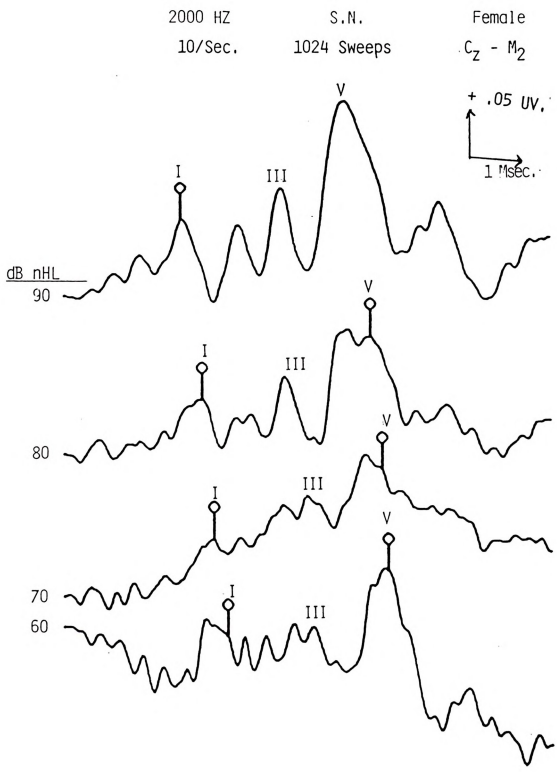
V
+ .05 UV.
1 Msec.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10/Sec.
S.N.
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1 Msec.
dB nHL
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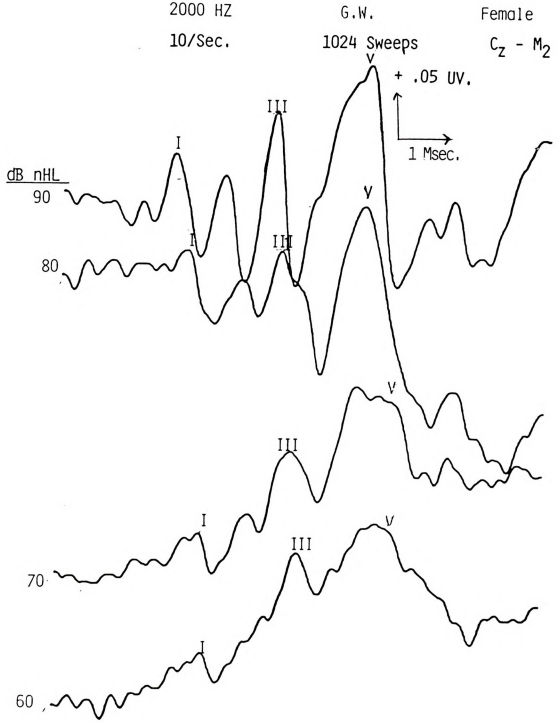
80
70
60
I
III
V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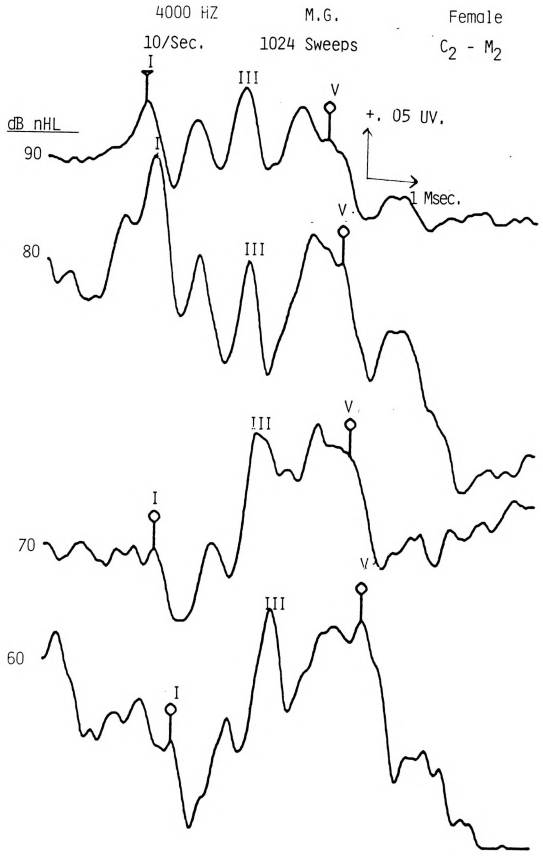
10/Sec.
G.W.
1024 Sweeps
Female
$C_z - M_2$
+ .05 UV.
1 Msec.
dB nHL
90
80
70
60
I
III
V

Appendix A2. Auditory brain-stem evoked responses of eleven subjects to filtered clicks at 4000 Hz at varying intensity levels of 60, 70, 80 and 90 dB nHL. Vertical calibration is .05 uv. Analysis time - 10 msec.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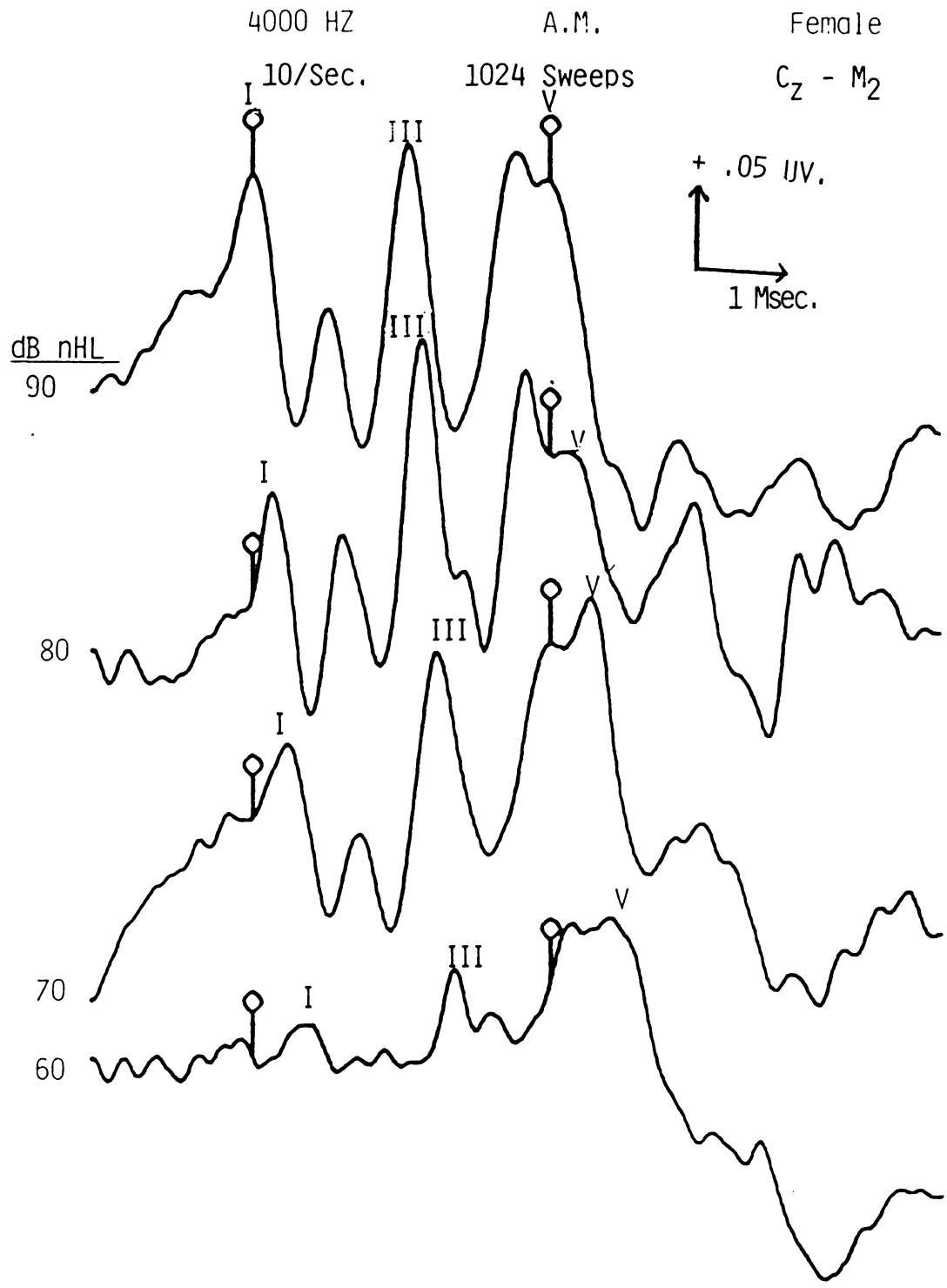
10/Sec.
M.G.
1024 Sweeps
Female
$C_2 - M_2$
dB nHL
90
80
70
60
I
III
V
+. 05 UV.
1 Msec.


4000 HZ
A.M.
Female
10/Sec.
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C_Z - M_2
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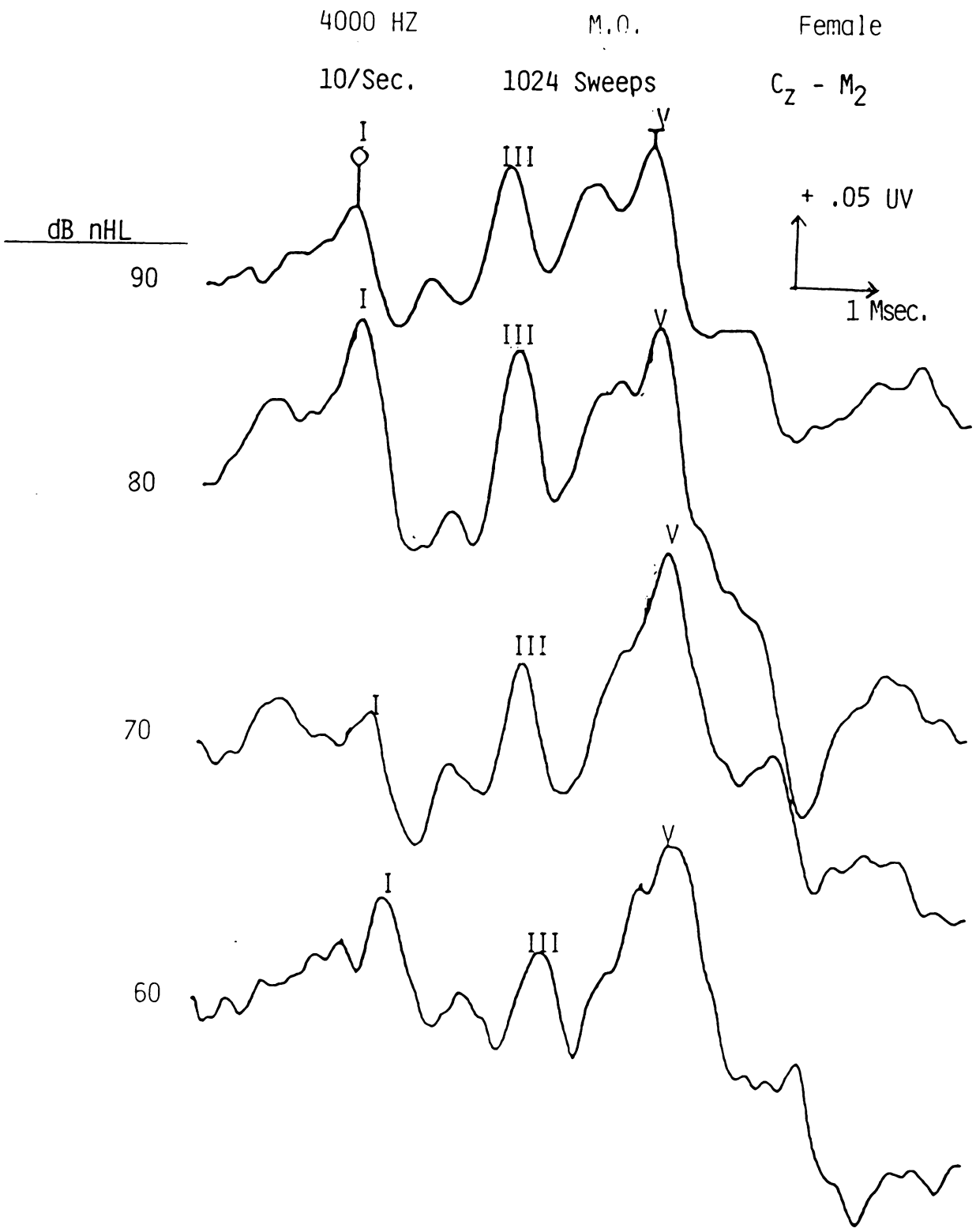
1 Msec.
dB nHL
90
80
70
60
I
III
V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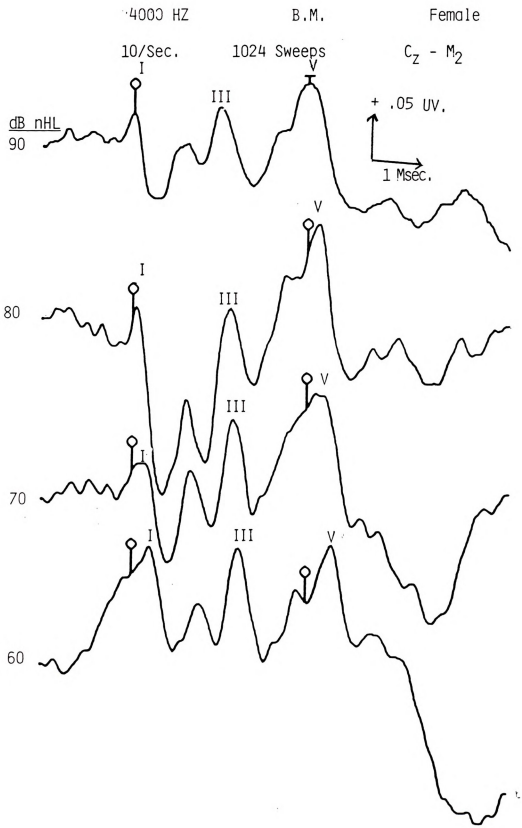
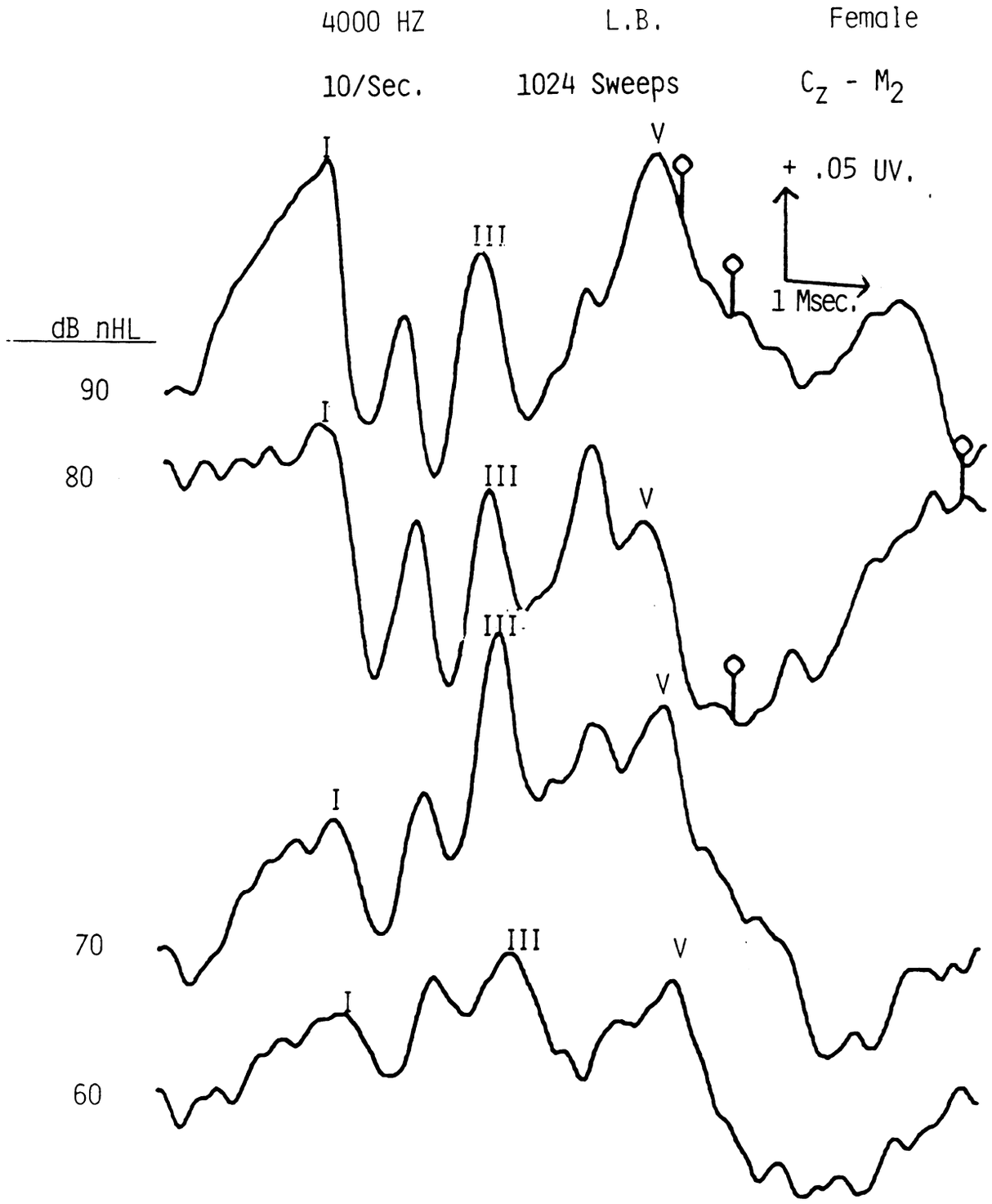
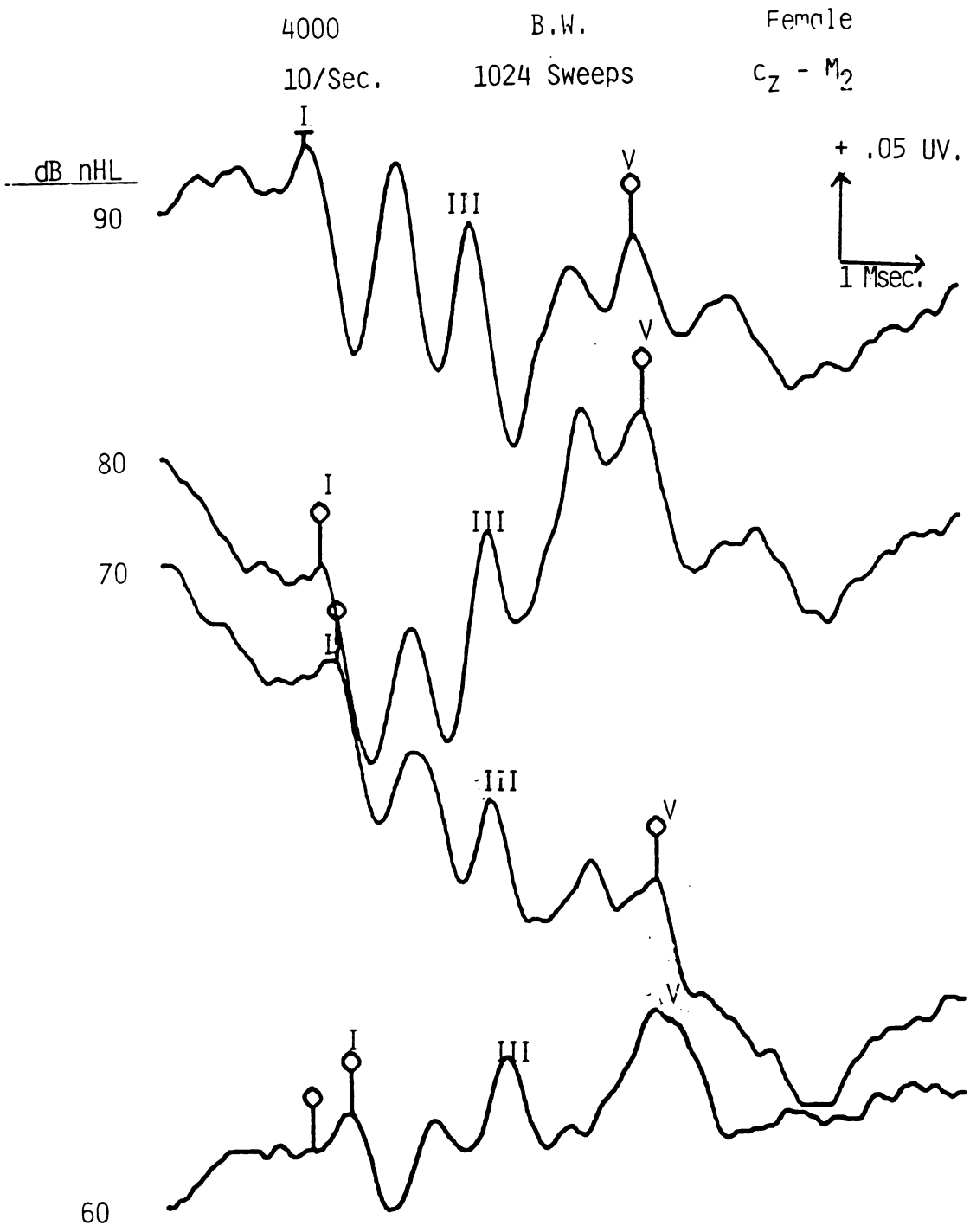
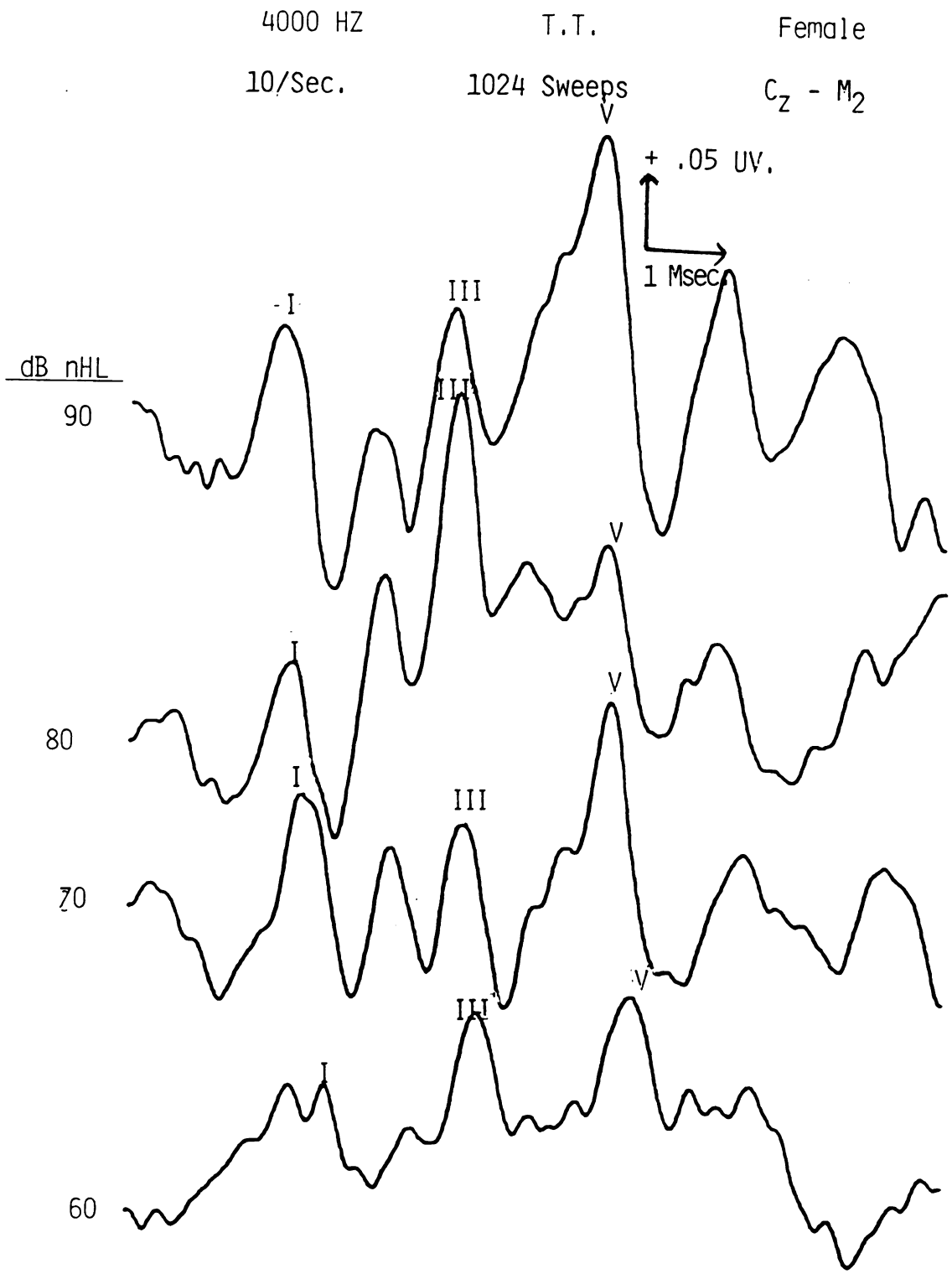
M.O.
Female
10/Sec.
1024 Sweeps
C_z - M_2
+ .05 UV
1 Msec.
dB nHL
90
80
70
60
I
III
V


