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AUDITORY EVOKED POTENTIALS FROM
HEARING-IMPAIRED AND NORMAL-HEARING HUMANS

by

ROBERT LEE FOLMER

DISSERTATION

Submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

SPEECH AND HEARING SCIENCE

in the

GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA

San Francisco



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by
Robert Lee Folmer

DEDICATION

I dedicate this dissertation to my parents, Lottie and Roy Folmer, and to my wife Kim. Their encouragement and support made it possible for me to complete this long and winding road. Thank you for everything.

PREFACE

I thank the following people for their help and guidance:

The members of my Qualifying Committee: Chuck Yingling, Mike Merzenich, Christoph Schreiner, David Galin, and Robert Sweetow.

The members of my Dissertation Committee: Chuck Yingling, Mike Merzenich, and David Woods. I am particularly grateful to Drs. Yingling and Merzenich for years of training, encouragement, and invaluable suggestions. This dissertation research took place in Dr. Yingling's labs at U.C.S.F. I thank him for the opportunity to conduct these studies. Also, I thank him and Margie Harrington for showing me how to record the electrical activity generated by the human brain. I thank Dave Becker and Roger Noss for their help in mapping these evoked potentials.

The excellent instructors I've had at U.C.S.F. and San Francisco State: John Williston, Peggie Phillips, Bob Turner, Bill Jenkins, Pat Leake, Mike Merzenich, Christoph Schreiner, Chuck Yingling, John Gardi, Pete and Diane Ralston, Allan Basbaum, Mike Stryker, Steve Lisberger, Howard Fields, Alan Salamy, Zach Hall, Lou Reichardt, Earl Mayeri, Julie Schnapf, and the Jans among others.

The many volunteers who sacrificed their hairstyles in order to donate their brain waves to science.

Michael Strong and the staff at the Center On Deafness.

Deaf people do not usually live in total silence. Most can hear the bass of a song or a voice or a heartbeat if it's loud enough. We should all listen more carefully to each other's songs and words and hearts.

ABSTRACT

Auditory Evoked Potentials From Hearing-impaired

and Normal-hearing Humans

Robert Lee Folmer

Long latency auditory evoked potentials (AEPs) were recorded from eight normal adults and from eight adults with profound bilateral high-frequency sensorineural hearing impairment. Stimuli included low frequency tones, noise bursts, and speech sounds. Grand averaged AEP waveforms and topographic maps were constructed for each experimental group. The findings of this study are:

1) Compared to N1 components recorded from subjects with normal hearing, latencies of N1 components were delayed in hearing-impaired responses to speech stimuli. Delays in processing of speech stimuli were not duplicated in normal-hearing subjects by reducing stimulus intensity. However, they were duplicated by using masking noise to simulate hearing loss.

2) In the tonal "oddball" paradigm, even though N1 and P2 amplitudes were smaller; and N1 and P2 latencies were greater from hearing-impaired subjects, P3 amplitudes and latencies were not significantly different from those recorded from normal subjects. These results indicate that profound hearing loss does not in itself cause a delay in the generation of this cognitive component.

3) Normal-hearing subjects produced large P3 components in response to their own first name, but not to other names. P3 components were not recorded from hearing-impaired subjects in response to their own name. This protocol could be

useful for assessing name recognition in non-responsive patients.

4) Topographies of AEPs recorded from subjects with normal hearing were different in response to tonal vs. vowel vs. consonant-vowel stimuli. These differences in distributions of AEP components probably represent differential neural processing of these stimuli.

5) Topographies of AEP components were different between the normal and hearing-impaired groups in responses to complex stimuli. Distributions of components recorded from normal subjects in response to the word "POT" remained distinct from hearing-impaired topographies even during conditions of simulated hearing loss. These findings support the hypotheses that AEP topographies are reflections of neural organization and that differences exist in the organization of central auditory pathways between normal and hearing-impaired humans.

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INTRODUCTION

Hans Berger, a German psychiatrist at the University of Jena, was one of the first clinicians (1929) to use scalp electrodes to record electrical activity generated by the brains of patients. This electrical activity is now routinely amplified and recorded as the electroencephalogram (EEG). Davis (1939) noticed consistent changes in the EEG in response to repeated auditory stimulation. These auditory evoked potentials were best recorded from the vertex, and were initially called "V potentials." However, the responses were quite small and difficult to measure within the ongoing EEG record.