4000 HZ
B.M.
Female
10/Sec.
1024 Sweeps
$C_Z - M_2$
dB nHL
90
80
70
60
I
III
V
+ .05 UV.
1 Msec.

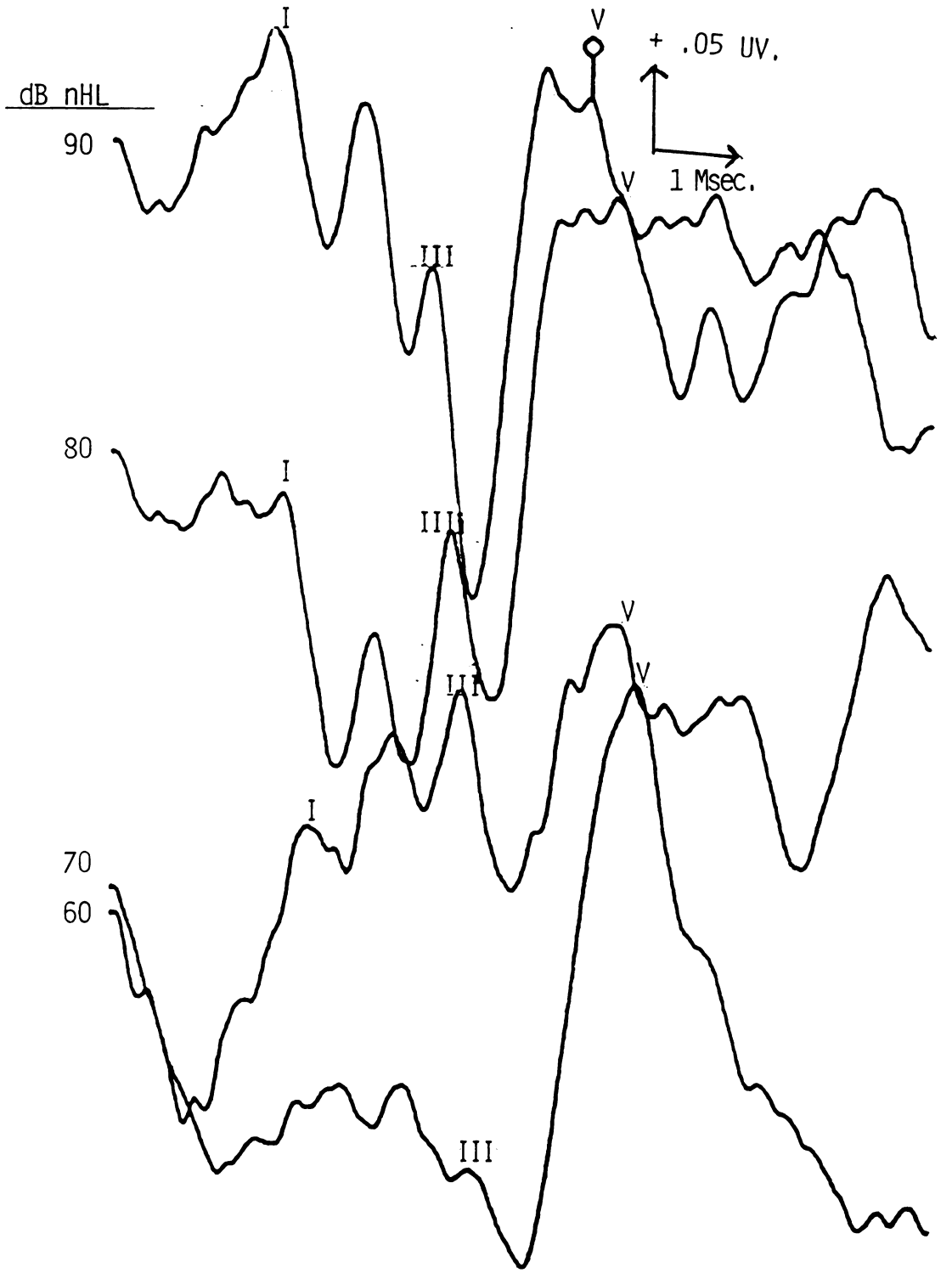

4000 HZ
L.B.
Female
10/Sec.
1024 Sweeps
C_Z - M_2
+ .05 UV.
1 Msec.
dB nHL
90
80
70
60
I
III
V


4000
10/Sec.
B.W.
1024 Sweeps
Female
$C_Z - M_2$
dB nHL
90
80
70
60
I
III
V
+ .05 UV.
1 Msec.


4000 HZ
T.T.
Female
10/Sec.
1024 Sweeps
C_Z - M_2
+ .05 UV.
1 Msec.
dB nHL
90
80
70
60
I
III
V

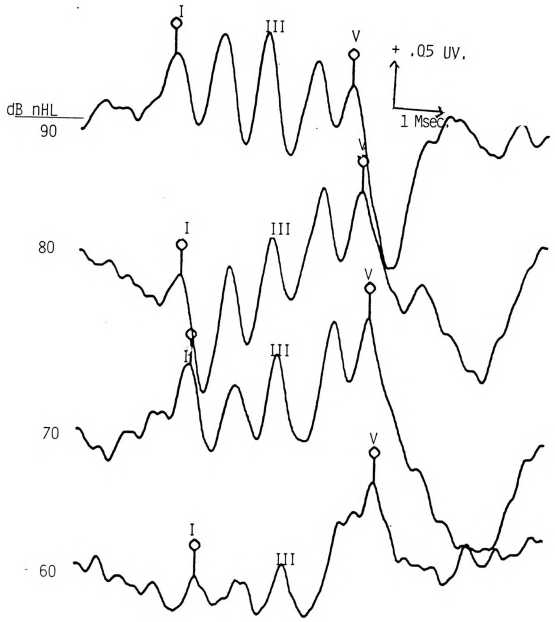
4000 HZ V.B. Female

10/Sec. 1024 Sweeps $C_z - M_2$



4000 HZ P.W. Female

10/Sec. 1024 Sweeps $C_z - M_2$



4000 HZ S.N. Female

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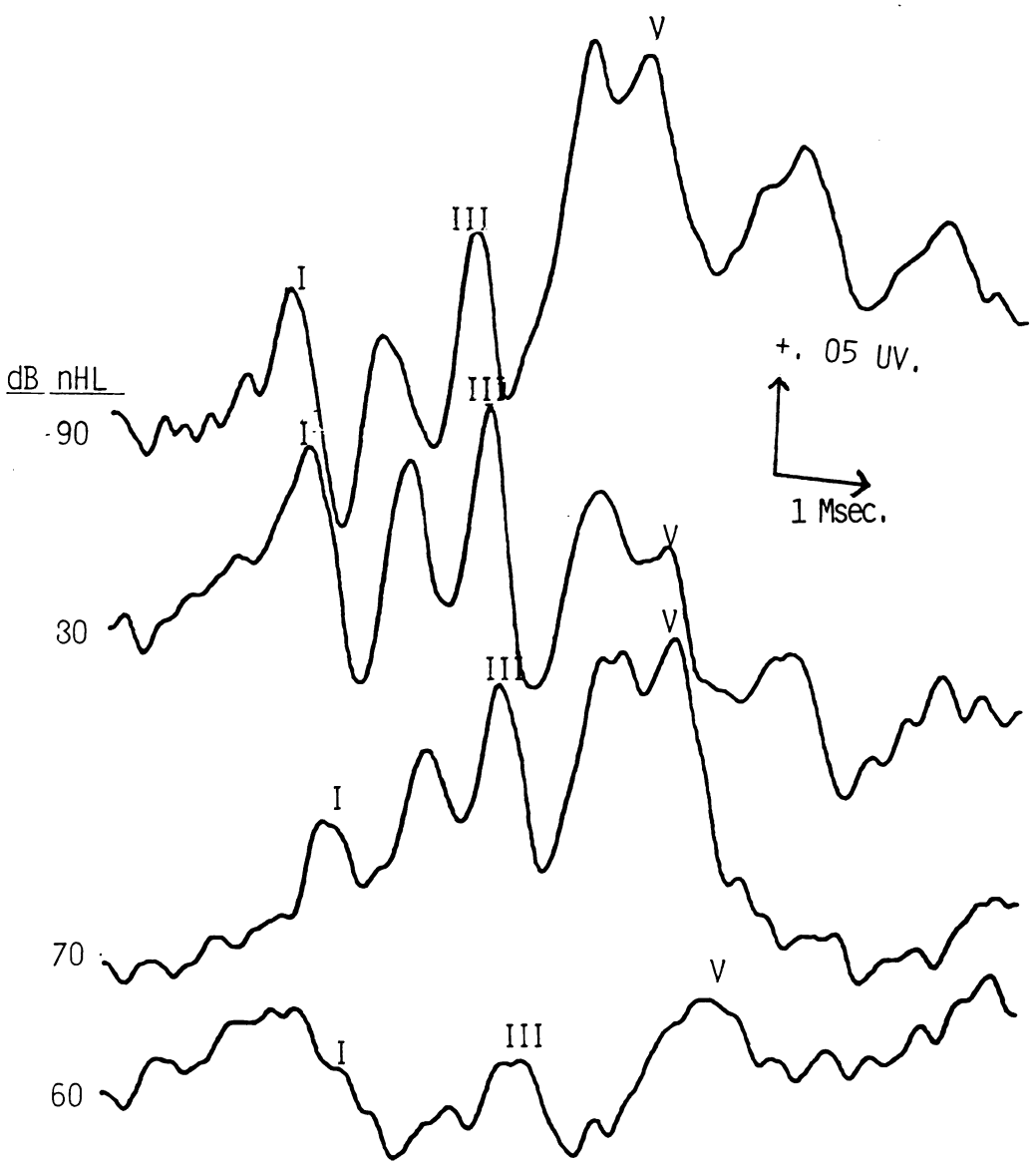
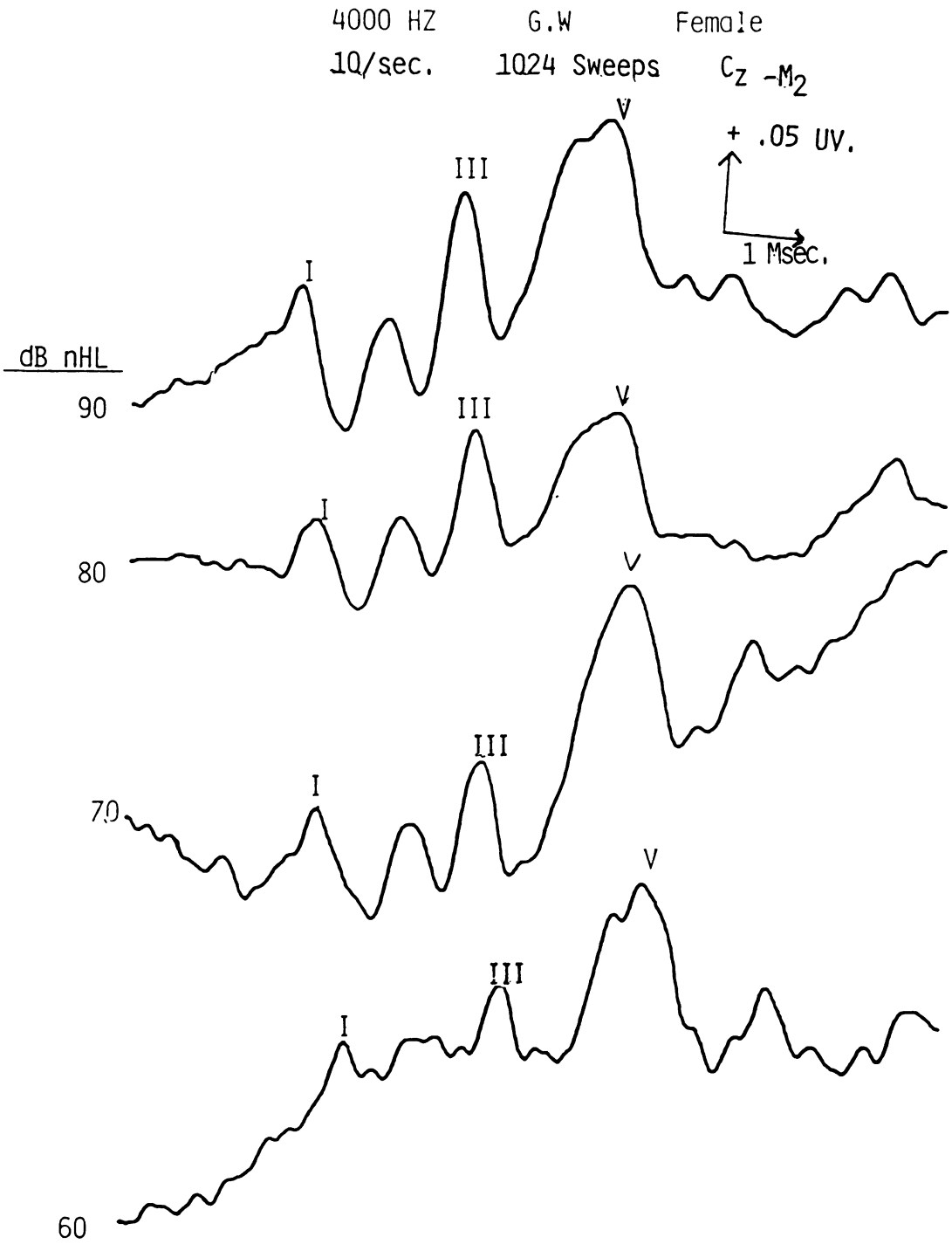



4000 HZ G.W Female
10/sec. 1024 Sweeps $C_z - M_2$
+ .05 UV.
1 Msec.
dB nHL
90
80
70
60
I
III
V

Appendix B1. Latency values at 2000 Hz for wave I, III and V, and for eleven subjects. Subtract 1.0 msec equipment correction factor from the latency values.

Latency of wave I (Msec) from 60-90 dB nHL at 2000 HZ.

	60	70	80	90
M.G.	2.9	2.5	2.6	2.0
A.M.	3.1	2.9	2.6	2.6
M.O.	2.8	2.7	2.4	2.2
B.M.	3.0	2.9	2.5	2.2
I.B.	2.7	2.5	2.5	2.2
B.W.	2.6	2.4	2.4	2.0
T.T.	2.8	2.4	2.5	2.0
V.B.	3.2	2.9	2.6	2.2
P.W.	3.2	3.0	2.7	2.2
S.N.	3.1	2.9	2.9	2.4
G.W.	2.9	2.8	2.6	2.2
TOTAL	32.3	29.9	28.3	24.3
MEAN	2.9	2.7	2.6	2.2
S.D.	.29	.22	.14	.18
VARIANCE	.40	.51	.20	.32

Latency of wave 111 (Msec) from 60-90 dB nHL at 2000 HZ.

	60	70	80	90
M.G	4.9	4.6	4.2	4.0
A.M.	5.2	4.8	4.5	4.2
M.O.	4.9	4.6	4.4	4.1
B.M.	5.2	4.7	4.3	4.1
L.B.	5.0	4.7	4.4	4.3
B.W.	4.7	4.5	4.4	4.1
T.T	5.2	4.8	4.4	4.3
V.B.	5.1	4.6	4.5	4.2
P.W.	5.3	4.8	4.5	4.2
S.N.	5.2	5.0	4.6	4.6
G.W.	5.0	4.6	4.4	4.0
TOTAL	55.4	51.7	48.6	42.3
MEAN	5.0	4.7	4.4	3.8
S.D.	.16	.14	.10	.17
VARIANCE	.26	.20	.11	.28

Latency of wave V (Msec) from 60-90 dB nHL at 2000 HZ.

	60	70	80	90
M.G	6.8	6.6	6.5	6.0
A.M.	7.1	6.4	6.1	5.8
M.O..	6.7	6.4	6.2	5.8
B.M.	6.8	6.5	6.3	6.1
L.B.	7.1	6.6	6.6	6.2
B.W.	6.8	6.6	6.5	6.0
T.T.	6.8	6.4	6.2	6.0
V.B.	6.7	6.5	6.0	5.6
P.W.	6.6	6.6	6.1	6.1
S.N.	6.7	6.5	6.3	5.8
G.W.	6.8	6.5	6.4	6.0
TOTAL	81.6	71.6	69.2	65.4
MEAN	7.4	6.5	6.3	5.9
S.D.	.15	.08	.20	.17
VARIANCE	.24	.06	.04	.30

Appendix B2. Latency values at 4000 Hz for waves I, III and V for eleven subjects. Subtract 1.0 Msec equipment correction factor from the latency values.

Latency of wave I (Msec) from 60-90 dB nHL at 4000 HZ.

	60	70	80	90
M.G.	2.5	2.1	2.0	1.7
A.M.	2.6	2.3	2.1	1.8
M.O.	2.4	2.2	2.0	1.9
B.M.	2.3	2.2	2.0	1.8
L.B.	2.2	2.1	2.0	1.9
B.W.	2.2	2.1	2.0	2.0
T.T.	2.4	2.3	2.0	1.8
V.B.	2.5	2.4	2.1	2.0
P.W.	2.6	2.4	2.1	2.1
S.N.	2.6	2.5	2.2	2.0
G.W.	2.4	2.4	2.0	1.9
TOTAL	26.7	24.6	22.5	20.9
MEAN	2.4	2.2	2.0	1.9
S.D.	.14	.18	.06	.29
VARIANCE	.22	.27	.04	.87

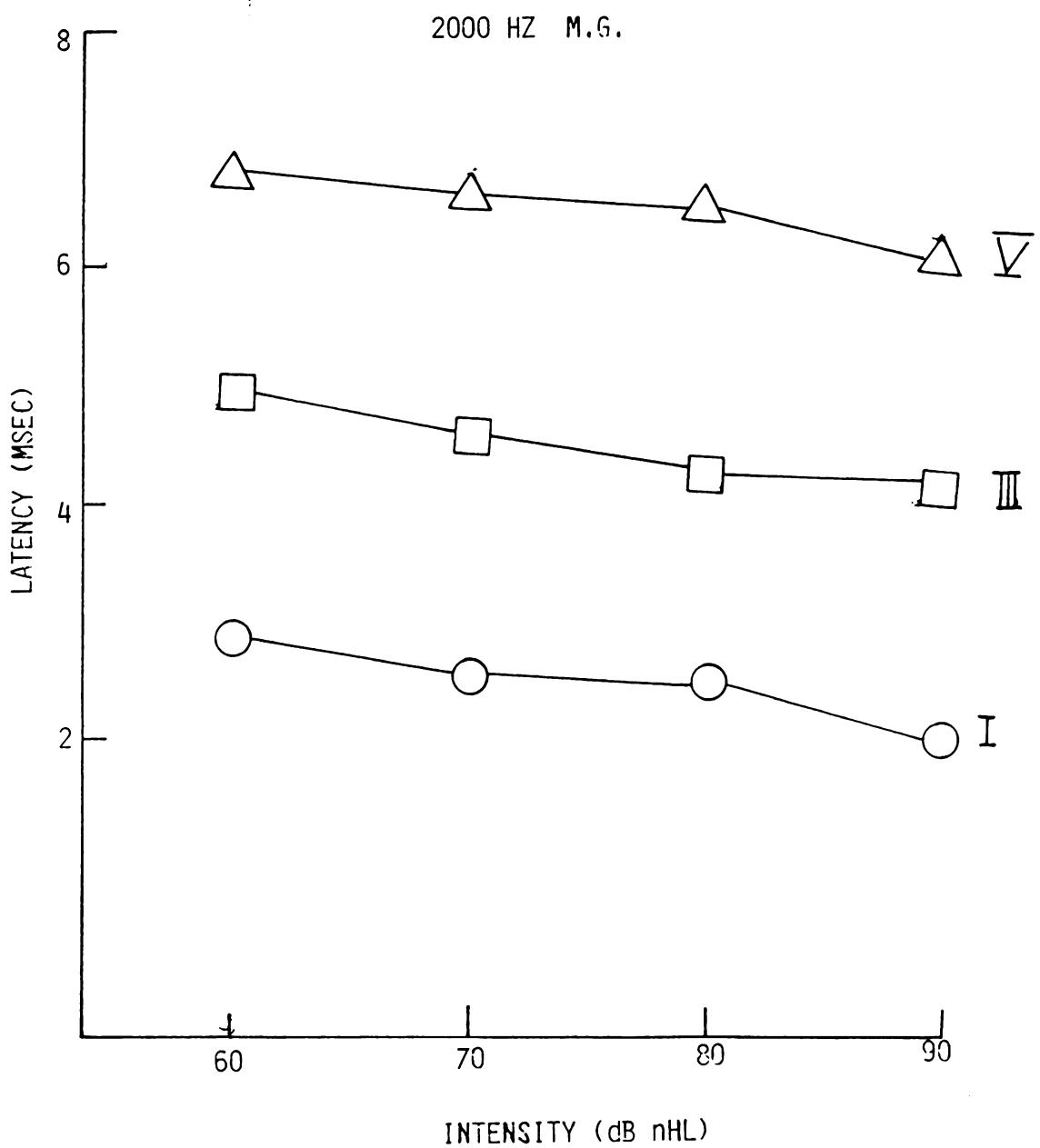
Latency of wave III (Msec) from 60-90 dB nHT at 4000 HZ.

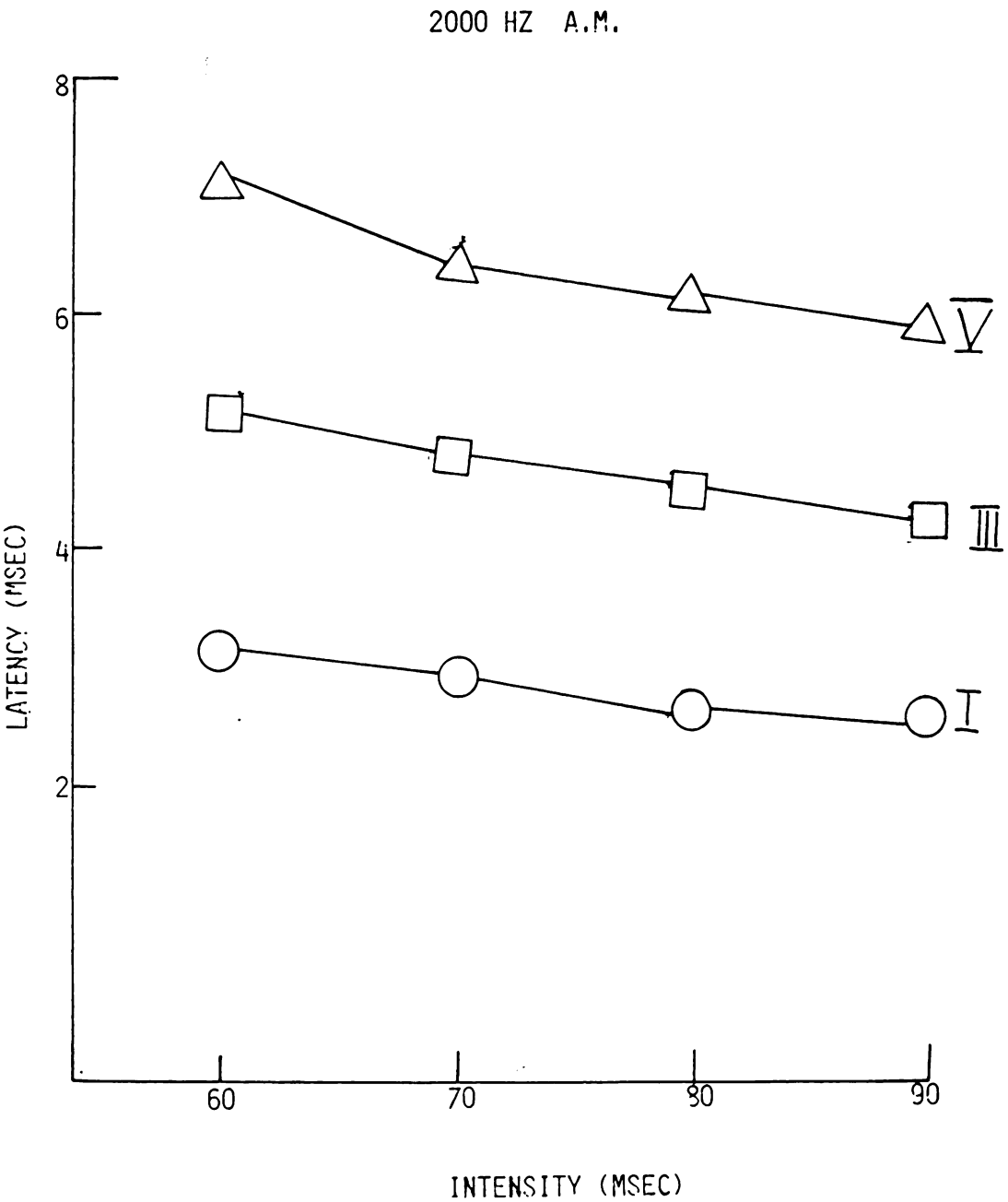
	60	70	80	90
M.G.	4.6	4.4	4.1	3.9
A.M.	4.3	4.1	4.2	3.7
M.O.	4.4	4.2	4.1	3.9
B.M.	4.2	4.1	4.0	3.8
L.B.	4.3	4.1	4.0	3.8
B.W.	4.3	4.1	4.0	4.0
T.T.	4.2	4.0	3.9	3.8
V.B.	4.9	4.3	4.2	3.9
P.W.	4.4	4.3	4.1	4.0
S.N.	4.6	4.3	4.1	3.9
G.W.	4.3	4.2	4.0	3.8
TOTAL	48.5	46.1	44.1	33.9
MEAN	4.1	4.2	4.0	3.9
S.D.	.21	.12	.07	.09
VARIANCE	.44	.14	.06	.09

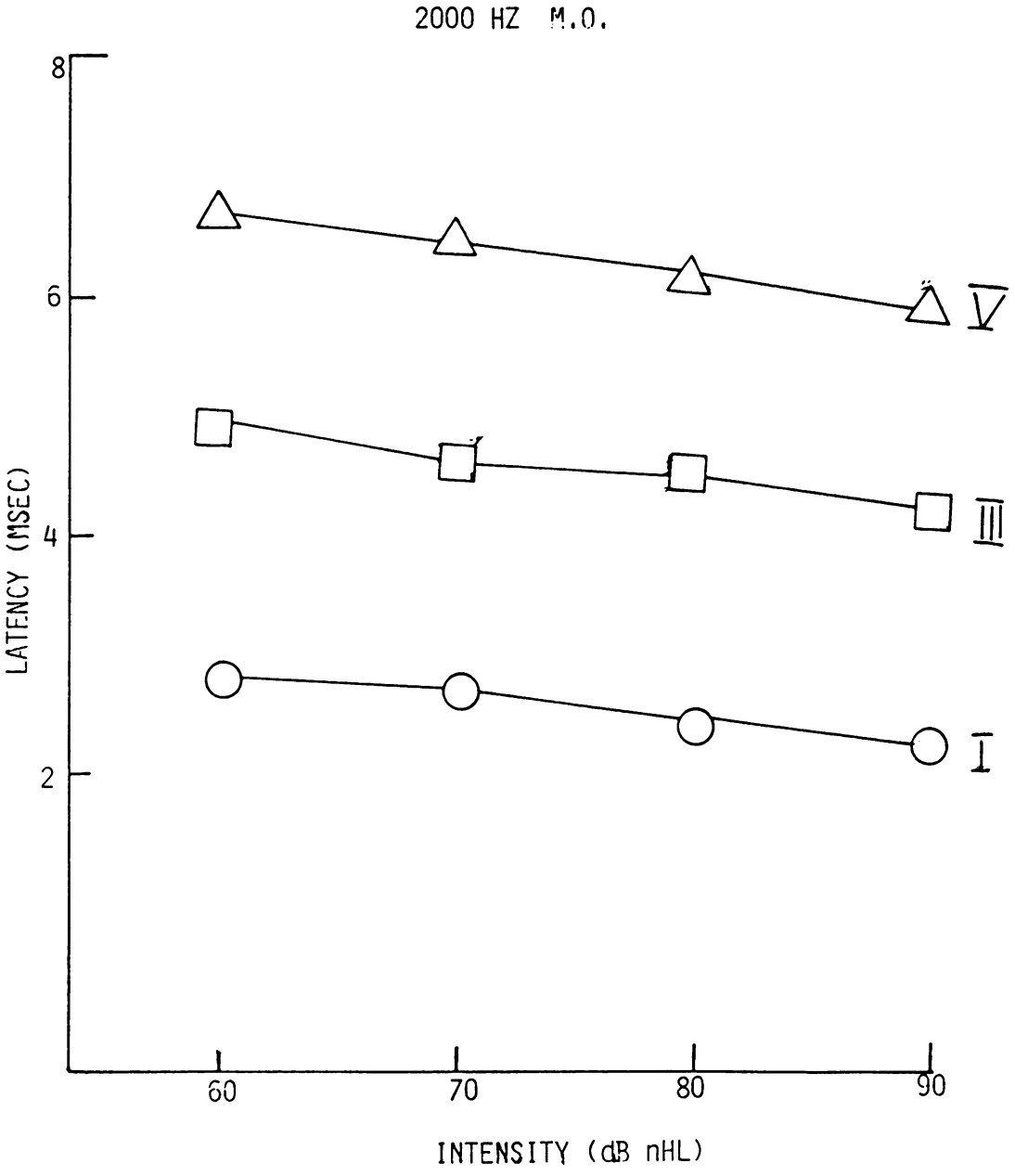
Latency of wave V (Msec) from 60-90 dB nHL at 4000 HZ.

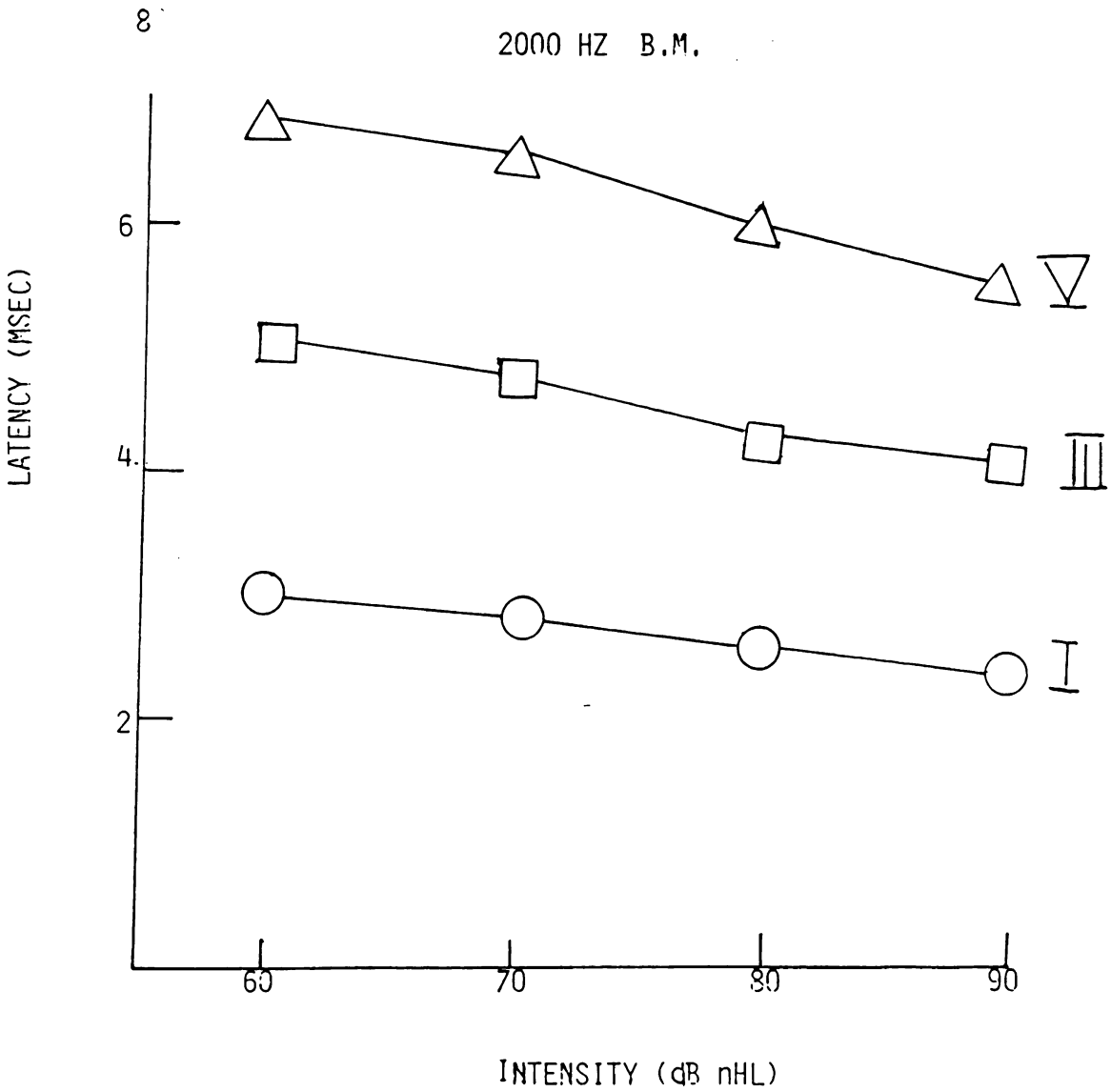
	60	70	80	90
M.G.	7.0	6.4	6.1	5.9
A.M.	6.1	6.9	5.8	5.4
M.O.	6.2	6.1	6.0	5.8
B.M.	6.3	6.2	6.0	5.8
L.B.	6.3	6.2	5.9	5.8
B.W.	6.4	6.2	6.0	6.0
T.T.	6.2	6.0	5.8	5.7
V.B.	6.2	6.2	6.2	5.8
P.W.	6.4	6.2	6.0	5.8
S.N.	6.5	6.2	6.1	5.8
G.W.	6.2	6.2	6.0	5.7
TOTAL	69.8	67.8	65.9	63.5
MEAN	6.3	6.1	6.0	5.8
S.D.	.24	.26	.12	.14
VARIANCE	.60	.68	.14	.21

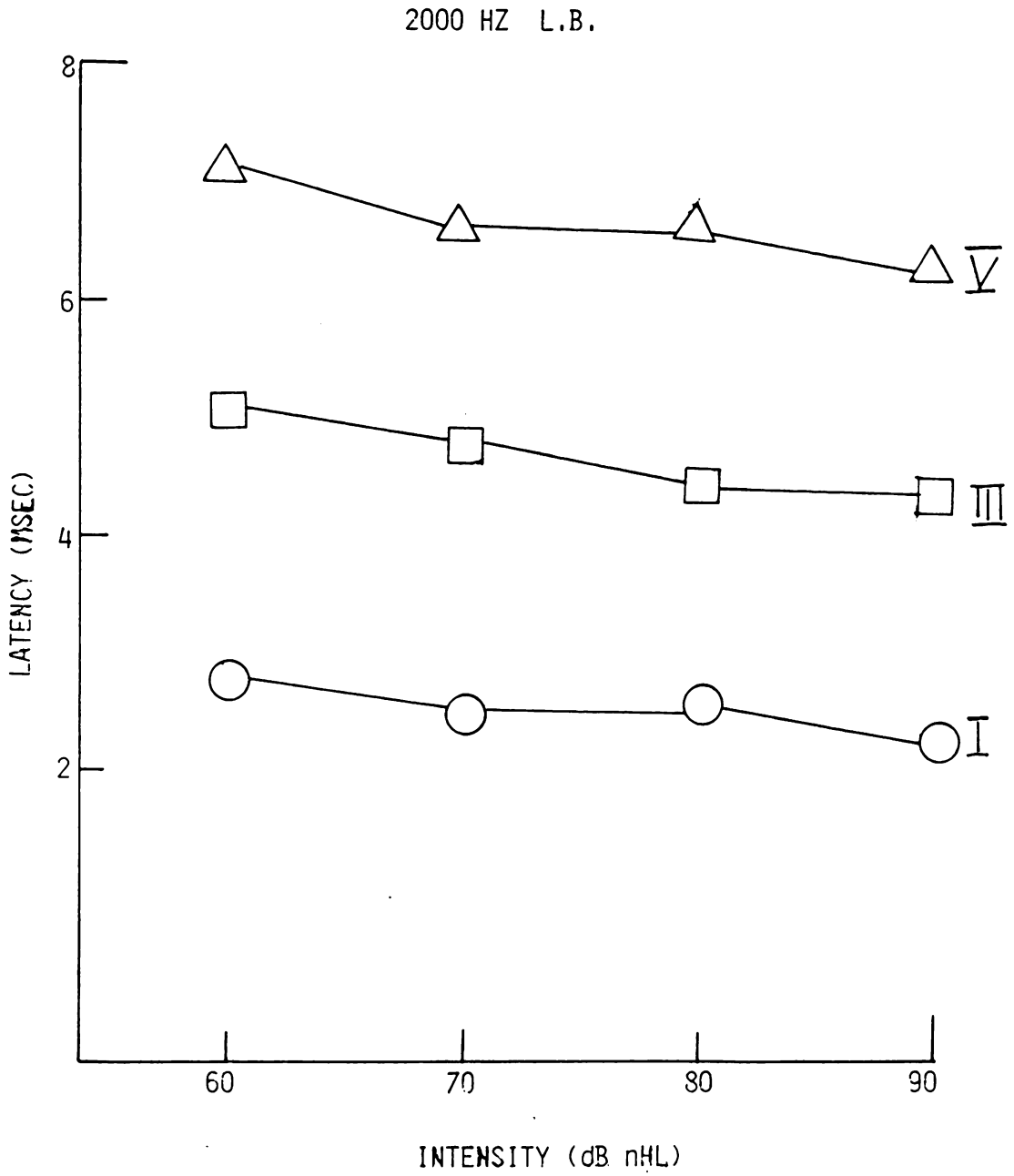
Appendix C1. Latency input-output functions for waves I, III and V at 2000 Hz for each subject.