The development of averaging computers facilitated more accurate analyses of small bio-electrical signals. Dawson (1951) was the first to describe a summation technique for the detection of evoked potentials within ongoing EEG. Sohmer & Feinmesser (1967) used this technique to record short latency auditory evoked responses from the vertex. Jewett et al. (1970) traced the origin of these early waves to the brainstem. Now known as the auditory brainstem response (ABR), this series of waves occurs approximately 0-10 msec after stimulus onset. Recordings of middle latency responses (or MLR -- a series of waves occurring approximately 10-65 msec after stimulus onset) were first published by Geisler et al. (1958) and their characteristics were described by Mendel & Goldstein (1969a,b). The "V potentials" (which occur after the MLR) have also been called cortical potentials (Cody et al., 1964), slow responses (Davis, 1965), long-latency or late auditory responses. Long latency responses include the N1 (negative voltage recorded at the scalp surface approximately 100 msec post-stimulus onset), P2 (positive voltage recorded approximately 200 msec post-stimulus onset), and

P3 (positive voltage recorded approximately 300 msec post-stimulus onset) waves.

Evoked potentials (EPs) of the brain are valuable tools for studying the neural mechanisms of cognition because they can be recorded non-invasively in both normal and clinical populations (see Hillyard & Picton, 1987; Regan, 1989; or Hall, 1992 for comprehensive reviews). EP components can be divided into two classes: 1) exogenous, or stimulus-driven; and 2) endogenous, which are associated with cognitive evaluation of stimulus significance (Donchin et al., 1978). Exogenous components elicited from human adults are obligatory responses to the stimulus, and are largely determined by the physical characteristics of the stimulus. Endogenous components are elicited only when stimuli are novel or task-relevant. The most extensively-studied endogenous component is the P3 which typically accompanies detection of "rare" or "target" stimuli (see Figure 1 below - from Owens & Kessler, 1989 - adapted from Picton et al., 1977).

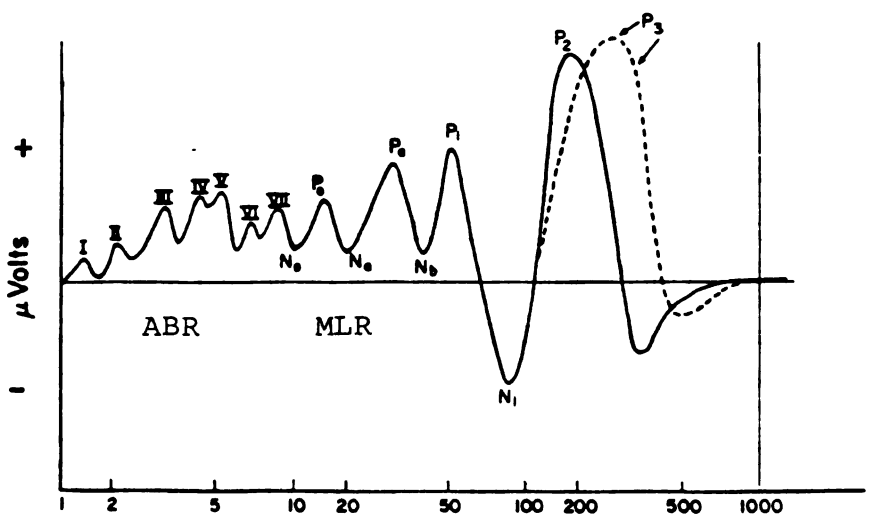


Table 1 (below) summarizes the latencies and proposed neural generators for most of the auditory evoked potentials illustrated in Figure 1. Note that most components probably have multiple neural generators.

	WAVE	NORMAL ADULT LATENCY (MSEC)	GENERATOR(S)
ABR	I	1.5-1.9	Auditory nerve
	II	2.6-3.0	Auditory nerve; cochlear nuc.
	III	3.7-4.1	Trapezoid body; superior olive
	IV	4.8-5.4	Superior olive; lateral lemniscus
	V	5.4-6.0	Lat. lemnis.; inferior colliculus
	VI	7.0-7.6	Inf. collic.; medial geniculate body
	VII	8-9	MGB; primary auditory cortex
MLR	Po	10-13	MGB; auditory cortex ?
	Na	15-25	MGB; auditory cortex ?
	Pa	25-35	MGB; auditory cortex
	Nb	35-45	MGB; auditory cortex ?
	P1	40-65	MGB; primary auditory cortex (AI)
LONG LATENCY	N1	75-140	AI; superior temporal gyrus; *plana temporale* and *polare*
	P2	150-230	AI; superior temporal gyrus ?
	P3	250-350	Reticulothalamus; med. septal area; frontal cortex ?