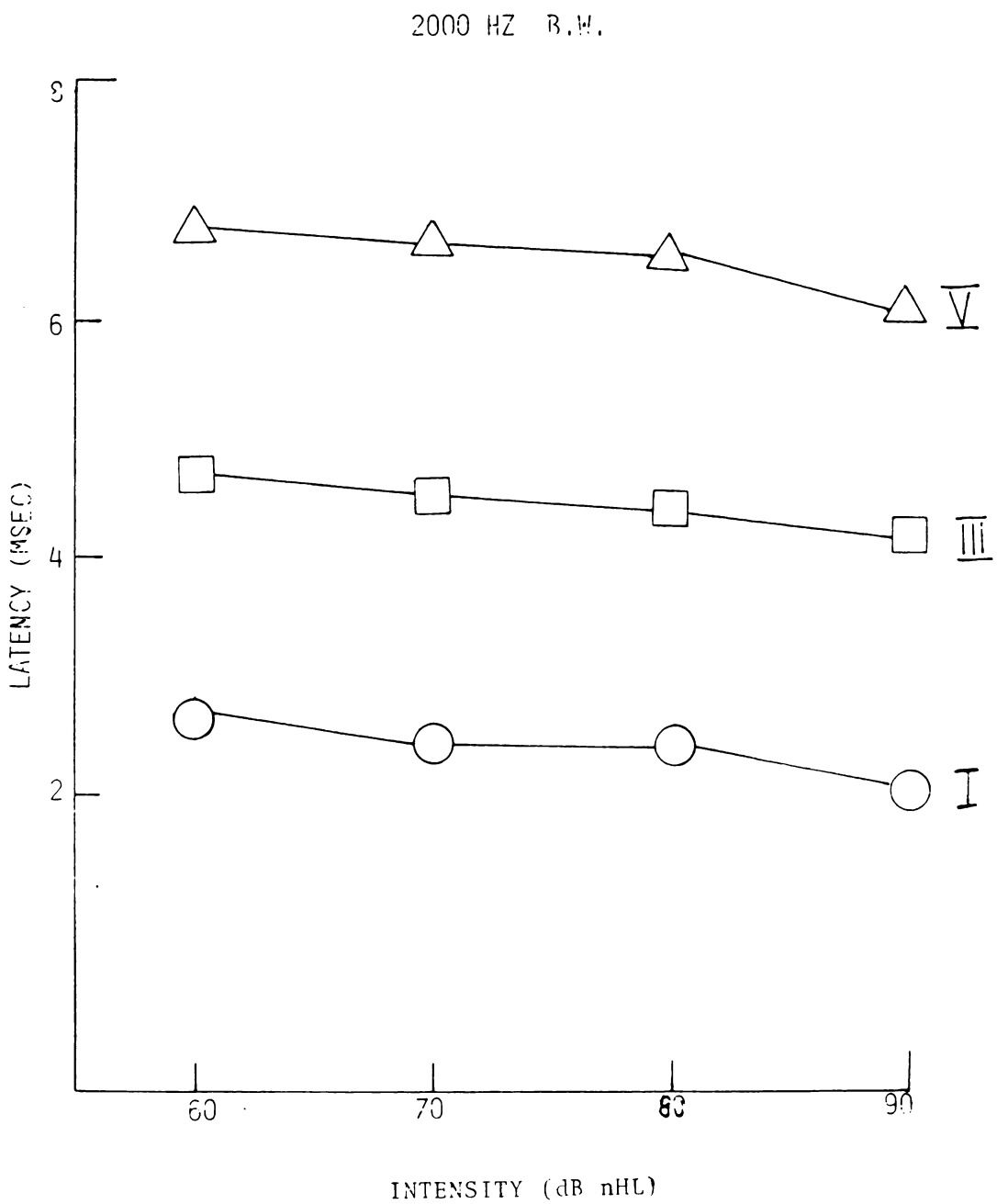

2000 HZ M.G.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

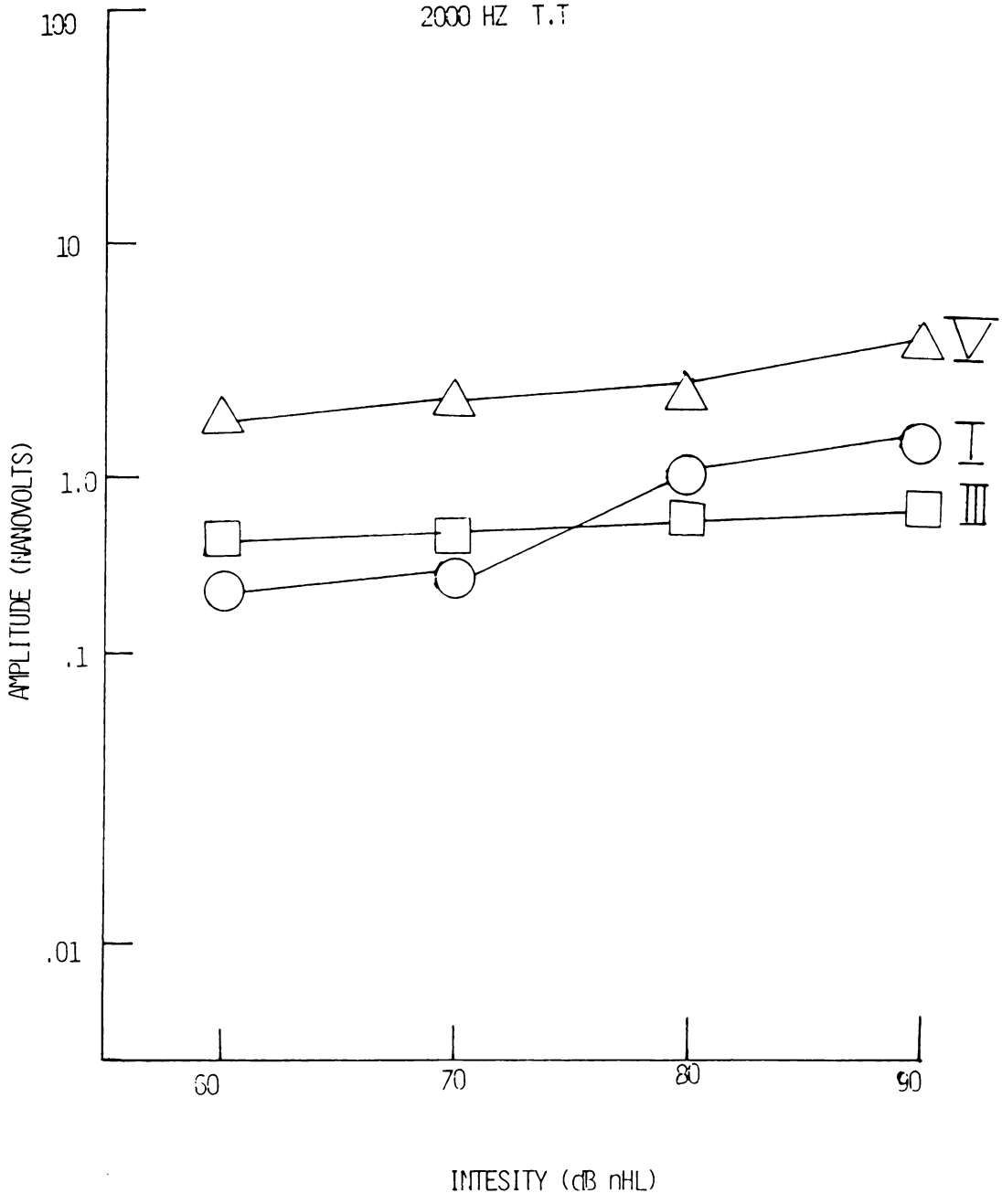

2000 HZ A.M.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (MSEC)

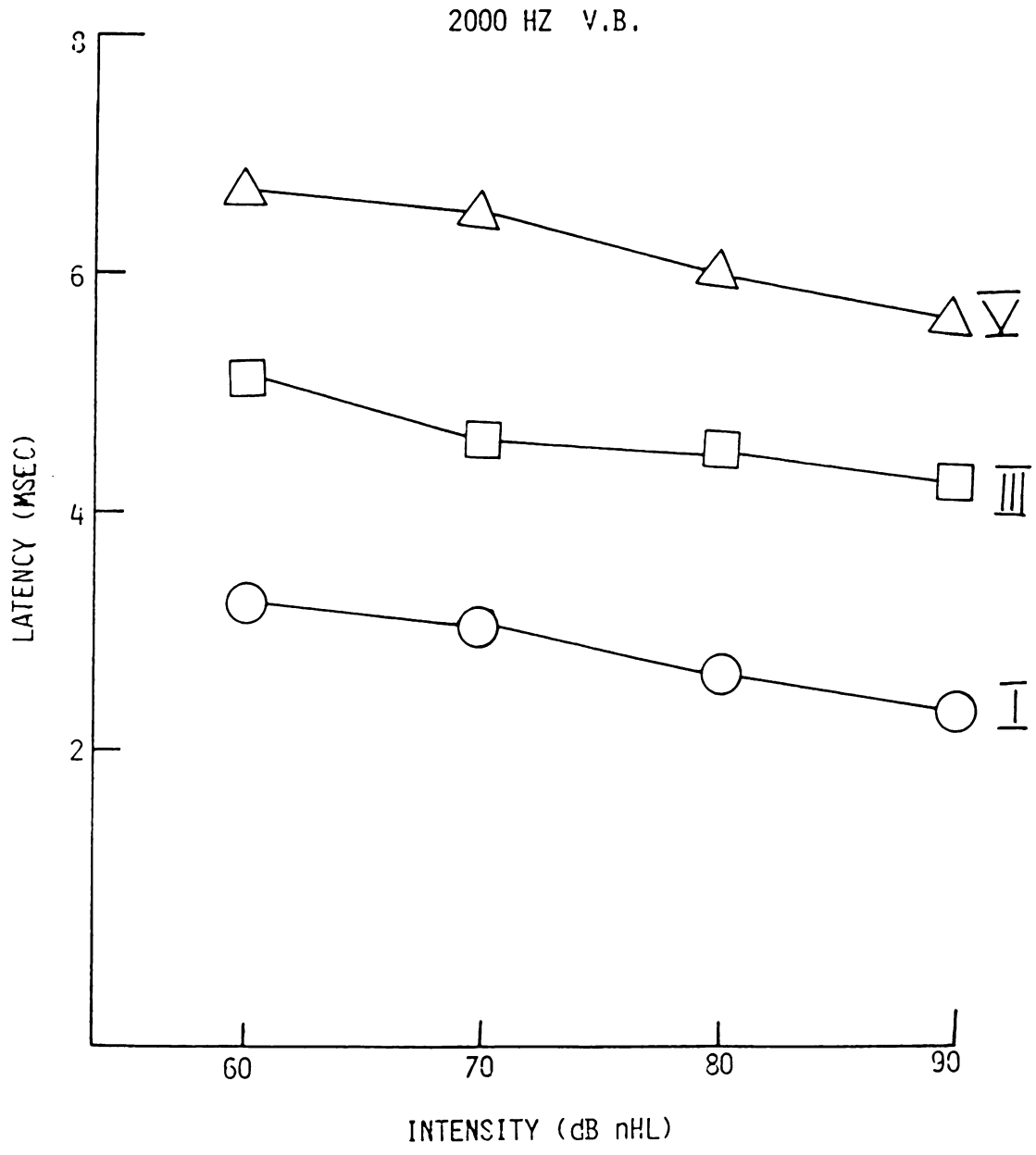

2000 HZ M.O.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

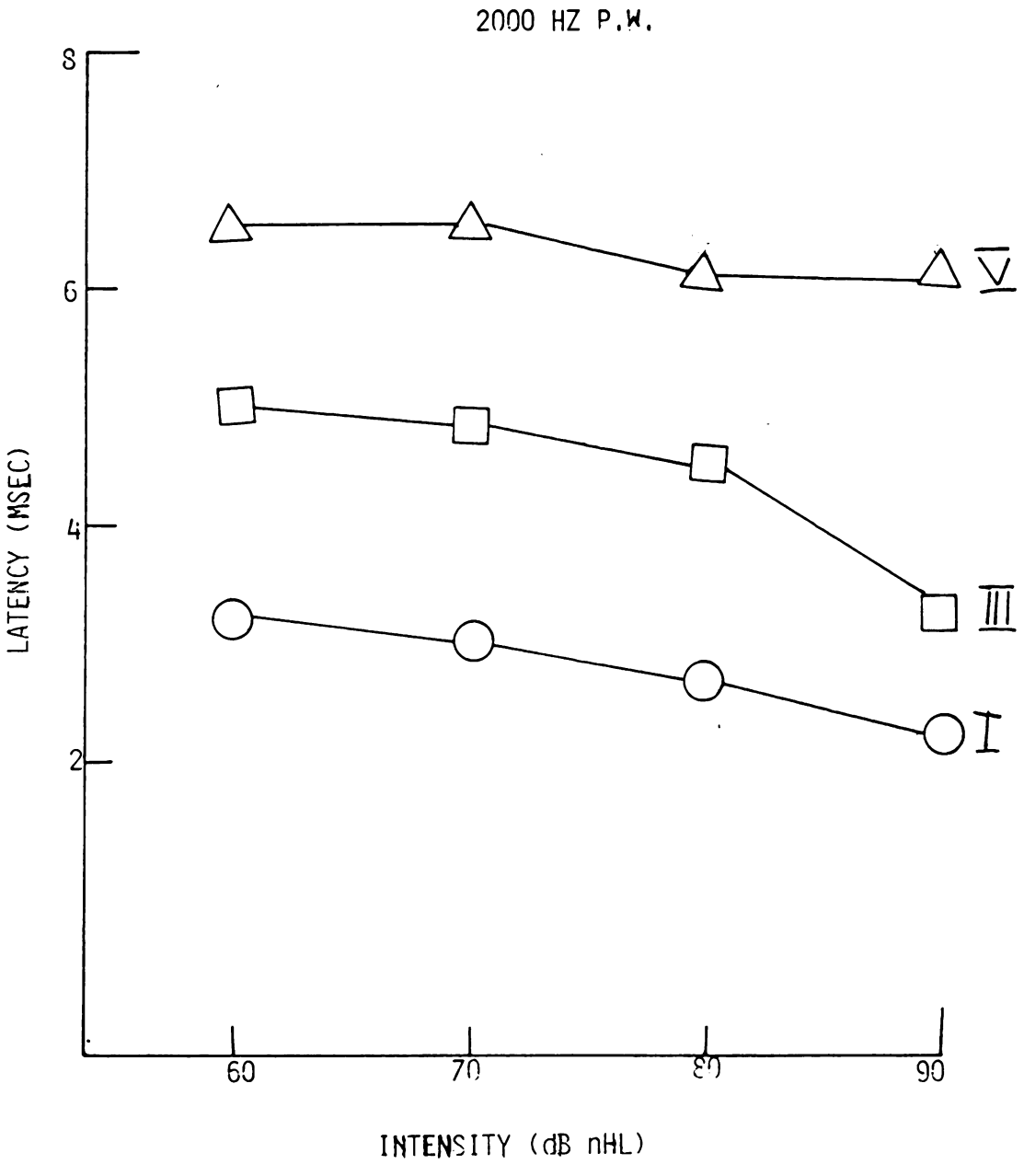

8
2000 HZ B.M.
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

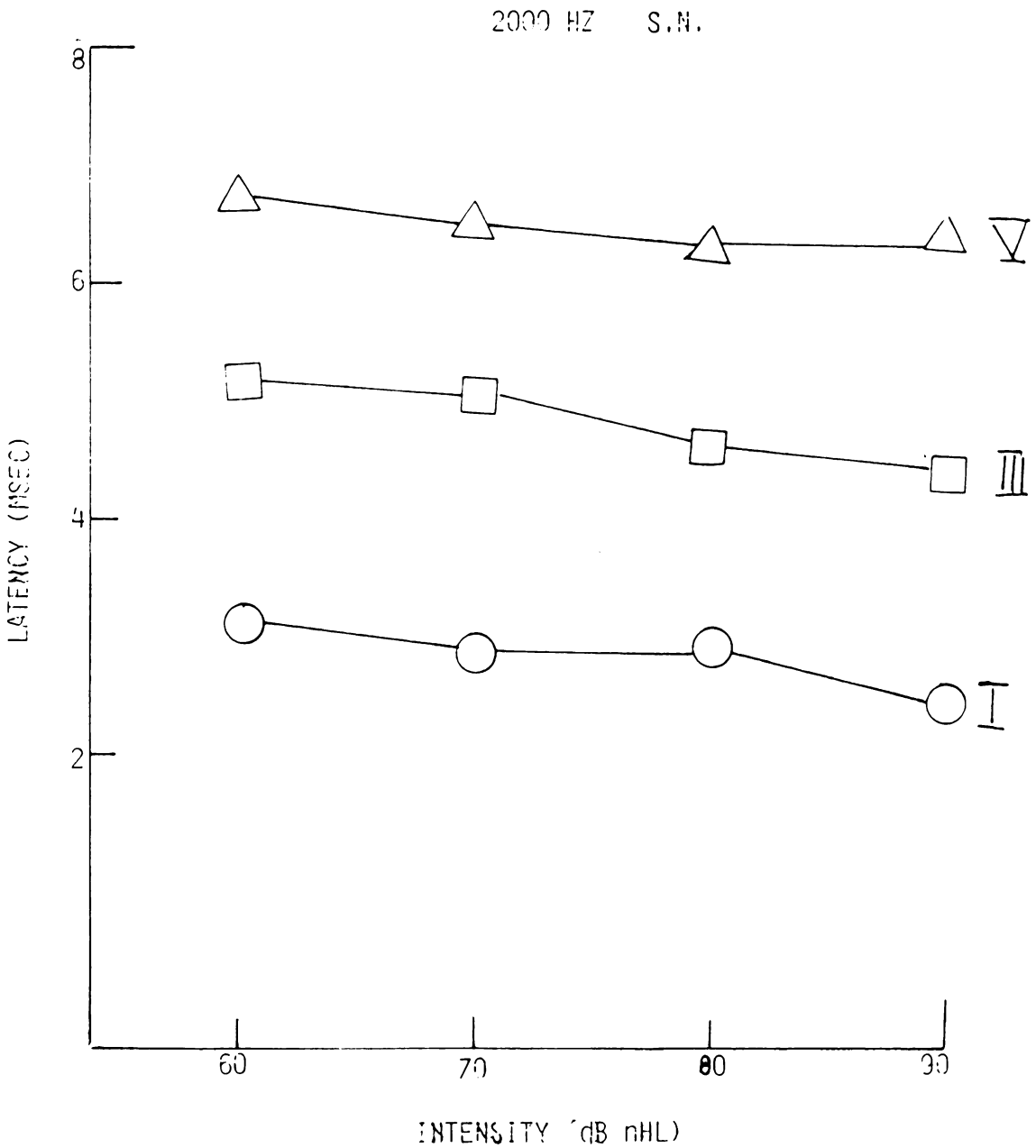

2000 HZ L.B.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

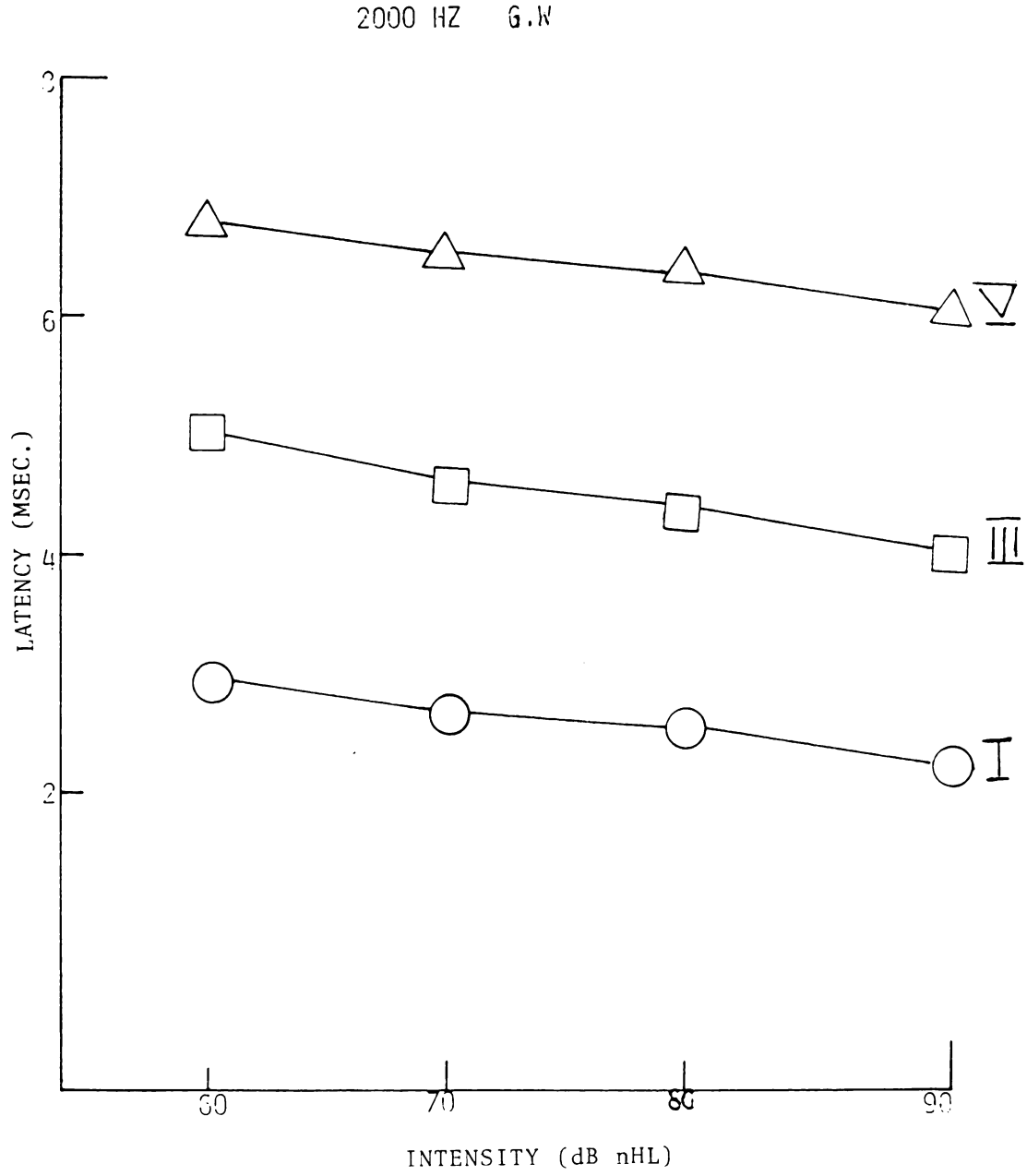

2000 HZ R.W.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)


2000 HZ T.T
100
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTESITY (dB nHL)
V
I
III

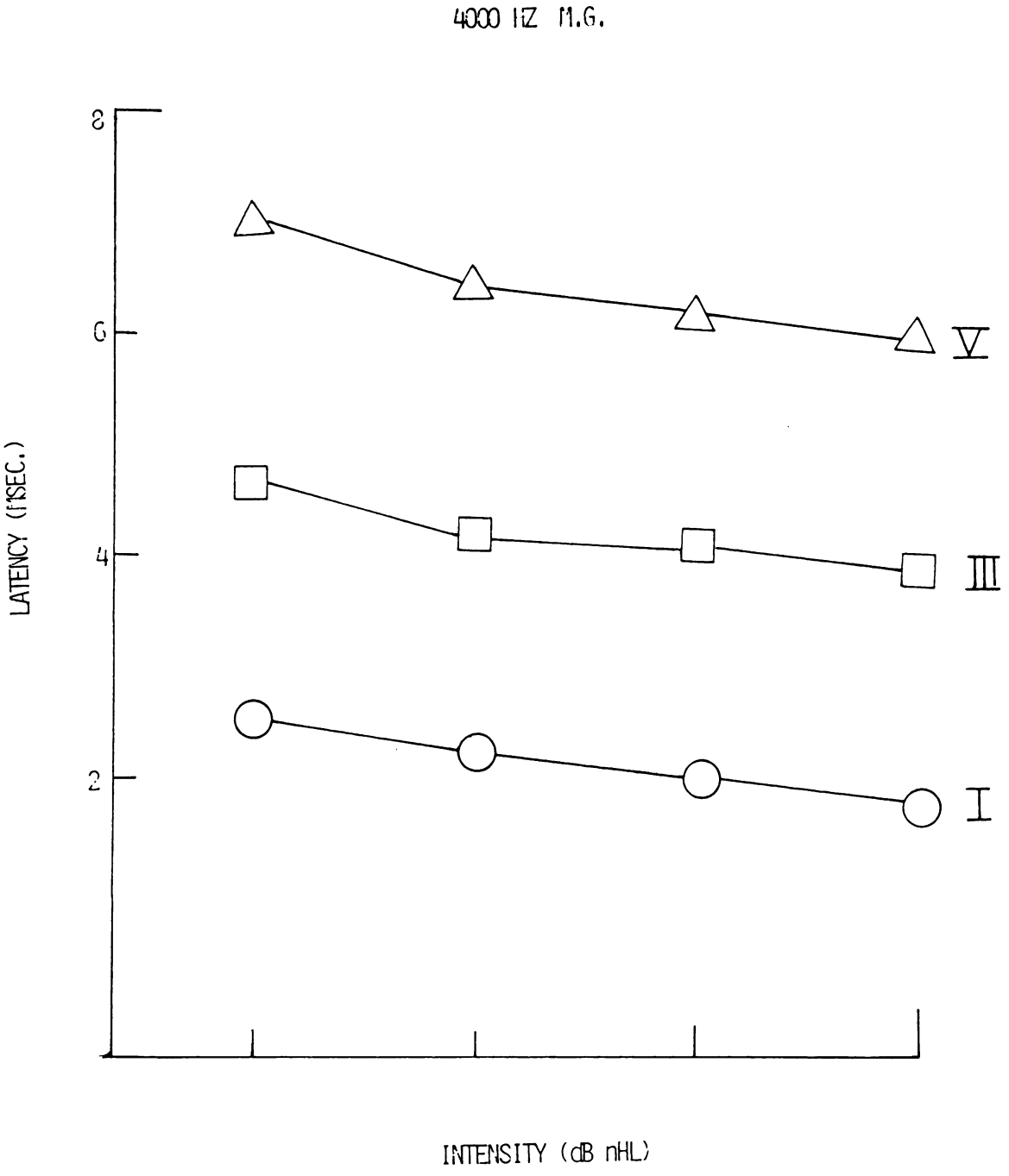

2000 HZ V.B.
8
6
4
2
LATENCY (MSEC)
60
70
80
90
INTENSITY (dB nHL)
V
III
I

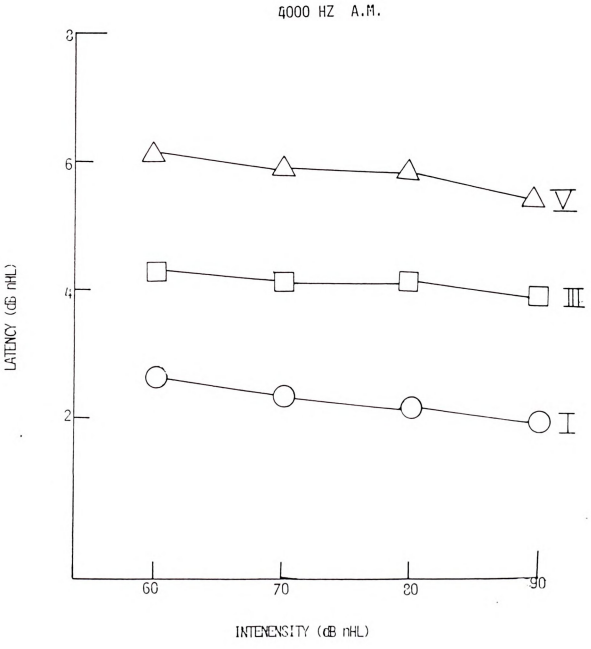

2000 HZ P.W.
8
6
4
2
LATENCY (MSEC)
60
70
80
90
INTENSITY (dB nHL)
V
III
I


2000 HZ S.N.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

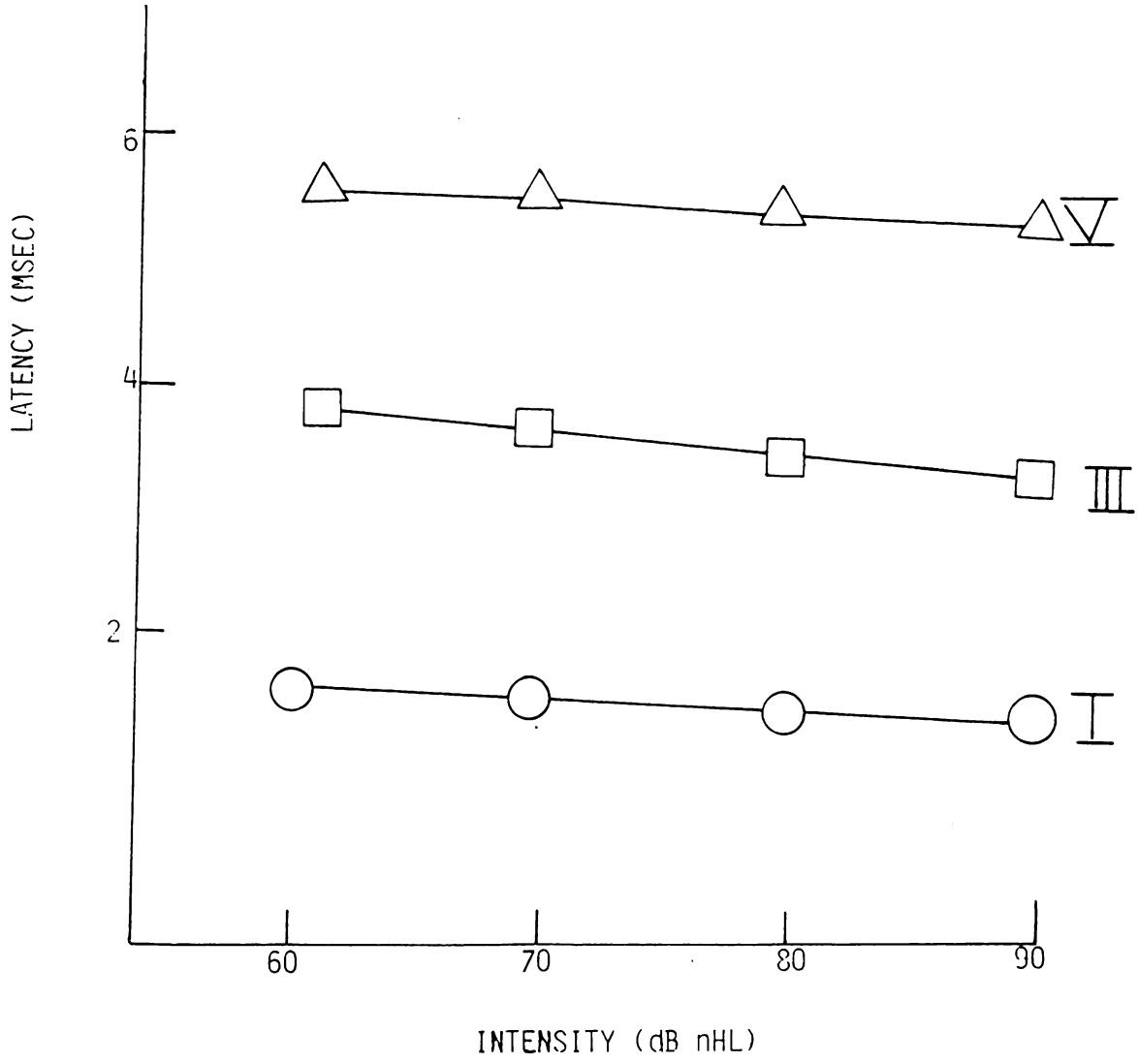

2000 HZ G.W
LATENCY (MSEC.)
6
4
2
70
INTENSITY (dB nHL)
V
III
I

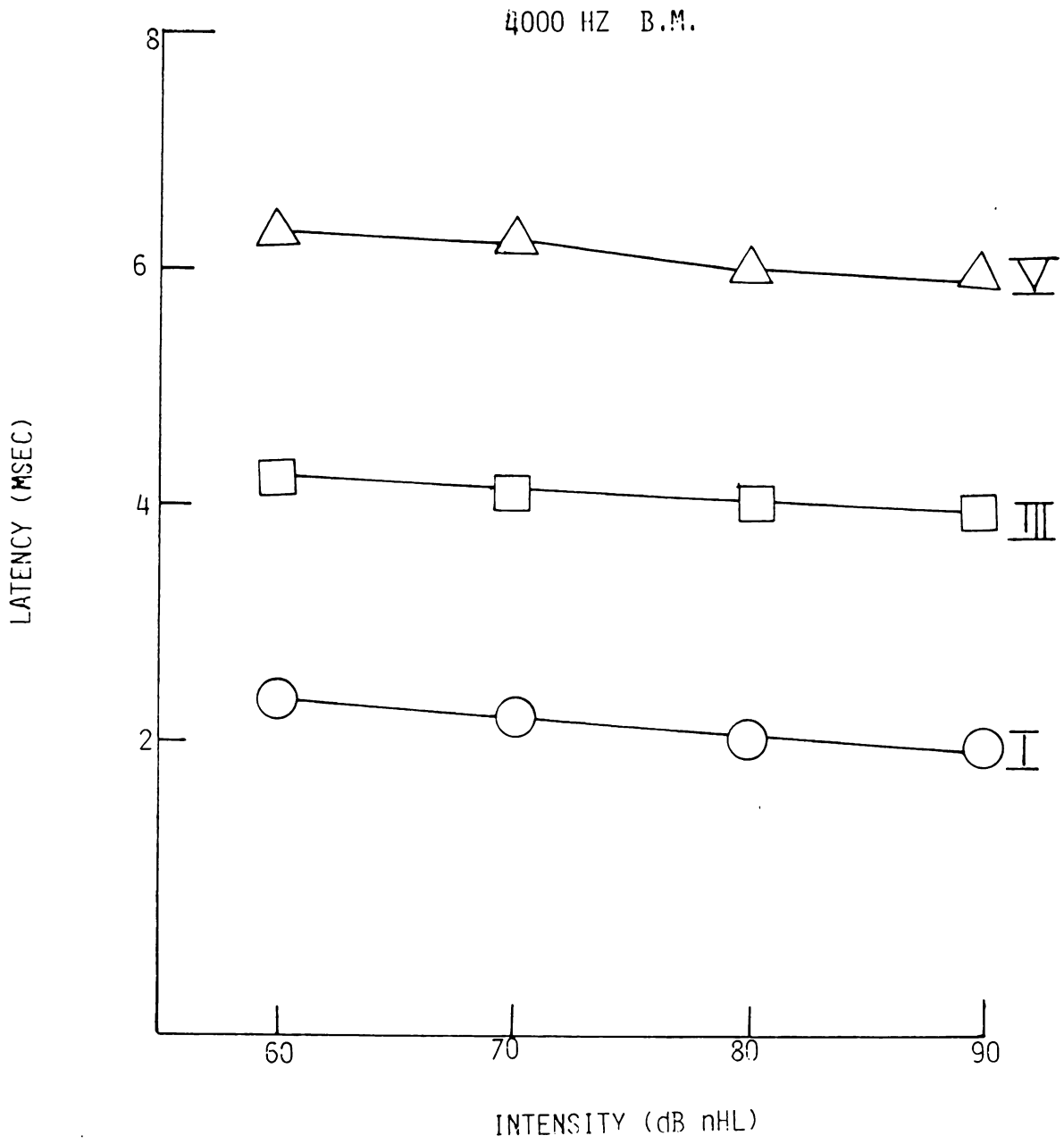
Appendix C2. Latency input-output functions for waves I, III and V at 4000 Hz for each subject.

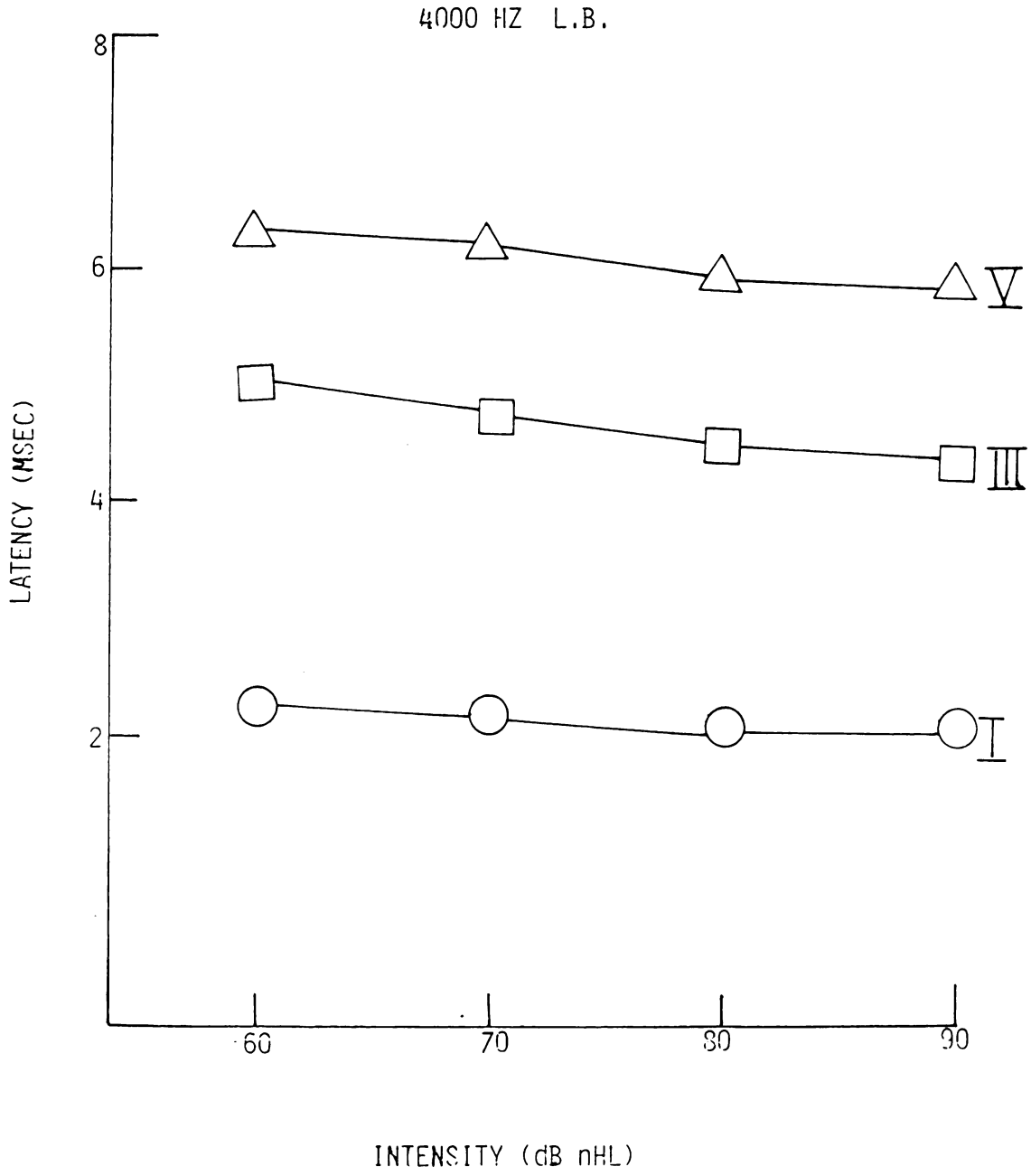

4000 HZ M.G.
8
6
4
2
LATENCY (MSEC.)
V
III
I
INTENSITY (dB nHL)

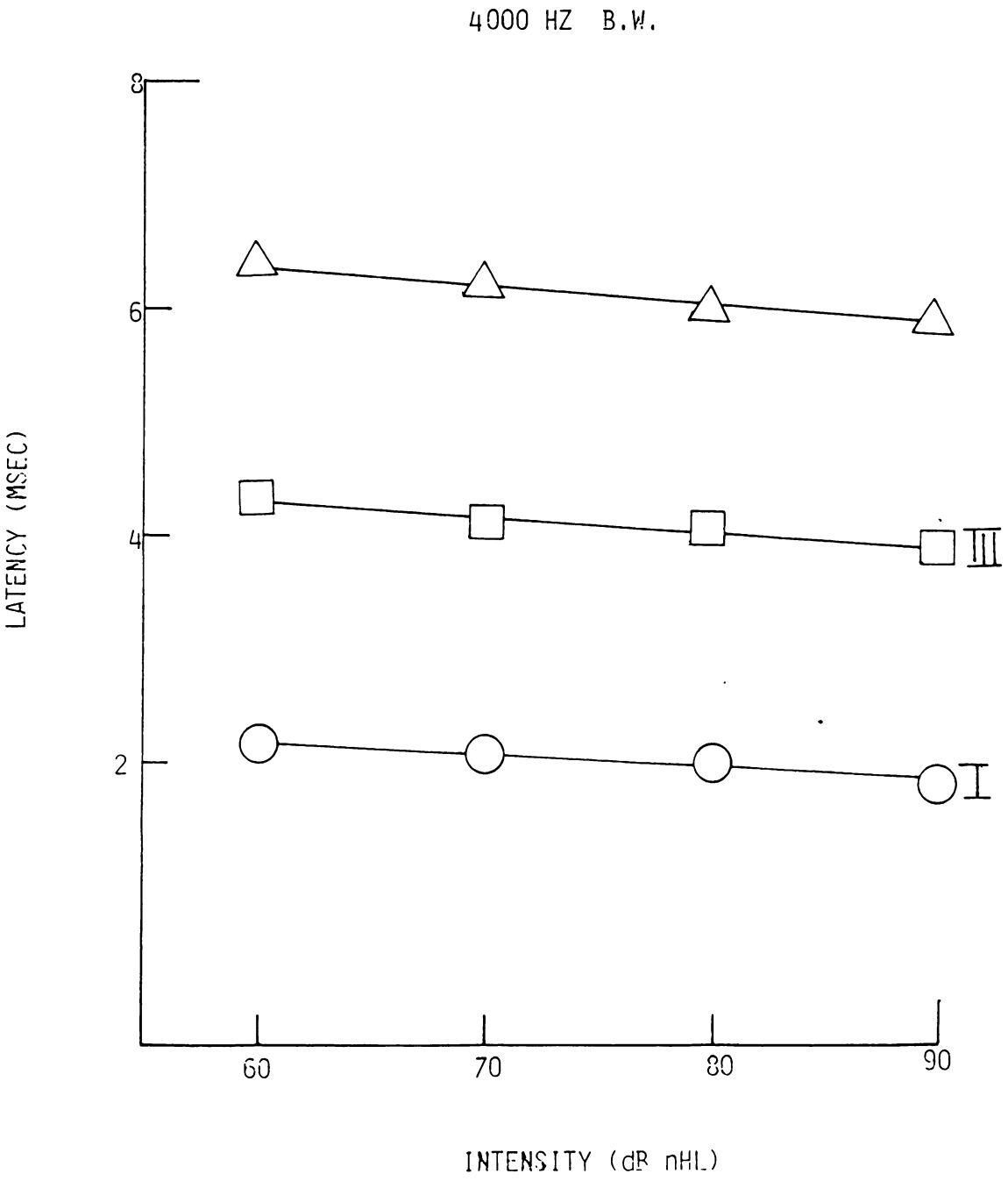

4000 HZ A.M.
8
6
4
2
LATENCY (dB nHL)
60
70
80
90
INTENENSITY (dB nHL)
V
III
I

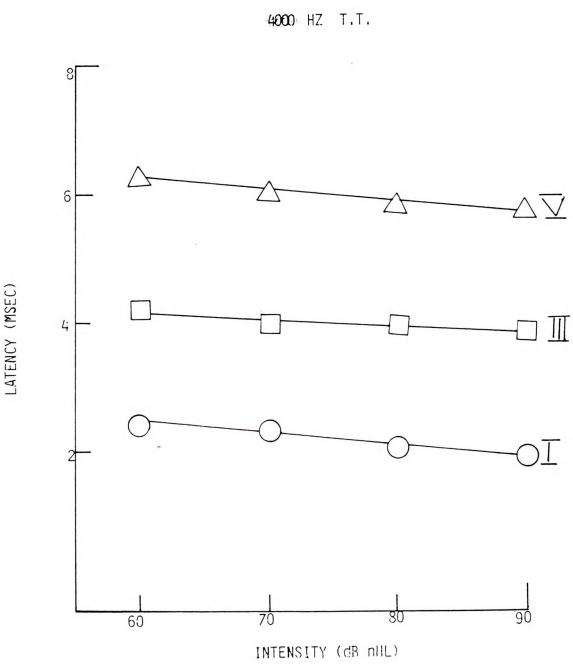
4000 HZ M.O.

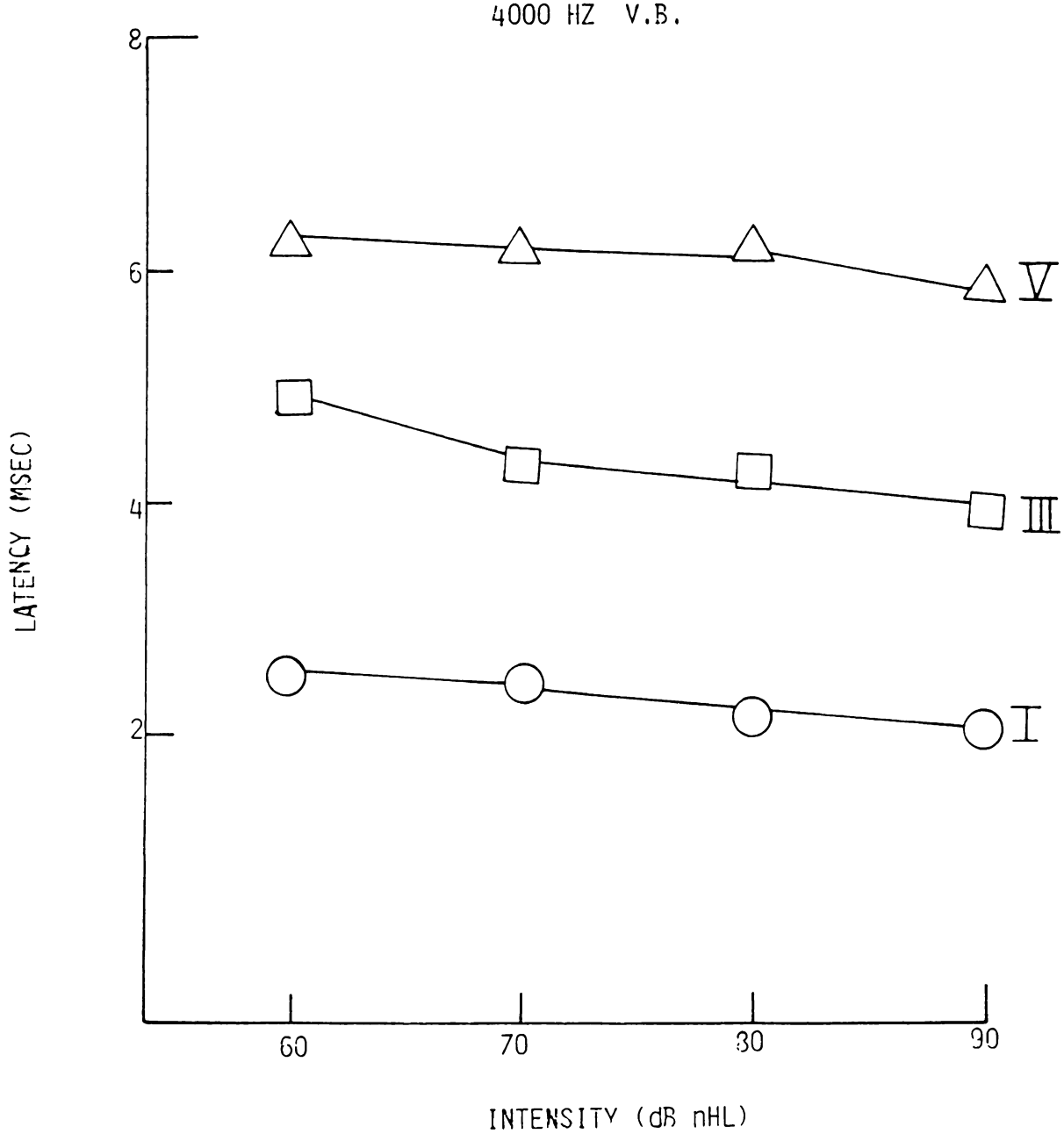


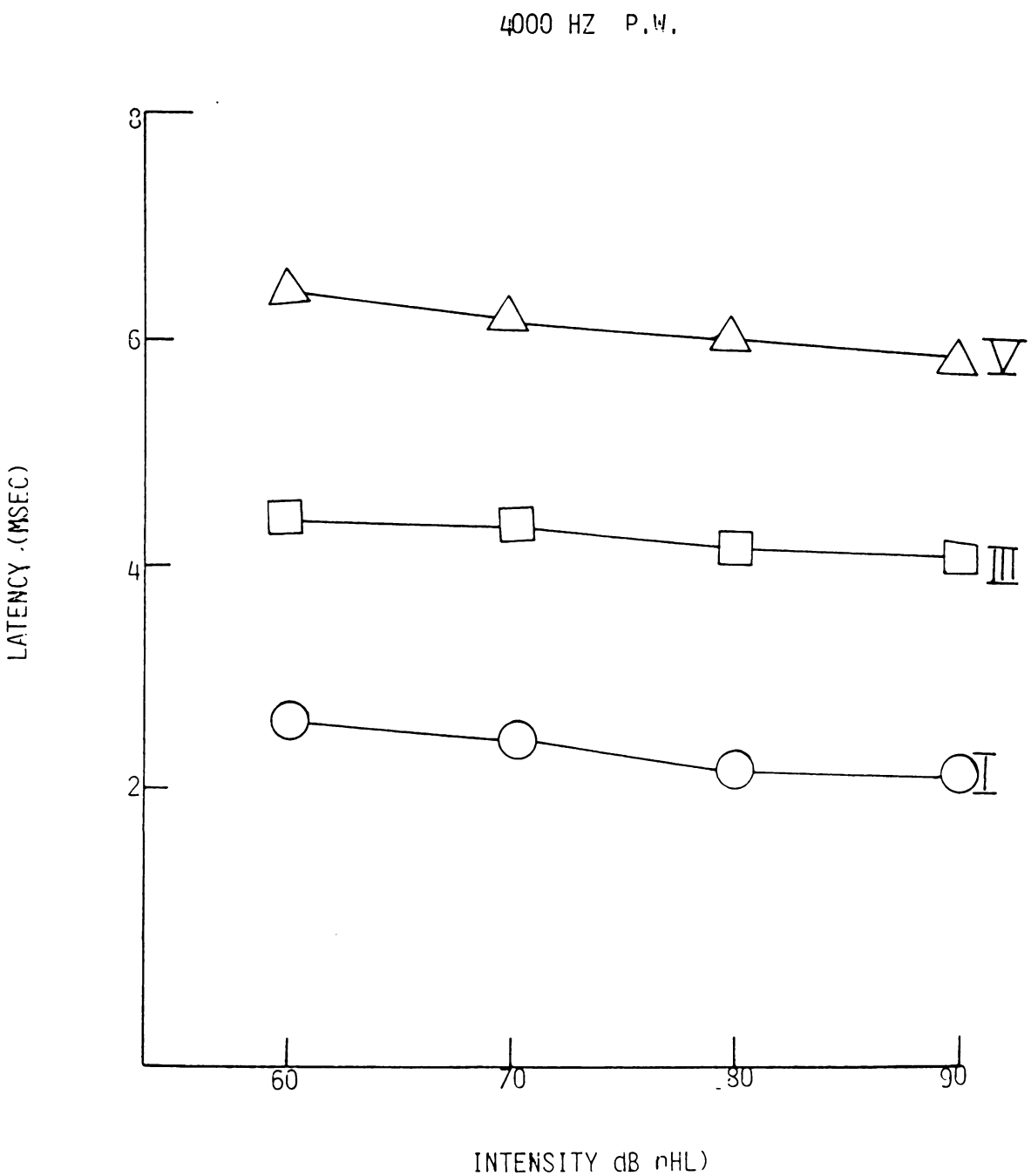

4000 HZ B.M.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

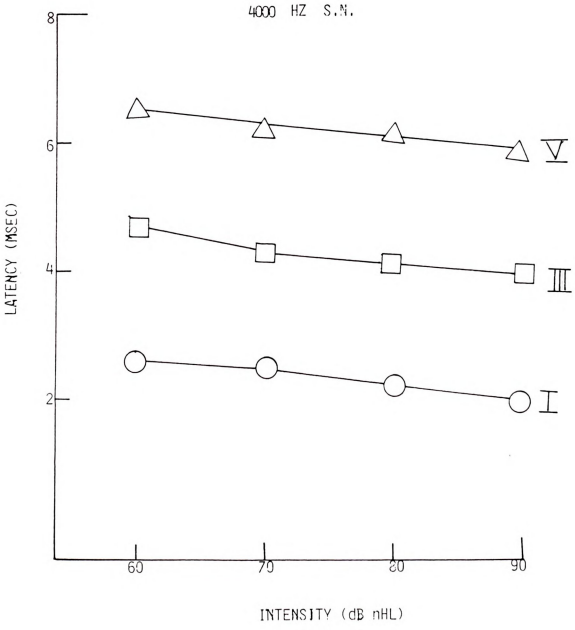

4000 HZ L.B.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

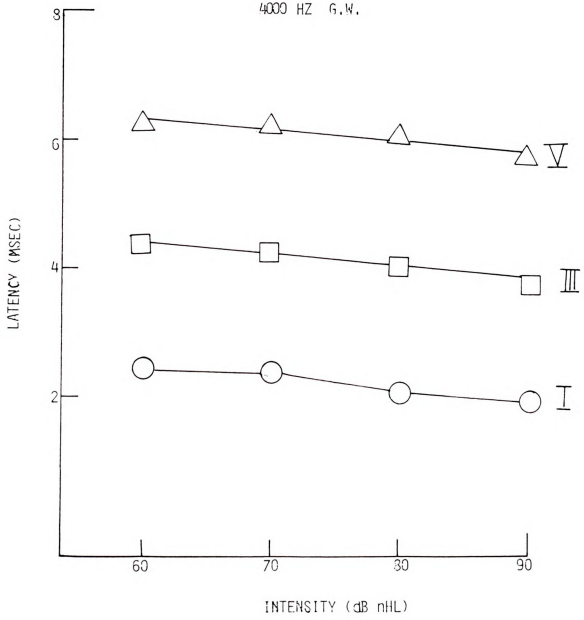

4000 HZ B.W.
8
6
4
2
LATENCY (MSEC)
III
I
60
70
80
90
INTENSITY (dB nHL)


4000 HZ T.T.
8
6
4
2
LATENCY (MSEC)
60
70
80
90
INTENSITY (dB nHL)
V
III
I


4000 HZ V.B.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)


4000 HZ P.W.
8
6
4
2
LATENCY (MSEC)
60
70
80
90
INTENSITY dB nHL)
V
III
I


4000 HZ S.N.
8
6
4
2
LATENCY (MSEC)
V
III
I
60
70
80
90
INTENSITY (dB nHL)


4000 HZ G.W.
8
6
4
2
LATENCY (MSEC)
60
70
30
90
INTENSITY (dB nHL)
V
III
I

Appendix D1. Amplitude values for waves I, III and V at 2000 Hz.

Amplitude of wave 1 (NANOVOLTS) from 60-90 dB nHL at 2000 HZ.

	60	70	80	90
M.G.	150	162	232	380
A.M.	40	44	152	196
M.O.	180	172	148	144
B.M.	112	104	152	288
L.B.	30	32	30	30
B.W.	124	296	304	512
T.T.	36	42	276	428
V.B.	32	40	216	272
P.W.	44	96	108	172
S.N.	30	32	128	236
G.W.	96	183	212	250
TOTAL	852	1223	1958	2112
MEAN	75	111.1	178.4	265
S.D.	46.3	88.7	78.3	121.3
VARIANCE	21455.9	75217.5	61382.5	14766.5

Amplitude of wave 111 (NANOVOLTS) from 60-90 dB nHL at 2000 HZ.

	60	70	80	90
M.G.	65	235	250	272
A.M.	124	176	172	160
M.O.	130	110	101	128
B.M.	116	184	328	520
L.B.	60	50	50	108
B.W.	162	335	416	280
T.T.	54	34	196	400
V.B.	48	124	268	360
P.W.	24	80	92	216
S.N.	44	80	164	236
G.W.	124	134	272	305
TOTAL	941	1548	2309	2893
MEAN	88.7	141	210	264
S.D.	49.9	88.4	92.4	103.5
VARIANCE	17572.1	78228	85410	107032

Amplitude of wave V (NANOVOLTS) from 60-90 dB nHL at 2000 HZ.

	60	70	80	90
M.G.	162	203	336	410
A.M.	174	230	240	588
M.O.	510	512	512	404
B.M.	404	412	424	576
L.B.	144	136	121	140
B.W.	304	320	336	416
T.T.	402	408	440	664
V.B.	152	328	404	344
P.W.	152	176	308	292
S.N.	76	77	216	388
G.W.	95	200	246	382
TOTAL	2311	2997	3582	4084
MEAN	210	264	326	425
S.D.	124.8	127.3	110.2	119.9
VARIANCE	155816.9	162030	121331	143955

Appendix D2. Amplitude values for waves I, III and V at 4000 Hz.

Amplitude wave 1 (NANOVOLTS) from 60-90 dB nHL at 4000 HZ.

	60	70	80	90
M.G	220	372	508	544
A.M.	72	256	336	380
M.O.	212	204	180	250
B.M.	268	268	269	464
L.B.	187	172	132	212
B.W.	304	512	608	664
T.T.	132	308	276	416
V.B.	140	180	428	334
P.W.	68	240	316	192
S.N.	44	80	328	332
G.W.	123	200	261	302
TOTAL	1727	2792	3662	3100
MEAN	180	253	365	374
S.D.	89.3	114.3	129.5	124.8
VARIANCE	79819.6	130626	167824.2	155728.7

Amplitude of wave 111 (NANOVOLTS) from 60-90 dB nHL at 4000 HZ.