Mean latency values ± 1 standard deviation from Chiappa et al. (1979); Spehlmann (1985); and Hall (1992). Stimulus = 60 dB SL click or tone pip.

Evoked Response Audiometry

All of the exogenous components of auditory evoked potentials (AEPs) have been used in clinical audiometry under the collective names electric response audiometry or evoked response audiometry (ERA in either case). Long latency responses were the first to be used in audiometric testing (Cody et al., 1964; Rapin, 1964; Davis, 1965). The goal was to develop tests of hearing sensitivity which could be used with patients who were unable to respond during standard audiometric procedures (especially newborns and infants). Results from electrophysiological measures compared favorably (within 5-10 dB) with hearing thresholds determined by standard audiometric tests (McCandless & Best, 1966; Davis et al., 1967).

Early investigators who recorded long latency AEPs from hearing-impaired subjects were most interested in threshold determinations which could be correlated with behavioral audiometry. Because of this emphasis on threshold determination, AEP component amplitude and latency data were often omitted from published reports (for example: Davis, 1965; Cody & Bickford, 1965).

Roeser & Rose (1968) recorded P1, N1, and P2 response components from 10 subjects with normal hearing and from 10 subjects with moderate to severe impairment. Stimuli were 4000 Hz tone bursts, 250 msec in duration, delivered at 15 and 30 dB above threshold. Roeser & Rose reported lower component amplitudes from the hearing-impaired subjects when compared to the normal-hearing group. However, the results were not statistically significant ($p > .05$). The authors also reported no significant difference ($p > .05$) in component latencies between the subject

populations for either stimulus intensity. It is likely that the decision to incorporate individuals with a wide range of impairment (from 35 to 90 dB) into one experimental group gave rise to large standard deviations of responses within the group. This would decrease the statistical significance of the results.

Sharrard (1969) recorded N1 response components from hearing-impaired and normal-hearing children to 1000 Hz tones, 260-290 msec in duration. He reported no significant difference in N1 latency between the groups when stimulus intensity was 90 dB. Sharrard neglected to specify the severity of hearing impairment of his subjects.

Clayton & Rose (1970) recorded AEPs from 8 subjects with unilateral sensorineural hearing impairment. Responses to 4000 Hz, 250 msec duration tones were compared between the normal and impaired ears of the subjects. Clayton & Rose reported no significant differences in P1 or N1 latency or amplitude in response to tones delivered to each ear. However, the authors neglected to deliver masking noise to the ear not receiving tonal stimulation.

In their AEP study of 20 subjects with unilateral sensorineural hearing loss, Townsend & Cody (1970) did mask the unstimulated ear with narrow-band noise. Evoked potentials were compared between normal and impaired ears in response to 30 msec duration 500, 1000, and 2000 Hz tones delivered at 50 dB SL. Townsend & Cody reported that mean P1 and N1 latencies were <u>shorter</u> (by 10.5 and 16.2 msec respectively) for impaired ears than for normal ears. Mean component amplitudes were one third <u>larger</u> for impaired ears. These findings are obscured by the fact that

Townsend & Cody did not specify (or measure?) the severity of hearing impairment of their subjects. Townsend & Cody suggested that enhanced cortical responses from stimulation of impaired ears is probably indicative of recruitment.

Because of their pre-occupation with threshold determination, the aforementioned investigators did not publish all of the data necessary to construct complete AEP profiles of hearing-impaired populations. Thorough, well-controlled, long latency AEP studies of hearing-impaired subjects were never undertaken because not long after Jewett & Williston (1971) suggested the clinical utility of the ABR, it began to replace long latency potentials in evoked response audiometry. This occurred for several reasons: 1) ABRs can be collected faster than long latency responses; 2) ABR is less affected when the subject is asleep or sedated -- this is particularly advantageous when recording responses from newborns or infants; 3) long latency responses are less stable and predictable than ABR -- especially in newborns and infants.

According to Regan (1989), ABR is "one of the leading clinical tools in the diagnosis of peripheral auditory dysfunction and in the assessment of brainstem integrity." (p. 264) While this statement is true, other researchers have suggested the value of using long latency AEPs in conjunction with (or instead of) ABR to evaluate the central auditory system. Pijl et al. (1988) concluded that "integration of ABR findings with those of cortical potentials may, in certain patients, resolve apparent discrepancies between behavioral and electrophysiological data, and provide additional diagnostic and audiologic information." (p. 366) Writing about long

latency AEPs, Keith & Pensak (1991) stated "Although these evoked potential techniques are well known, their specific applications to investigations of central auditory function, language, and learning disabilities have not yet been realized. Systematic investigation using these techniques among special populations can potentially increase our understanding of the central auditory nervous system." (pp. 378-379)

Below are several potential applications of long latency AEPs in evoked response audiometry:

1) Long latency AEPs can be used to evaluate hearing at a complete range of frequencies not possible using ABR. Laukli & Mair (1985) demonstrated that ABR is not reliable for the detection of low frequency hearing loss. Short durations (or high frequencies if clicks are used) of ABR stimuli often are not processed by the auditory systems of hearing impaired subjects. Telian & Kileny (1989) used 1000 Hz tone bursts instead of clicks (which typically have much of their energy in the 2000 to 4000 Hz range) as stimuli in their ABR evaluations of 17 patients with acoustic neuromas. However, even this improved ABR method could not detect lower frequency hearing loss.

2) Long latency AEPs can be used to evaluate auditory processing at the cortical level. Vaughan & Ritter (1970), Wood & Wolpaw (1982), Peronnet et al. (1984), and Woods (1995) have demonstrated that the N1 component is generated by neurons within auditory cortex. Long latency AEPs could be particularly useful in the audiologic assessment of brain injuries. According to Musiek & Baran (1987), ABR

"is not sensitive to lesions of the high brain stem or cortex." (p. 26S) Knight et al. (1980) described abnormal patterns of long latency auditory responses which corresponded with specific unilateral lesions of either frontal or temporal-parietal cortex. Rudge (1983) reported abnormal late AEPs in a case of cortical deafness. Harris & Hall (1990) used long latency AEPs to monitor the progress of 50 brain injury patients undergoing rehabilitation.

3) Long latency auditory potentials can be used to investigate normal and disordered cognitive processes. Applications include studies of the effects of aging (Picton et al., 1984), dementia (Gordon et al., 1986; Goodin & Aminoff, 1986), schizophrenia (Pfefferbaum et al., 1984) and learning disorders (Byring & Jarvilheto, 1987; Dawson et al., 1989).

4) Long latency auditory potentials can be used to evaluate cochlear prothesis recipients pre- and post-operatively. Pelizzone et al. (1986), Oviatt & Kileny (1991) and Kaga et al. (1991) used long latency AEPs to evaluate cochlear prosthesis recipients after implantation. However, no studies to date have compared pre-implantation AEPs with post-implantation responses from the same patients.

5) Long latency AEPs can be used to evaluate the processing of speech. This could be one of the most practical applications of late AEPs. The scale often used for estimating the extent of hearing loss (developed by the Committee on Hearing of the National Research Council, 1949) is based on a person's ability to comprehend speech. This scale defines a person with profound hearing impairment as one who "cannot understand even amplified speech." And yet there are no comprehensive

studies of long latency AEPs from hearing-impaired people in response to speech stimuli. This appears to be a major gap in the research of central auditory processing.

There are numerous tests of speech comprehension commonly used during audiologic exams. These include the California Consonant Test (CCT), the Nonsense Syllable Test (NST), Speech Perception in Noise (SPIN), the Minimal Auditory Capabilities (MAC) Battery, the Staggered Spondaic Word Test (SSW), the Speech Sounds Perception Test (SSPT) and many more. Of course, these tests can only be used with cooperative and responsive patients. Hall et al. (1983) attempted to correlate ABR and MLR recordings with results from the Staggered Spondaic Word Test in evaluations of patients with severe head injuries. Using long latency AEP responses to speech would have provided 1) information about cortical processing in these patients; 2) a more direct correlation between speech stimuli and neural activity. This is one example of the clinical utility of long latency AEPs. The clinical potential of these procedures can be realized only if the responses from cooperative hearing-impaired individuals are known.