	60	70	80	90
M.G.	100	368	260	352
A.M.	96	308	352	432
M.O.	230	230	220	412
B.M.	312	260	212	440
L.B.	158	256	256	260
B.W.	268	392	396	720
T.T.	192	292	352	216
V.B.	140	308	260	516
P.W.	144	236	172	332
S.N.	124	260	392	236
G.W.	153	253	300	352
TOTAL	1881	3165	3204	8208
MEAN	176	316	289	382
S.D.	70.1	52.9	73.4	143.8
VARIANCE	49149.9	28056.9	53818.2	207018.7

Amplitude of wave V (NANOVOLTS) from 60-90 dB nHL at 4000 HZ.

	60	70	80	90
M.G.	200	276	492	544
A.M.	312	416	244	428
M.O.	308	304	304	286
B.M.	272	356	416	664
L.B.	212	216	216	205
B.W.	308	376	404	420
T.T.	208	428	296	628
V.B.	416	144	280	460
P W.	216	248	320	500
S.N.	88	212	184	308
G.W.	162	213	311	412
TOTAL	2702	3189	3447	4882
MEAN	246	289	289	442
S.D.	89.0	93.6	92.2	132.0
VARIANCE	79254.5	87632.9	84972	174156.0

Appendix D3. Mean amplitude values for waves I, III and V at 2000 Hz.

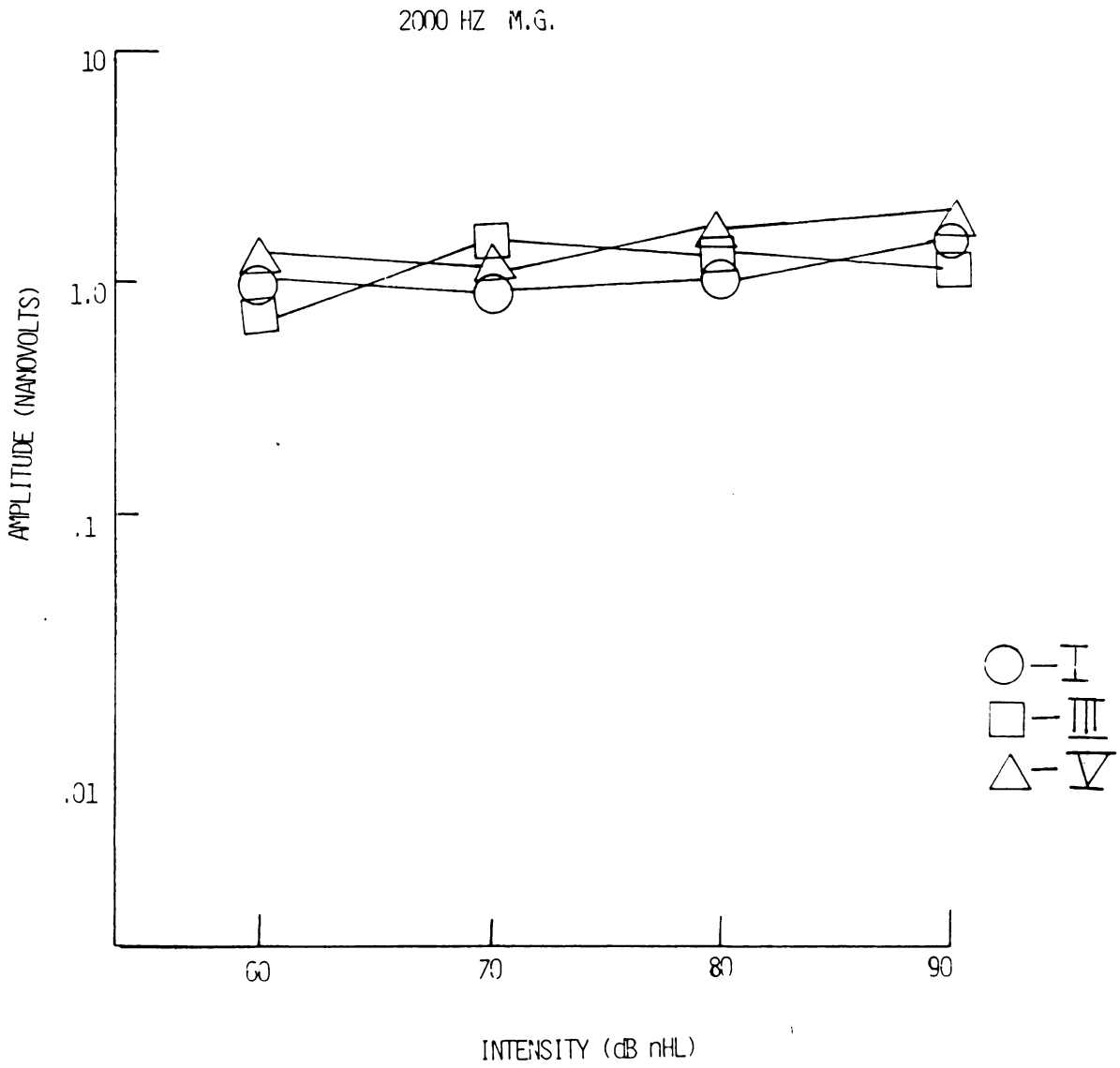
Table 1. Mean amplitude values of waves I, III and V at 2000 Hz.

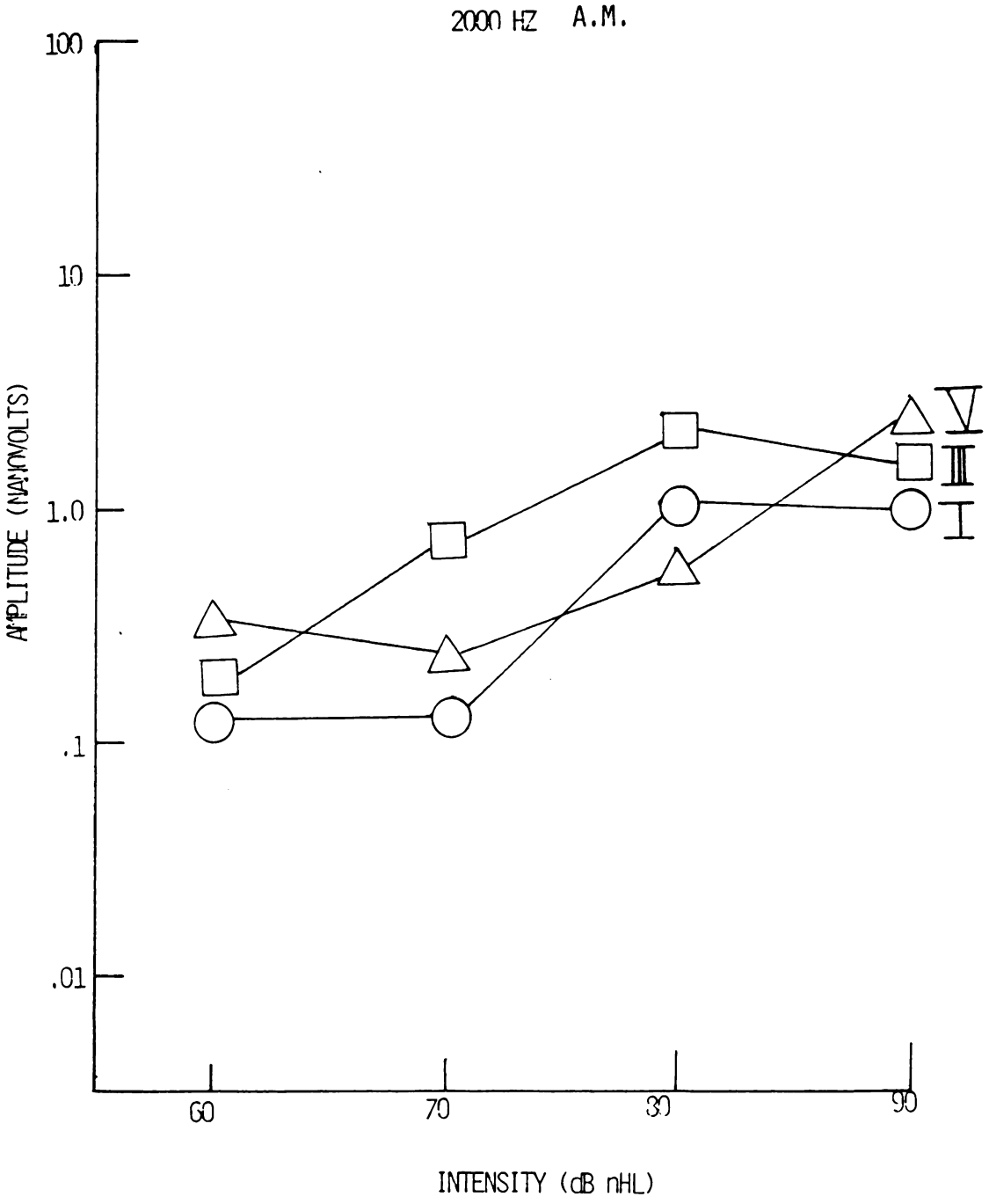
Waves	60	70	80	90
I	75	111	178	265
III	89	141	210	264
V	210	264	326	425

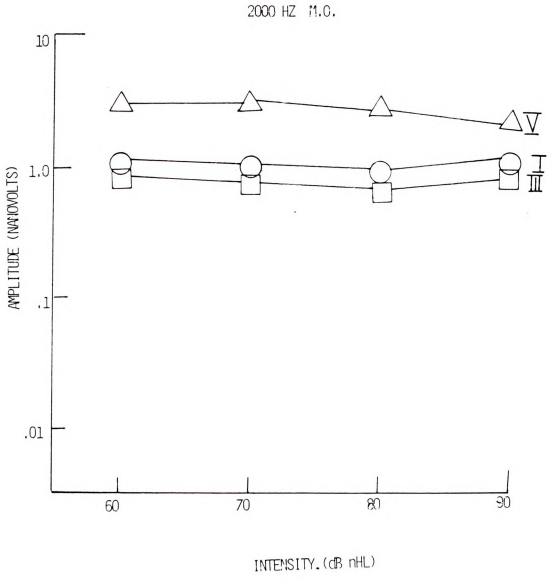
Table 2. Mean amplitude values of waves I III and V at 4000 Hz.

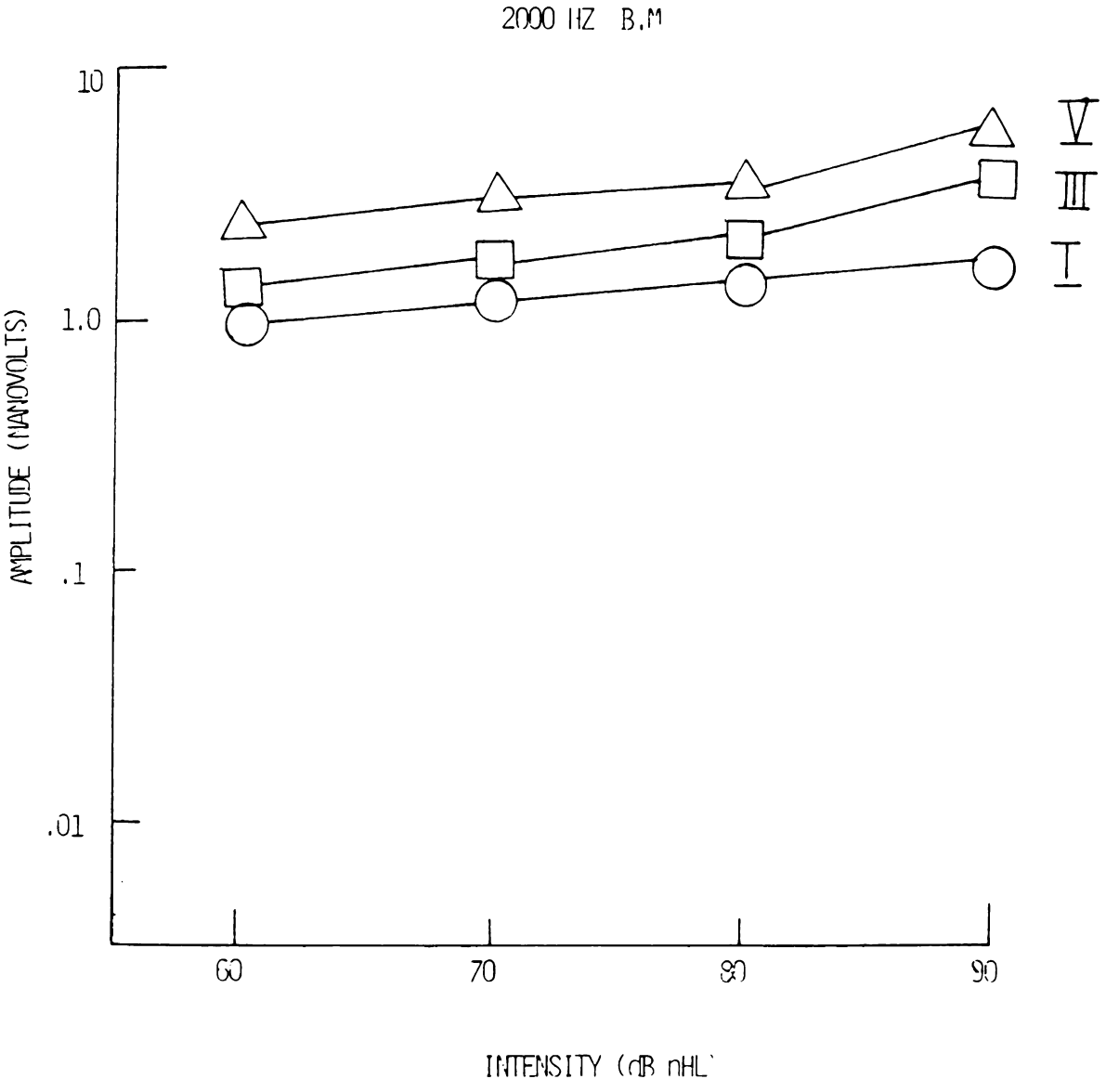
Waves	60	70	80	90
I	180	253	365	374
III	180	316	289	383
V	246	289	324	442

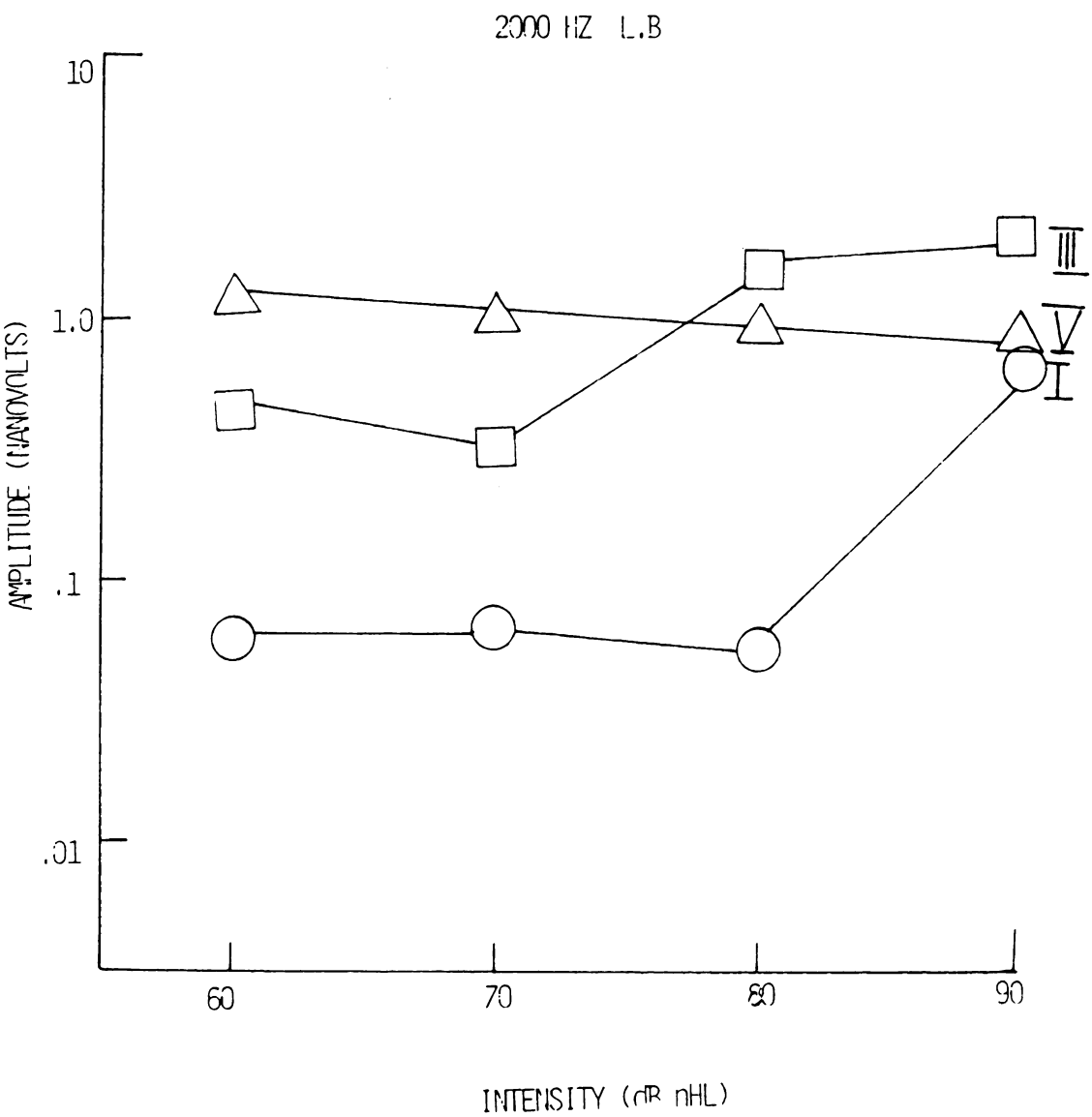
Appendix E1. Amplitude input-output functions for waves I, III and V at 2000 Hz for each subject.