Neville et al. (1983) recorded visual evoked potentials (VEPs) from congenitally deaf and from normal-hearing adults. They reported larger amplitude N150 and P230 VEP components recorded from deaf subjects compared to the responses from subjects with normal hearing. Neville et al. concluded that "early auditory deprivation alters the intermodal organization of the brain's sensory systems in man." Their results suggested that, in deaf people, some brain areas normally designated as auditory cortex are assigned to process visual input. Investigations involving deafened cats

(Rebillard et al., 1977) and ferrets (Sur et al., 1988) have also demonstrated reassignment of auditory areas of the brain to visual processing. These studies provide evidence of reorganization of auditory cortex in response to reduced auditory input.

Additional evidence comes from a study conducted at Gallaudet University by Wolff & Thatcher (1990). They mapped the electroencephalographic coherence, phase, and power of 79 deaf and 92 normal-hearing children. They reported that the deaf children "manifested higher coherence and lower phase in certain left hemispheric areas, suggesting less neural differentiation; but lower coherence and higher phase in certain right hemispheric areas, suggesting greater differentiation." Wolff & Thatcher concluded that "deafness results in a partial reorganization of the cerebral cortex."

Courchesne (1990) asked "To what extent do individual differences in experience (and presumably in synapse selection) translate into individual differences in the chronology of [EP] component development and in the final developmental expression (scalp topography, morphology, latency, amplitude) of components? There is vanishingly little [EP] evidence that speaks to these important questions." (p. 237)

In order to address some of these questions, the present study investigated the effects of auditory experience on AEPs recorded from hearing-impaired and normal-hearing humans. The central hypotheses of this research are:

Differences in experience with sound between normal-hearing and hearing-impaired humans produce differences in the organization of central auditory pathways. This causes delays in temporal processing of complex auditory

stimuli within the brains of people with hearing impairment. These delays are reflected in increased AEP component latencies. Differences in AEP topography to similar stimuli reflect differences in the organization of central auditory pathways between normal and hearing-impaired populations.

AEP experiments were designed to investigate the following:

1) Where in central auditory pathways do differences in organization occur between normal and hearing-impaired populations? That is, latencies of which AEP components are delayed in responses from hearing-impaired subjects?

2) Which types of stimuli elicit delays in AEP component latencies?

3) Can hearing-impaired responses be simulated in normal subjects?

4) Are there differences in exogenous AEP components, endogenous, or both?

5) Are there significant differences in responses to speech stimuli?

6) Are there differences in hearing-impaired vs. normal topography of AEP components in response to the same stimuli? Which types of stimuli elicit topographical differences and which do not?

AIMS OF THIS STUDY

1) To describe and compare auditory evoked potentials recorded from normal-hearing and hearing-impaired humans in response to stimuli of various durations. Responses to stimuli ranging in duration from 50 to 500 msec were recorded from subjects with normal hearing as well as from those with profound, bilateral sensorineural hearing impairment.

2) To record responses to speech stimuli from each subject. Previous studies have

not recorded AEP responses to speech from hearing-impaired subjects. The inability to comprehend speech is the primary trait which characterizes a person as "deaf." Most profoundly hearing-impaired humans can hear low frequency sounds (125 or 250 Hz). However, spoken words cannot be understood because the higher frequency components of speech sounds are not perceived.

3) To compare the topography of AEP responses from hearing-impaired and normal-hearing humans.

Pilot investigations were conducted involving six subjects with normal hearing and six with profound, bilateral, high-frequency sensorineural hearing impairment. Results from those initial investigations were used to design experiments and to generate the central hypotheses to be tested in the present study.



METHODS

The following subject populations participated in the experiments:

1) 8 right-handed subjects (3 male; 5 female) with normal hearing (10 dB HL or better sensitivity for pure tone octave frequencies from 125 to 8000 Hz).

2) 8 right-handed subjects (3 male; 5 female) with congenital profound bilateral high-frequency sensorineural hearing impairment (90-110 dB HL average threshold for 500, 1000, and 2000 Hz tones). All hearing-impaired subjects were able to communicate using American Sign Language (ASL) and lipreading.

Table 2 on the following page contains the ages of each subject. Also included on the following page are the absolute audiometric thresholds of each hearing-impaired subject.

Procedure/AEP Recording

Subjects sat in a comfortable reclining chair within a sound-attenuated booth. Hearing thresholds were established by pure tone audiometry.

Auditory stimuli were delivered binaurally through two Sony Walkman speakers, one positioned 20-25 cm lateral to each of the subject's ears. Pure tones, noise, and speech stimuli were recorded and digitized on one channel of the MacRecorder program using a Macintosh II computer. Speech stimuli consisted of components of the word "POT" (such as "PA," "AH," and "POT") pronounced by a male speaker. A synch pulse (5 msec duration square wave) was generated on the other recording channel precisely 40 msec before the onset of each stimulus. Synch pulses triggered the beginning of recording epochs. Stimuli and corresponding synch pulses were recorded

TABLE 2a. Normal-hearing Subjects:

SUBJECT	SEX	AGE
62	M	20
63	F	27
64	F	21
66	M	22
67	M	35
68	F	31
69	F	36
85	F	33

$\bar{x} = 28.1$

$\sigma = 6.5$

TABLE 2b. Hearing-impaired Subjects:

SUBJECT	SEX	AGE
80	F	31
82	F	37
84	F	23
86	F	44
87	F	32
88	M	28
89	M	30
95	M	35

$\bar{x} = 32.5$

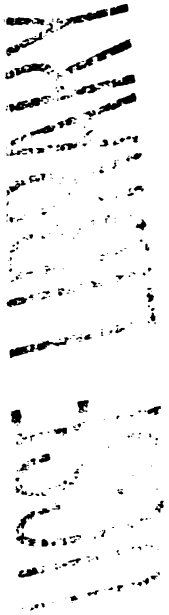
$\sigma = 6.3$

TABLE 2c. Absolute Audiometric Thresholds (dB HL) of Hearing-impaired Subjects

	FREQUENCY (Hz)						
SUBJECT	125	250	500	1000	2000	4000	8000
80	70	80	95	105	110	-	-
82	55	70	90	105	-	-	-
84	60	75	95	100	105	110	-
86	70	90	100	110	-	-	-
87	80	95	110	-	-	-	-
88	65	80	90	100	-	-	-
89	65	70	90	105	105	110	-
95	80	90	95	105	-	-	-

onto magnetic audio tape. Acoustic stimuli were played on a Panasonic 608D cassette deck, amplified through one channel of a Realistic SA-1001 stereo amplifier, and delivered to subjects through the Sony speakers. The other recording channel (which contained synch pulses only) was connected to the data acquisition computer.

Long latency AEP responses were recorded from fifteen scalp electrode positions (Fpz, F3, F4, C3, C4, P3, P4, T3, T4, T5, T6, Fz, Cz, Pz, and Oz according to the International 10-20 system) referenced to linked earlobe electrodes. All electrode impedances were below 5000 ohms. Fifteen scalp electrodes were used for three reasons: 1) to detect any laterality of responses; 2) to allow future analyses of neural generators using dipole source localization techniques (Turetsky et al., 1990); 3) to provide data which was used in mapping the topography of AEP responses.



Single trial data were collected by a Grass Model 12 Neurodata Acquisition System. EEG activity was amplified 50,000 times. EOG activity was amplified 20,000 times. High pass filters within the amplifiers were set at 0.1 Hz; low pass filter settings were 100 Hz; 6 dB/octave. Evoked signals were digitized by a DT2821 Data Translation card within an IBM-compatible computer. 30 to 40 responses to each stimulus were averaged, digitally low-pass filtered at 30 Hz (5 dB/octave) and plotted using the Erpsystem program developed by Neurobehavioral Laboratory Software. Recording epoch durations were either 800 or 855 msec with sampling rates of either 500 or 556 Hz. Recording epochs began 40 msec prior to stimulus onset. Interstimulus intervals varied between 1.5 to 2.0 sec within each protocol. Single trial EPs accompanied by

eye movements greater than 60 μV in peak to peak amplitude were not included in the averages.