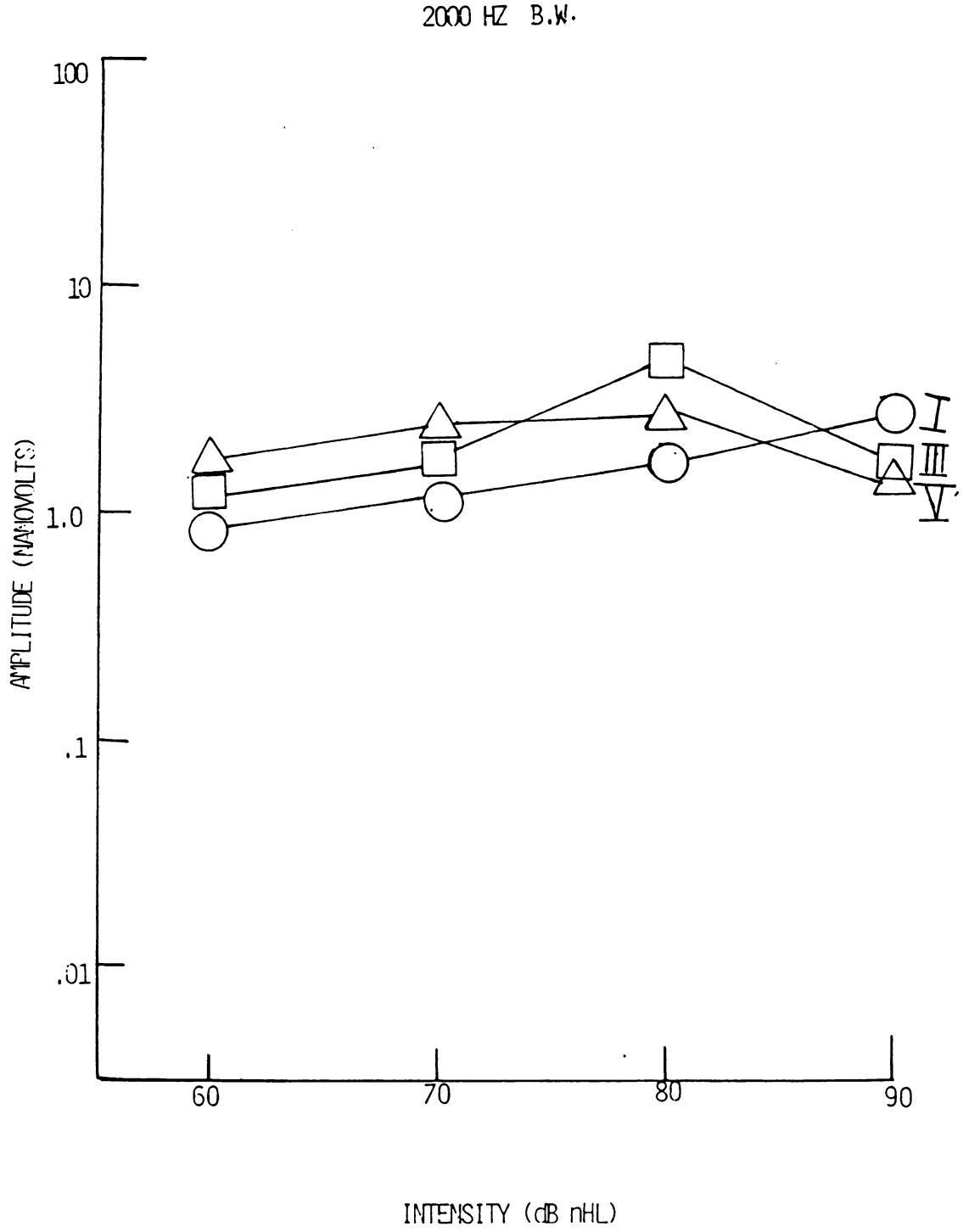

2000 HZ M.G.
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
I
III
V


2000 HZ A.M.
100
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
III
I


2000 HZ M.O.
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY.(dB nHL)
V
I
III


2000 HZ B.M
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
V
III
I
60
70
80
90
INTENSITY (dB nHL)


2000 HZ L.B
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
III
V
I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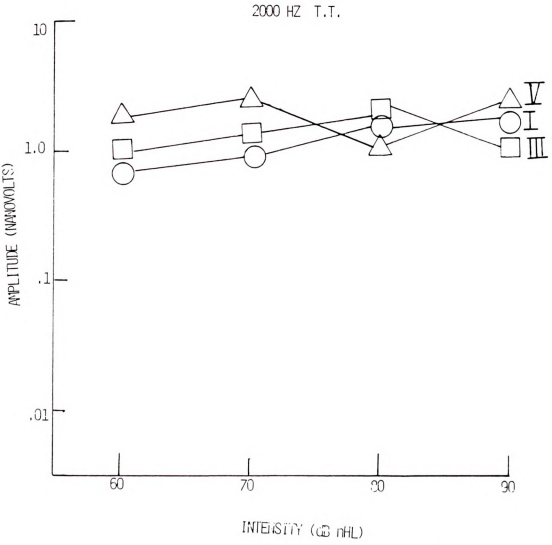
100
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
I
III
V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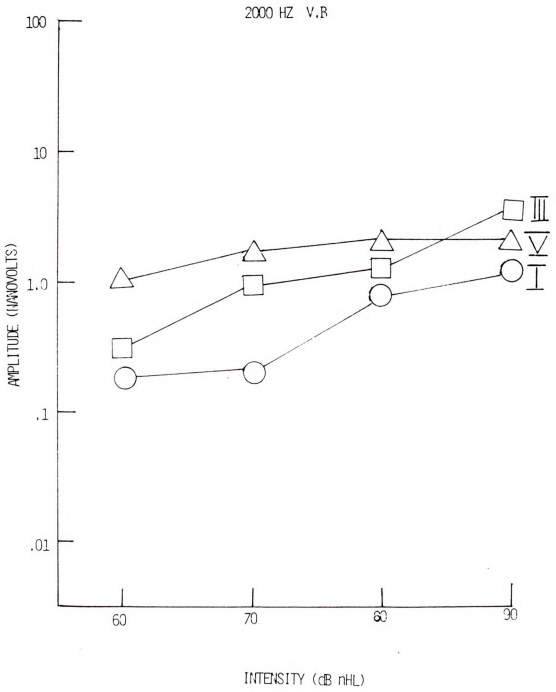
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
I
III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100
10
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AMPLITUDE (NANOVOLTS)
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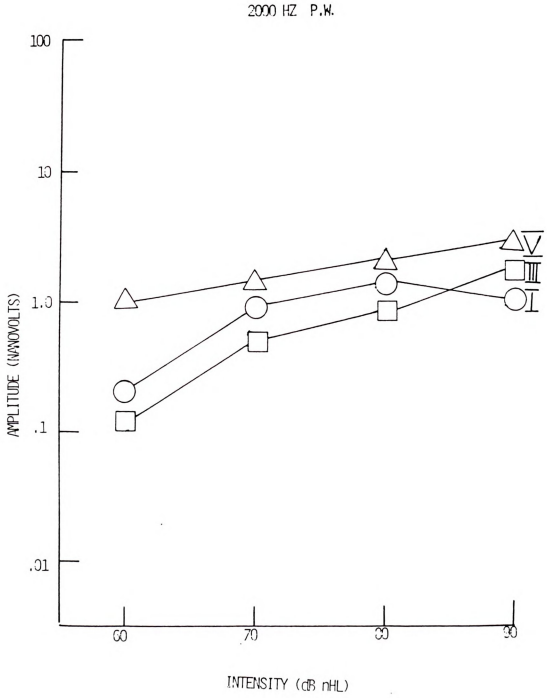
80
90
INTENSITY (dB nHL)
III
V
I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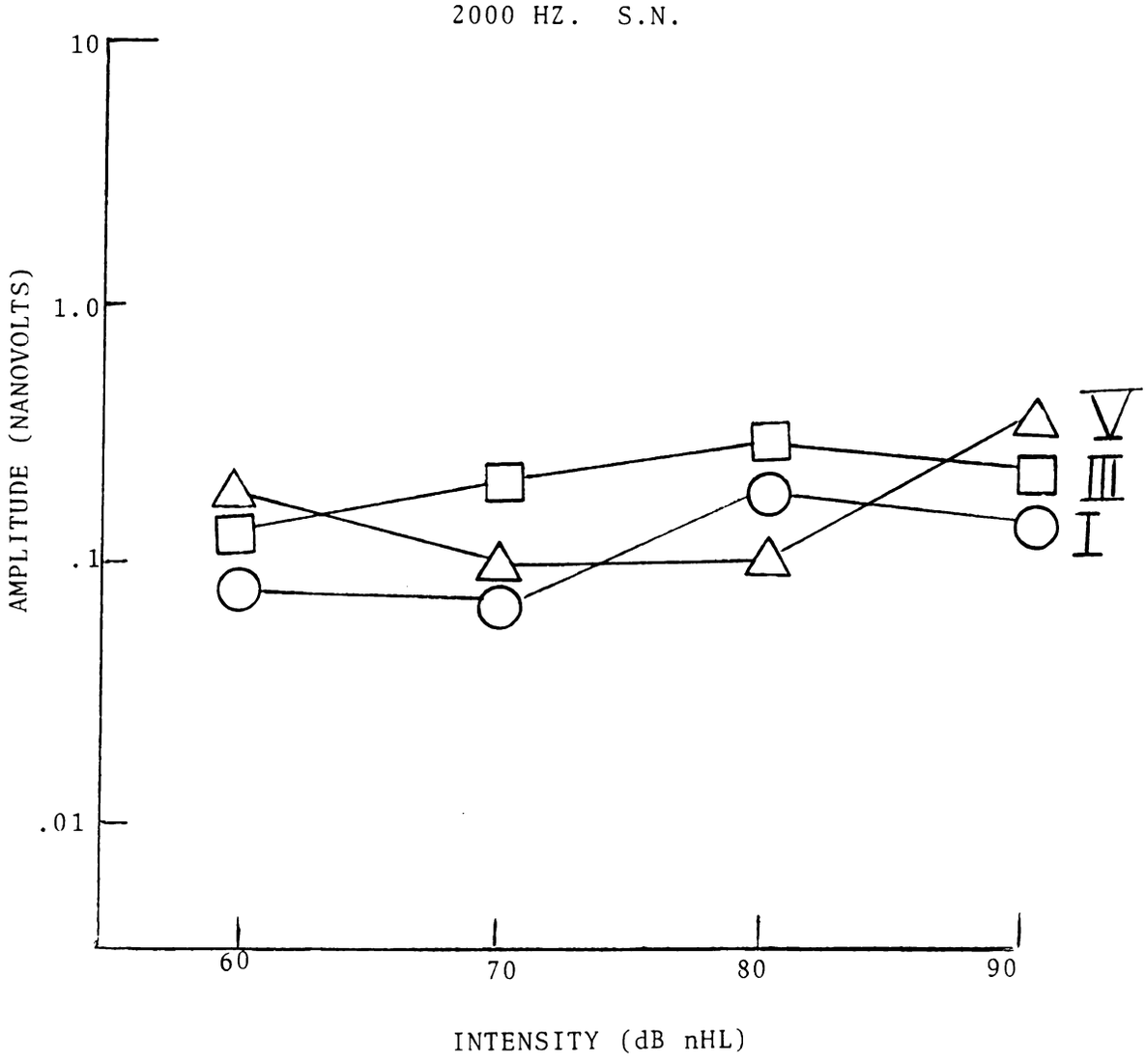
100
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
III
I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1.0
.1
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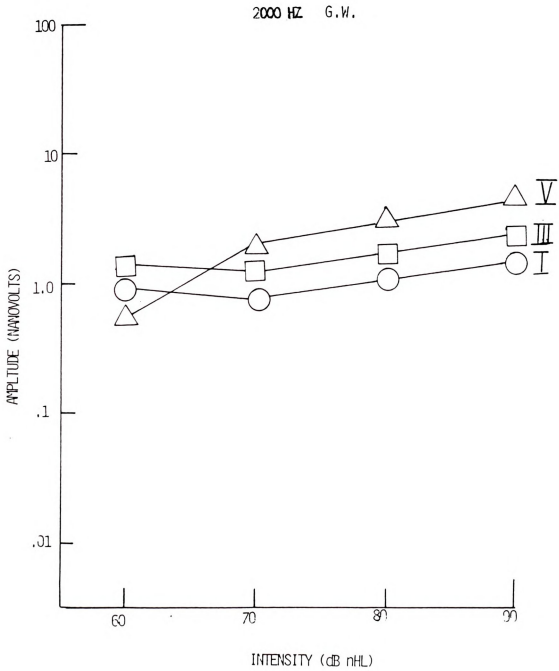
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
III
I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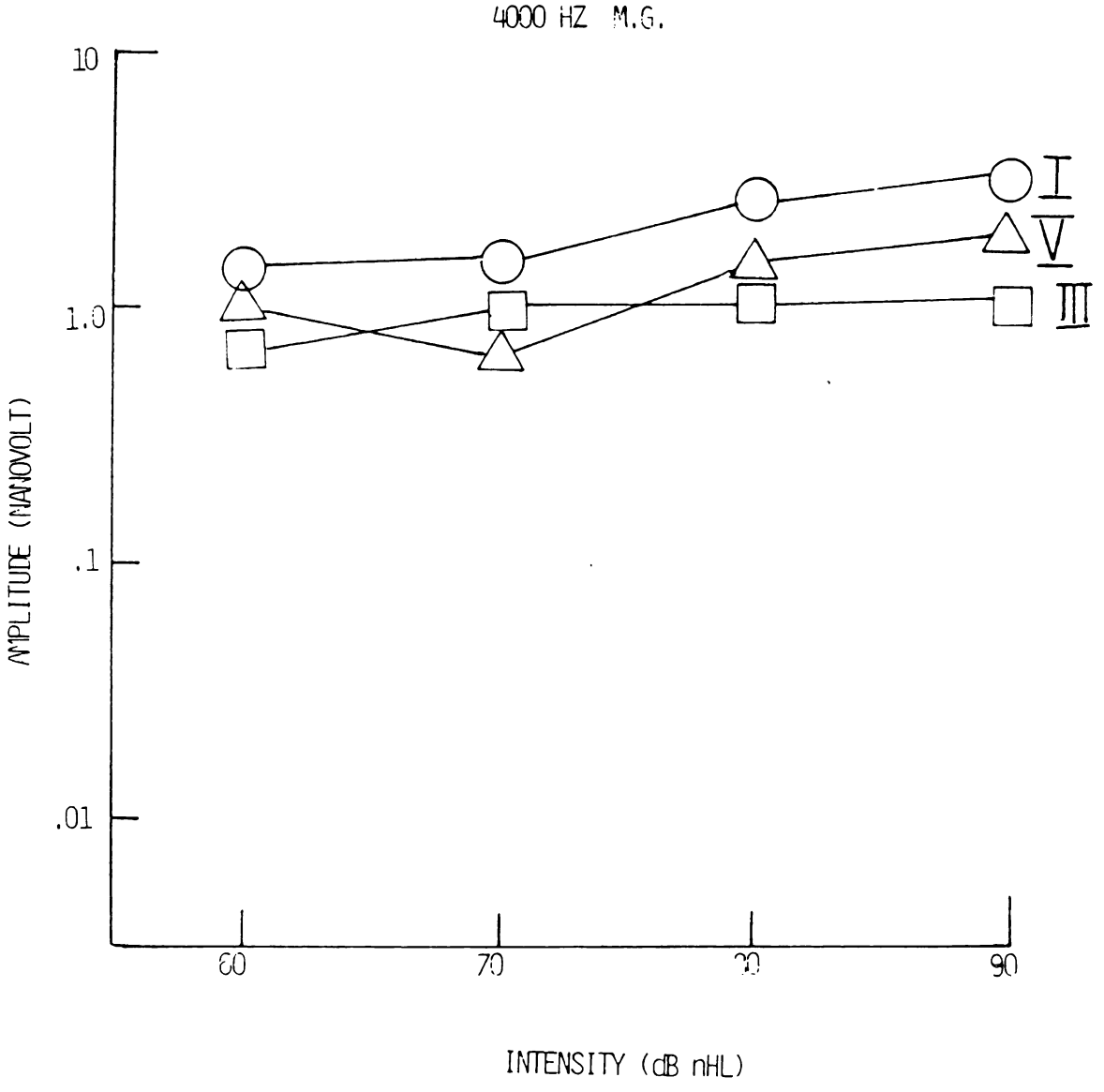
100
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
V
III
I
60
70
80
90
INTENSITY (dB nHL)

Appendix E2. Amplitude input-output functions for waves I, III and V at 4000 Hz for each subject.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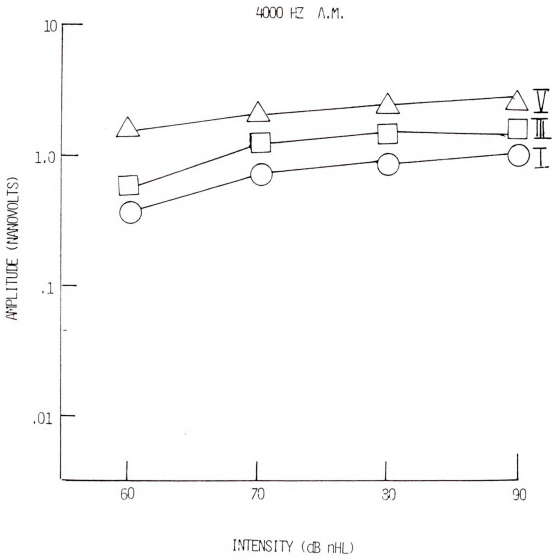
.1
.01
AMPLITUDE (NANOVOLT)
I
V
III
60
70
80
90
INTENSITY (dB nHL)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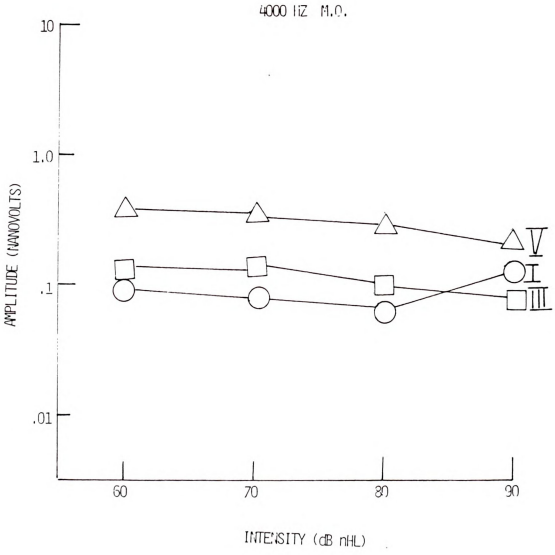
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
III
I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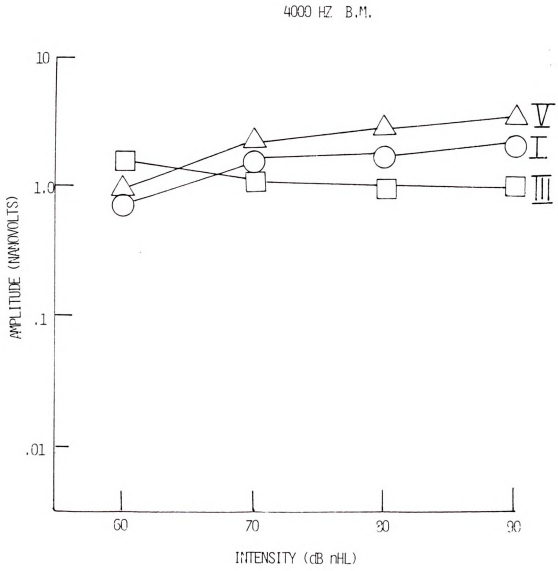
.01
AMPLITUDE (MICROVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
I
III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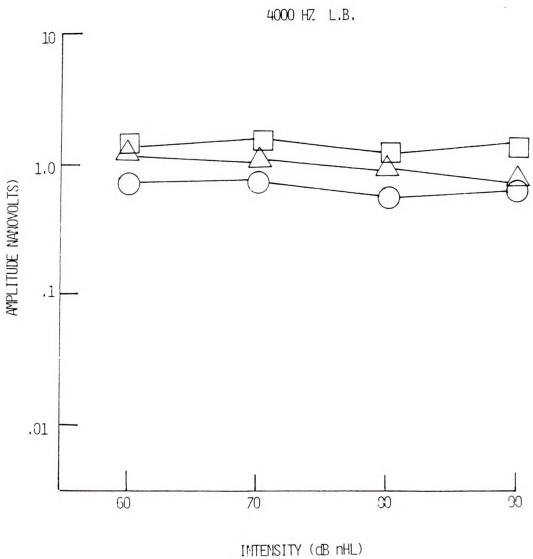
10
1.0
.1
.01
AMPLITUDE (MICROVOLTS)
V
I
III
60
70
80
90
INTENSITY (dB nHL)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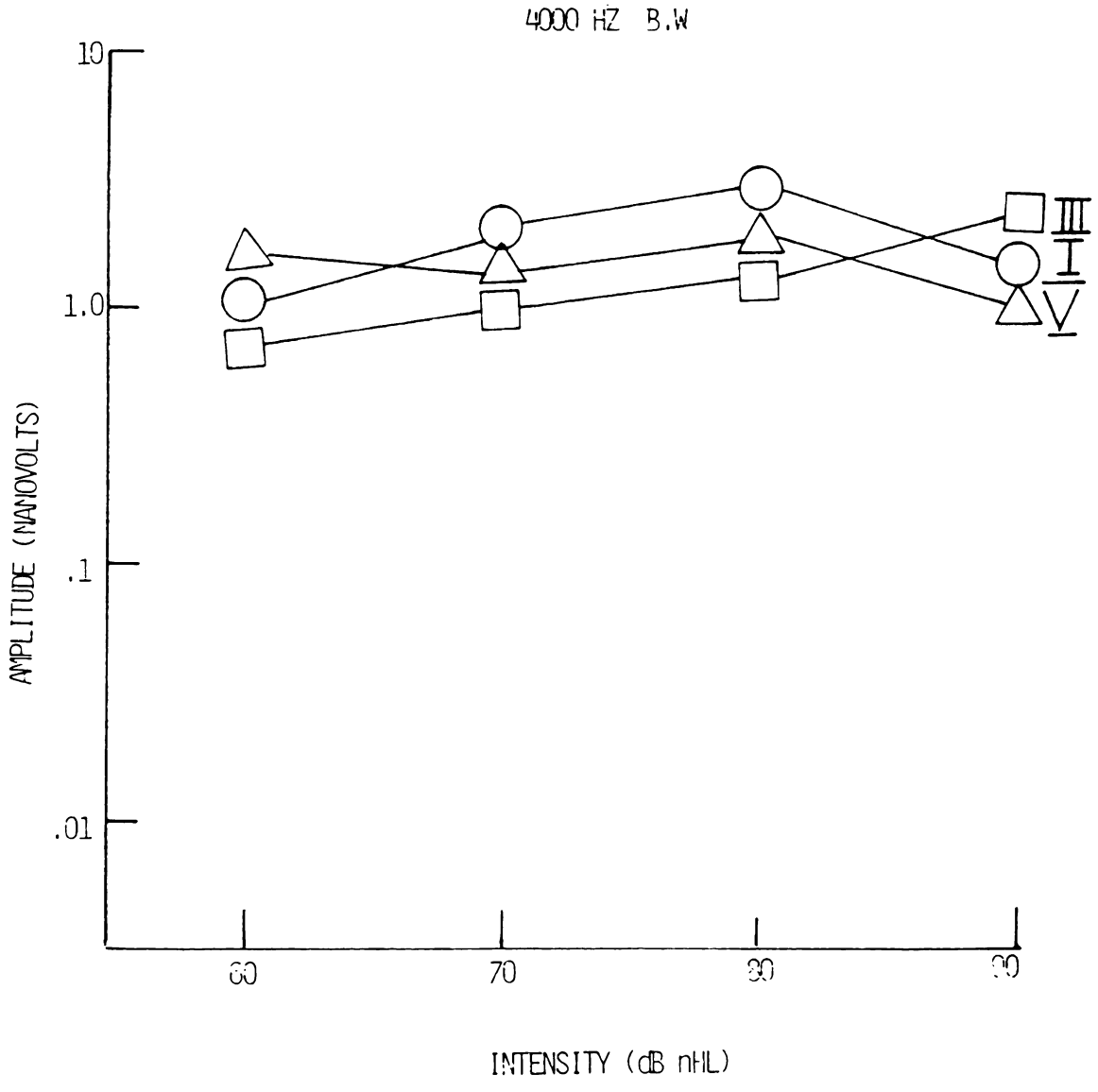
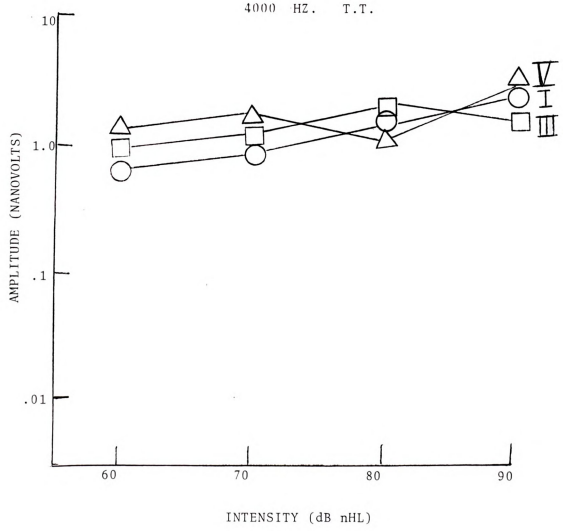
1.0
.1
.01
AMPLITUDE NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)


4000 HZ B.W
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
III
I
V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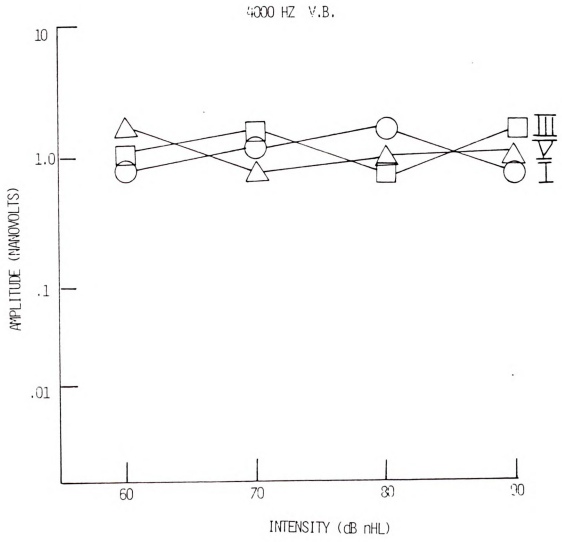
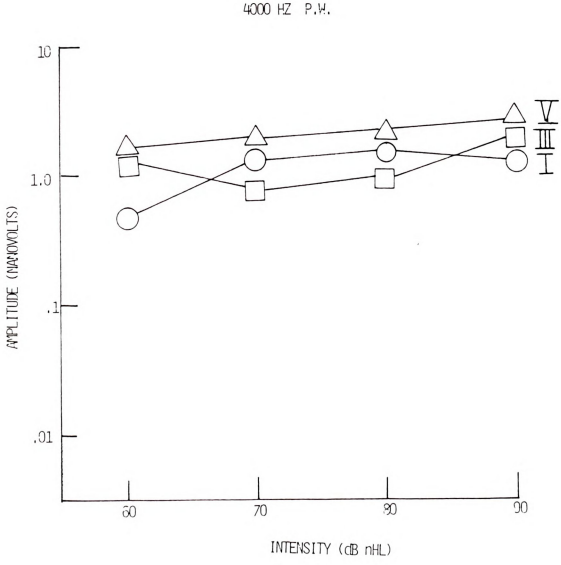
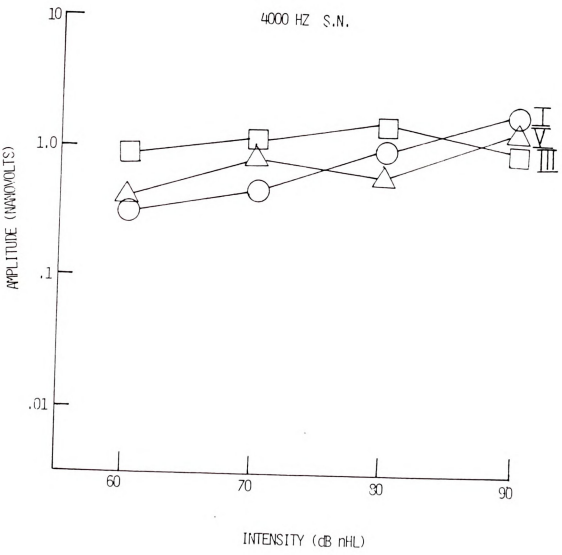
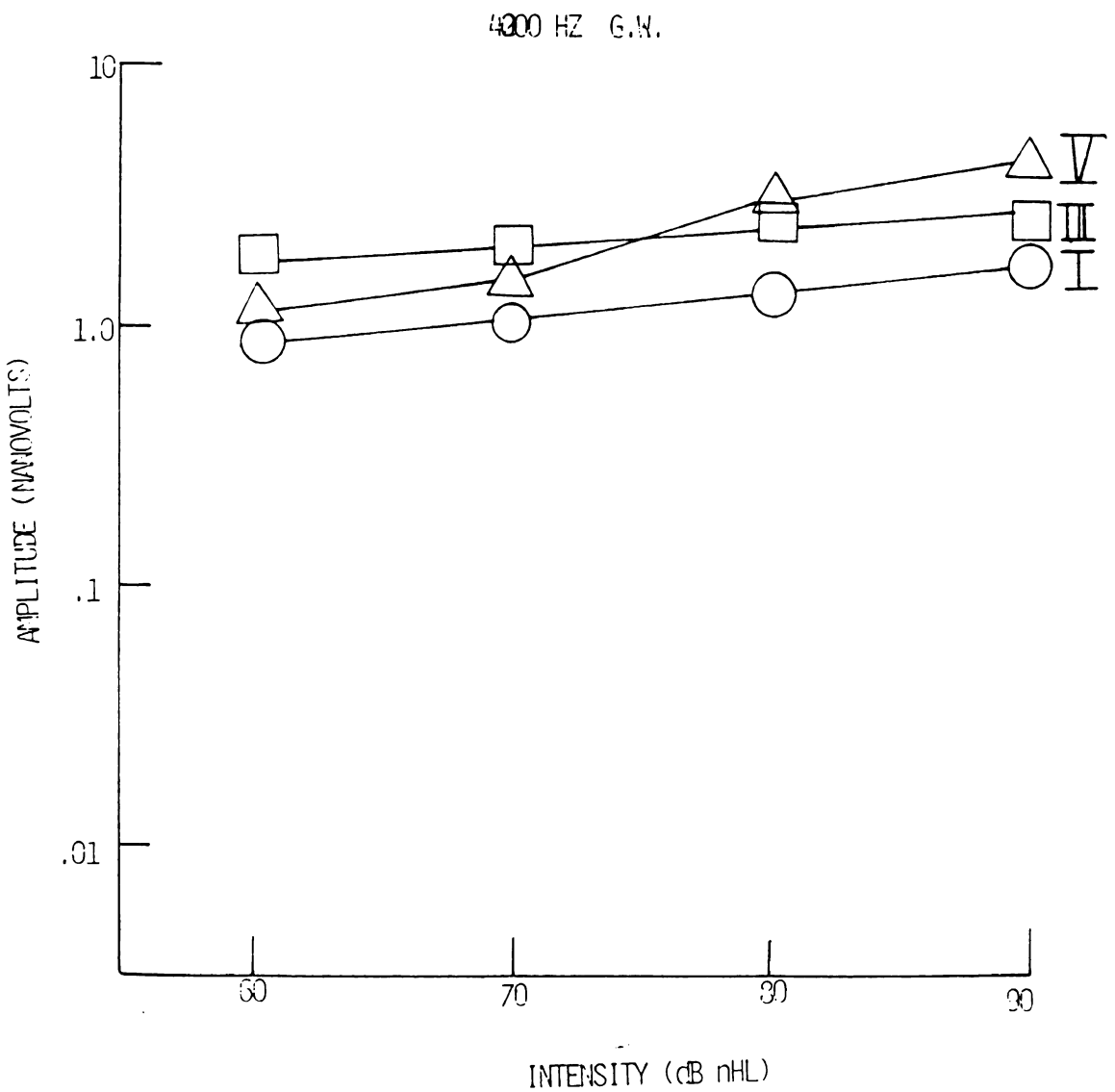
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
I
III


4000 HZ V.B.
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
III
V
I


4000 HZ P.W.
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
80
90
INTENSITY (dB nHL)
V
III
I


4000 HZ S.N.
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
60
70
90
90
INTENSITY (dB nHL)
I
V
III


4000 HZ G.W.
10
1.0
.1
.01
AMPLITUDE (NANOVOLTS)
50
70
90
90
INTENSITY (dB nHL)
V
III
I

Appendix F1. Linear regression latency values for waves I, III and V latency at 2000 and 4000 Hz.

Table 3. Linear regression latency values for waves 1, 111 and V at 2000 Hz.

Waves	Slope	Y-Intercept	R
1	-.02	3.2	-.60
111	-.01	5.5	-.84
V	-.01	6.5	-.45

Table 4. Linear regression latency values for waves 1,111 and V at 4000 Hz.

Waves	Slope	Y- Intercept	R
1	-.02	4.1	-.32
111	-.01	5.2	-.37
V	-.01	6.5	-.77

Appendix F2. Linear regression amplitude values for waves I, III and V at 2000 and 4000 Hz.

Table 5. Linear regression amplitude values for waves 1, 111 and V at 2000 Hz.

Waves	Slope	Y-Intercept	R
1	2.5	151	.21
111	1.6	176	.40
V	1.5	206	.25

Table 6. Linear regression amplitude values for waves 1, 111 and V at 4000 Hz.

Waves	Slope	Y-Intercept	R
1	7.6	167	.62
111	3.7	193	.25
V	1.5	210	.17

Appendix G1. Summary of one-way ANOVA for waves I, III and V for intensity and latency at 2000 Hz.

Table 7. Summary of intensity x Latency x ANOVA for ABR at 2000 Hz.

Wave	Source	SS	DF	MS	F	P	S/NS
1	intensity xLatency	3.083	3	1.027	28.362	.001	S
	Error	1.452	40	.036			
	Total	4.536	43				
111	intensityxLatency	4.400	3	1.466	67.376	.001	S
	Error	.870	40	.021			
	Total	5.271	43				
V	intensity x Latency	4.289	3	1.429	55.473	.001	S
	Error	1.030	40	.054			
	Total	5.319	43				

Appendix G2. Summary of one-way ANOVA for waves I, III and V for intensity and latency at 4000 Hz.

Table 8. Summary of intensity x Latency x ANOVA for ABR at 4000 Hz.

Wave	Source	SS	DF	MS	F	P	S/NS
1	intensity x Latency	2.272	3	.757	21.389	.001	S
	Error	1.416	40	.035			
	Total	3.689	43				
111	Intensity x Latency	1.607	3	.535	28.345	.001	S
	Error	.756	40	.016			
	Total	2.364	43				
V	Intensity x Latency	2.055	3	.685	16.545	.001	S
	Error	1.656	40	.041			
	Total	3.711	43				

Appendix H1. Summary of one-way ANOVA for waves I, III and V for intensity and amplitude at 2000 Hz.

Table 9. Summary of intensity x amplitude x ANOVA at 2000 Hz.

Wave	Source	SS	DF	MS	P	F	S/NS
1	Intensity x Amplitude	268844.4	3	89614.8	11.7	.001	S
	Error	305422.7	40	7635.6			
	Total	574267.2	43				
111	Intensity x Amplitude	319776.5	3	106592.1	14.8	.001	S
	Error	288243.1	40	7206.2			
	Total	608019.6	43				
V	Intensity x Amplitude	302846.7	3	1000948.9	6.9	.001	S
	Error	583134.7	40	14578.4			
	Total	885981.6	43				

Appendix H2. Summary of one-way ANOVA for waves I, III and V for intensity and amplitude at 4000 Hz.

Table 10. Summary of intensity x Amplitude x ANOVA for ABR at 4000 Hz.

Wave	Source	SS	DF	MS	F	P	S/NS
1	Intensity x Amplitue	399407.3	3	133135	9.97	.oo1	S
	Error	533999.5	40	13349.9			
	Total	933406	43				
111	Intensity x Amplitude	248021.9	3	82673.9	9.78	.001	S
	Error	338041.8	40	8451.0			
	Total	586063.7	43				
V	Intensity x Ampliyude	340412.0	3	113470.7	10.65	.001	S
	Error	426015.6	40	10650.4			
	Total	766427.2	43				

Appendix H3. Summary of two-way ANOVA for waves I, III and V for intensity and amplitude at 2000 Hz.

Table 11. Summary of intensity x Amplitude x Subject x ANOVA for ABR at 2000 Hz.

Wave	Source	SS	DF	MS	F	S/NS
1	Intensity x Amplitude	230395	3	76798	10.8	S
	Subjects	235780	10	23578	3.3	S
	ESS	312515	30	7071		
	Total	778690	43			
111	Intensity x Amplitude	387442	3	129147	8.8	S
	Subjects	274436	10	27444	2.9	S
	ESS	438893	30	14629		
	Total	1000771	43			
V	Intensity x Amplitude	300201	3	100067	3.6	S
	Subjects	446024	10	44602	2.7	S
	ESS	728155	30	21605		
	Total	1304199	43			

Appendix H3. Summary of two-way ANOVA for waves 1,111 and V for intensity and amplitude at 2000 Hz.

Table 12. Summary of intensity x Amplitude x ANOVA for ABR at 4000 Hz.

Wave	Source	SS	DF	MS	F	S/NS
1	Intensity x Amplitude	404481	3	134827	7.9	S
	Subjects	344096	10	34410	2.3	S
	ESS	510641	30	17021		
	Total	1259218	43			
111	Intensity x Amplitude	269005	3	89668	5.4	S
	Subjects	78288	10	78291	4.7	S
	ESS	495569	30	16518		
	Total	842862	43			
V	Intensity x Amplitude	234619	3	78206	8.4	S
	Subjects	174740	10	17474	2.7	S
	ESS	274700	30	7136		
	Total	778690	43			

Appendix I. Slope of the input-output latency and amplitude functions for waves I, III and V.

Table 13. Slope of the input-output Latency functions for waves 1, 11 and V at 2000 and 4000 Hz.

Wave	Slope	Range of differences at 60-90 dB
1	2KHz -.02	.7
	4KHz -.02	.5
111	2KHz -.03	1.2
	4KHz .03	.5
V	2KHz-.04	1.5
	4KHz .02	.5

Table 14. Slope of the input-output amplitude functions for waves 1,1 and V at 2000 and 4000 Hz.

Wave	Slope	
1	2KHz	6.3
	4KHz	6.5
111	2KHz	5.7
	4KHz	6.8
V	2KHz	6.7
	4KHz	6.5

Appendix J1. Interpeak latencies (IPL) for waves I, III and V at 2000 and 4000 Hz.

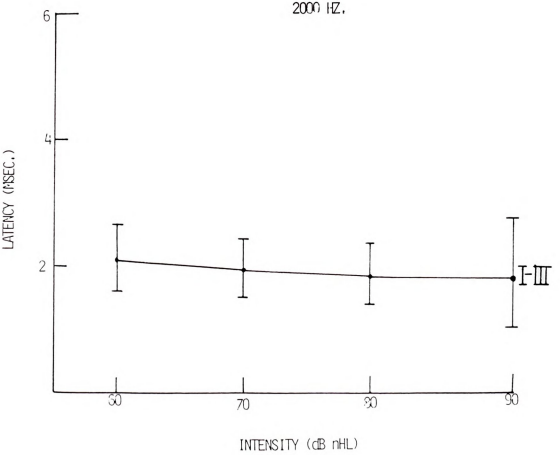
Table 15. Interpeak latencies of waves 1, 111 and V at 2000 Hz.

IPL		60	70	80	90
1-111	X	2.1	2.0	1.9	1.8
	sd	0.15	0.15	0.16	0.32
	x+3sd	2.6	2.4	2.4	2.8
	x-3sd	1.7	1.5	1.4	.88
1-V	x	3.9	3.8	3.7	3.7
	sd	0.29	0.25	0.26	0.30
	x+3sd	4.7	4.5	4.5	4.6
	x-3sd	3.0	3.0	2.9	2.8
111-V	x	1.7.	1.8	1.9	1.8
	sd	0.41	0.25	0.27	0.43
	x+3sd	2.9	2.5	2.7	3.0
	x-3sd	0.45	1.0	1.0	0.60

Table 16. Interpeak latencies of waves 1. 111 and V at 4000 Hz.

IPL		60	70	80	90
I -III	x	2.0	2.0	2.0	2.0
	sd	0.19	0.13	0.07	0.08
	x+3sd	2.6	2.3	2.2	2.2
	x-3sd	1.5	1.6	1.8	1.7
I -V	x	3.9	3.9	4.0	3.9
	sd	0.29	0.20	0.12	0.16
	x+3sd	4.7	4.5	4.3	4.4
	x-3sd	3.0	3.0	3.6	3.4
III-v	x	1.9	2.0	1.9	1.9
	sd	0.27	0.10	0.12	0.09
	x+3sd	2.7	2.3	2.3	1.7
	x-3sd	0.12	1.7	1.6	1.6

Appendix J2. Interpeak latency-intensity function for waves I, III and V at 2000 and 4000 Hz.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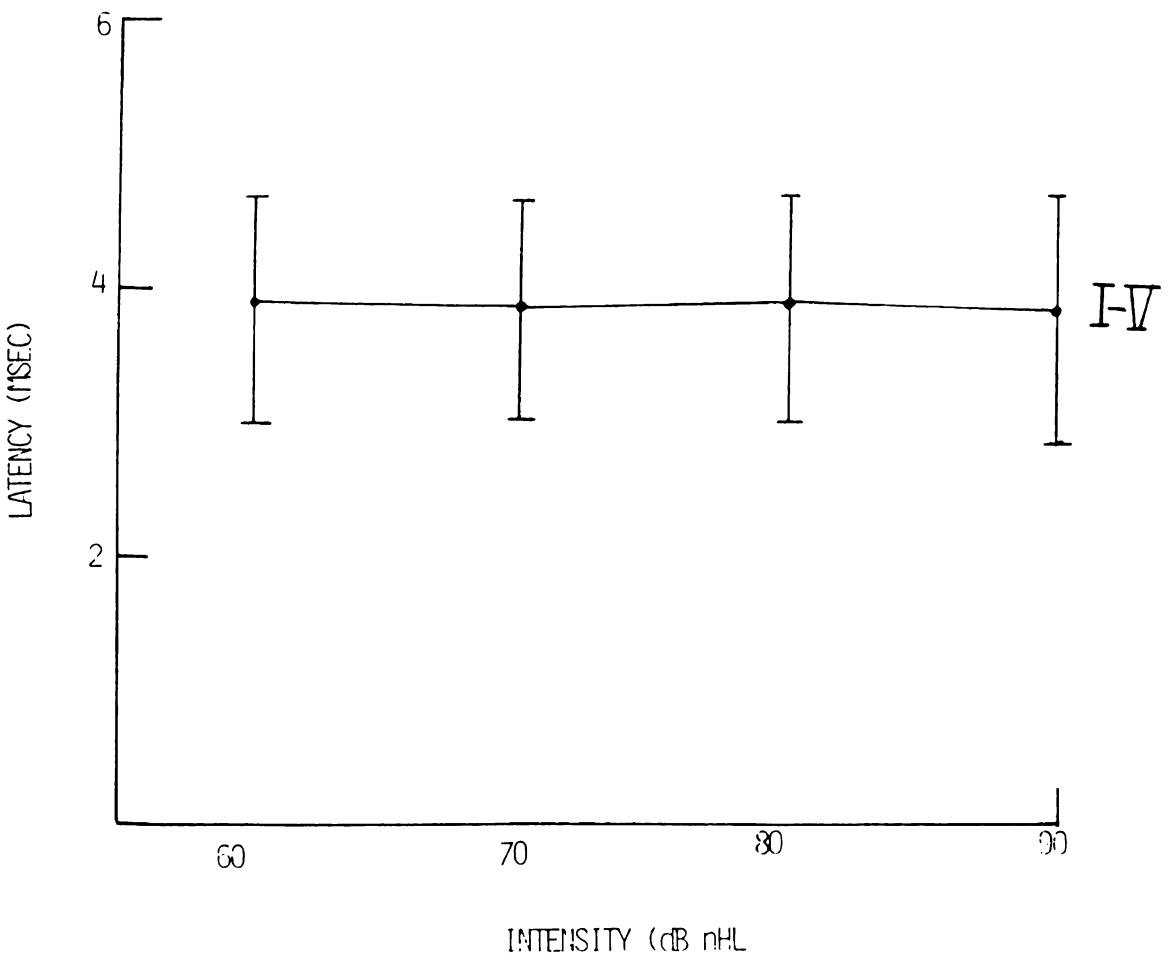
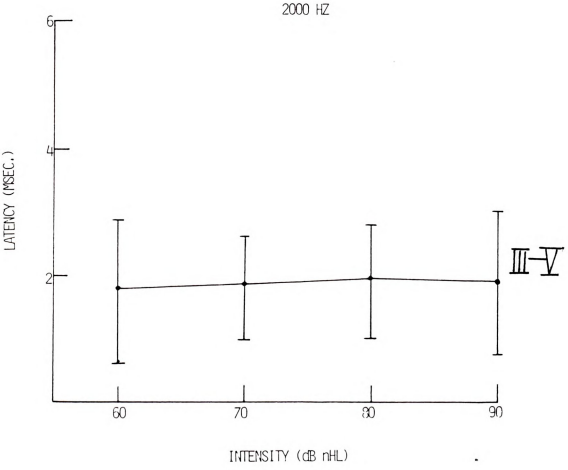
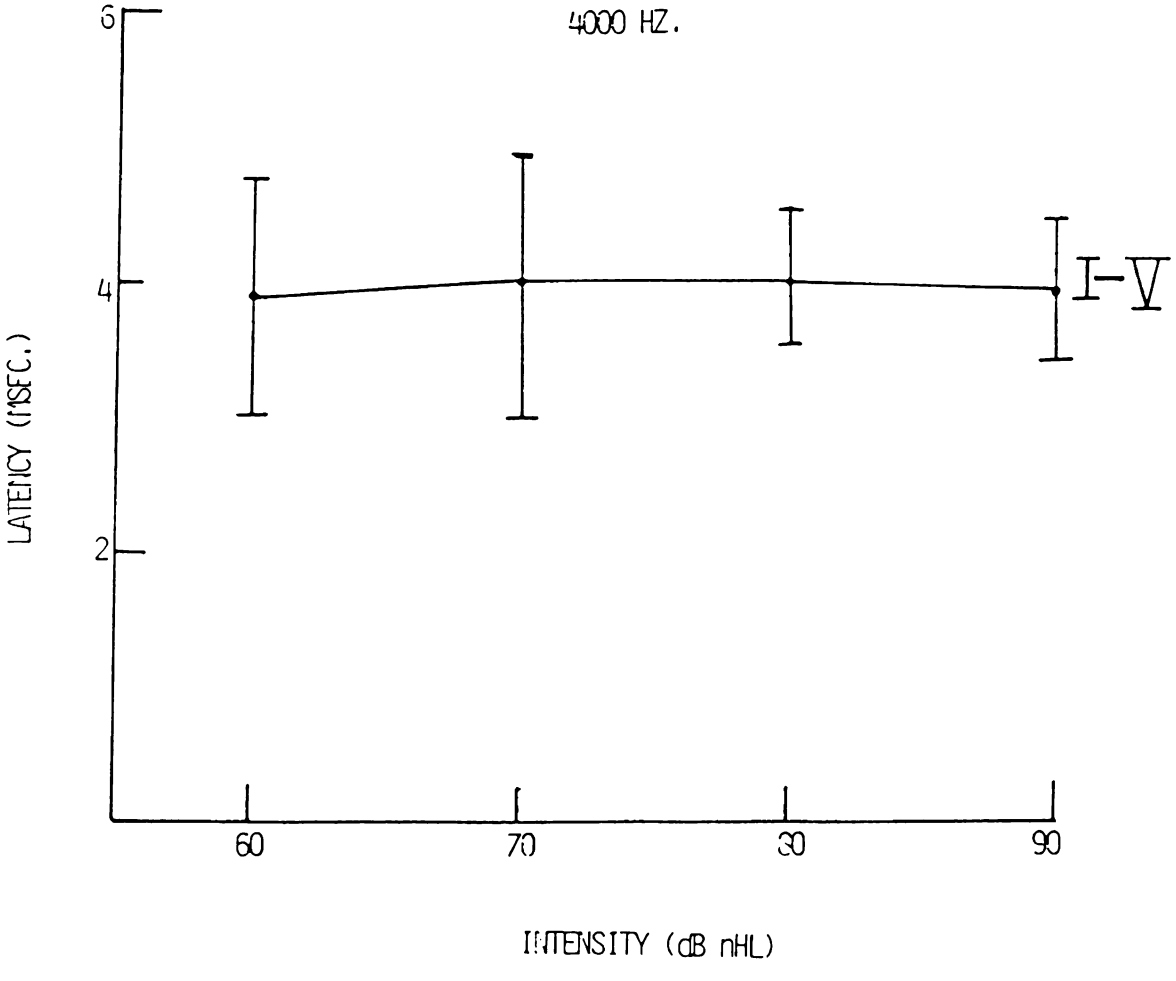
6
4
2
LATENCY (MSEC.)
I-III
50
70
90
INTENSITY (dB nHL)

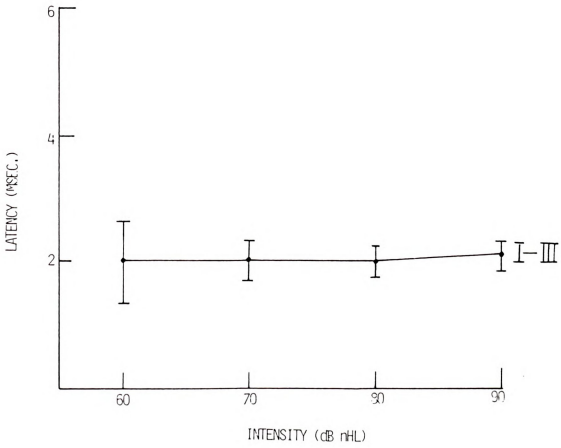
2000 HZ.

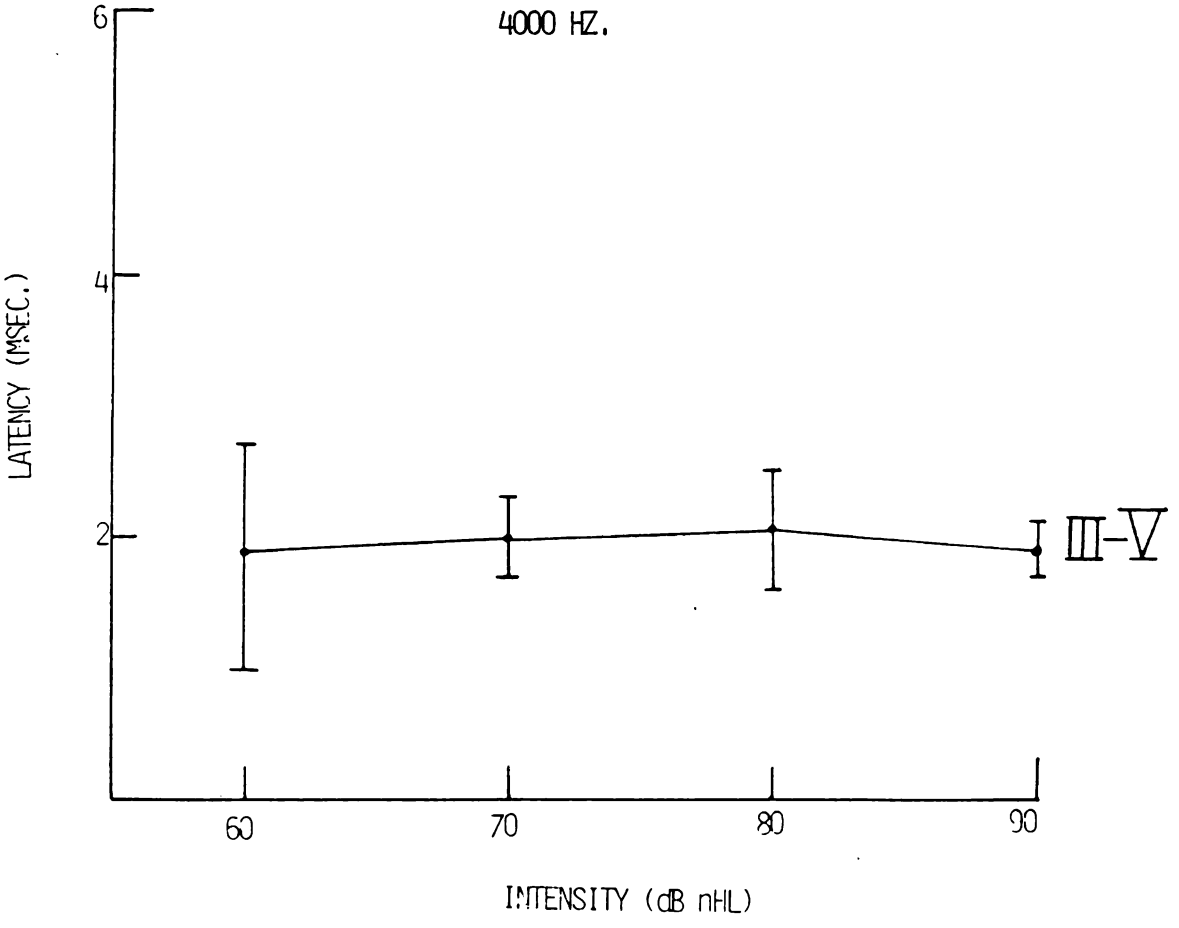



2000 HZ
6
4
2
LATENCY (MSEC.)
III-V
60
70
80
90
INTENSITY (dB nHL)


4000 HZ.
6
4
2
LATENCY (MSEC.)
I-V
60
70
80
90
INTENSITY (dB nHL)

4000 HZ




4000 HZ.
6
4
2
LATENCY (MSEC.)
III-V
60
70
80
90
INTENSITY (dB nHL)

Appendix K. Relative amplitude values for waves I, III and V at 2000 and 4000 Hz.

Table 17. 1/V amplitude ratio at 2000 Hz.

	60	70	80	90
1/V	1.6	1.1	1.1	1.2
sd.	.51	.61	.72	.95

Table 18. 1/V amplitude ratio at 4000 Hz.

	60	70	80	90
1/V	2.9	2.3	1.8	1.6
sd.	.96	8.7	8.4	7.8