Auditory evoked responses were recorded from each subject according to the following experimental protocols:

	NORMAL STIMULI DURATION & INTENSITY	HEARING-IMPAIRED STIMULI DURATION & INTENSITY
PROTOCOL 1	A random presentation of 30-40 each: 500 msec 250 Hz tones 500 msec words ("POT") 74 dB SPL	Same tape used for normal-hearing subjects: 500 msec 250 Hz tones 500 msec words ("POT") 114 dB SPL
PROTOCOL 2	Same tape as in Protocol 1 34 dB SPL	Not presented to hearing-impaired subjects
PROTOCOL 3	Same tape as in Protocol 1 74 dB stimuli with masking noise (increased audiometric thresholds 40 dB)	Not presented to hearing-impaired subjects
PROTOCOL 4	A random presentation of 30-40 each (at 74 dB SPL):	Same stimuli at 114 dB SPL
Condition 1:	50 msec 250 Hz tone burst	
Condition 2:	100 msec 250 Hz tone burst	
Condition 3:	500 msec 250 Hz tone burst	
Condition 4:	50 msec pause between noise bursts	
Condition 5:	100 msec pause between noise bursts	
Condition 6:	silence (an indicator of background EEG activity)	
Condition 7:	250 msec vowel ("AH")	
Condition 8:	250 msec consonant-vowel ("PA")	
Condition 9:	500 msec word ("POT")	

	NORMAL STIMULI DURATION & INTENSITY	HEARING-IMPAIRED STIMULI DURATION & INTENSITY
PROTOCOL 5	A random presentation of the following 74 dB SPL	Same stimuli at 114 dB SPL

Condition 1: 80 each 100 msec 125 Hz tones
Condition 2: 30 each 100 msec 250 Hz tones ("rare")
Condition 3: 30 each 100 msec 125 Hz tones ("pre-rare")
Condition 4: 30 each 100 msec 125 Hz tones ("post-rare")
(Responses were segregated this way so that the "pre-rare" averages would contain the same number of single trials as the "rare" averages. "Rare" tones constituted 21% of the total tones presented during this protocol in order to elicit the P3 component.)

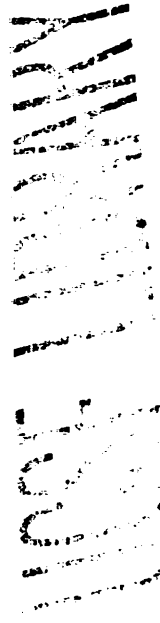
PROTOCOL 6	A random presentation of the following 74 dB SPL	Same stimuli at 114 dB SPL

Condition 1: 80 first names other than the subject's first name (matched with subject's first name for gender, # of syllables, intensity, and duration)
Condition 2: 30 repetitions of the subject's first name

PROTOCOL 7	A random presentation of the following 74 dB SPL	Not presented to hearing-impaired subjects

Condition 1: 80 first names other than the subject's first name (matched with subject's first name for gender, # of syllables, intensity, and duration)
Condition 2: 30 repetitions of a first name other than the subject's first name (matched with subject's first name for gender, # of syllables, duration and intensity; placed in the same positions within the stimulus sequence as the names in Protocol 6, Condition 2)

During Protocols 1 through 3 and 5 through 7, subjects were asked to sit quietly in the sound booth with their eyes open and still as possible. During Protocol 4 subjects were allowed to read something of their choice (a book, magazine, or newspaper). Vertical eye movements (most likely to contaminate AEP recordings) were reduced during these protocols. Even though these were all "passive" paradigms (that is, no response was required to any stimulus), it was possible to assess differences in responses to the same stimuli with variations in subject attention. Subjects were monitored via a video camera to ensure that they did not close their eyes or fall asleep while they were in the booth. The entire procedure (including audiometry, applying the Electro-cap & eye electrodes, and recording data) lasted approximately 3 to 4 hours for each subject.



RESULTS

Single trial AEPs were averaged for each subject in response to each stimulus. AEP component latencies and amplitudes were calculated from these averaged responses by peak-picking computer programs. All of the waveforms presented on the following pages are grand averages which were constructed using ASYST computer programs. Statistical comparisons were made (between grand averaged responses recorded from Cz) by t tests of mean values. Statistical significance is defined as $p < .05$.

All waveforms shown in this section are grand averaged responses of eight normal and eight hearing-impaired subjects.

Topographic maps of long latency grand averaged responses were made using several ASYST computer programs. The Nearest Neighbor method of interpolation was employed in which the four nearest electrodes were used to calculate each interpolated value (Shepard, 1968; Buchsbaum et al., 1982). Each electrode's value was weighted by the inverse of the squared distance between it and the interpolation point. Data were digitally low pass filtered (at 30 Hz) before the maps were generated. Figure 2 shows the electrode positions used during these experiments.

PROTOCOL 1 - A random sequence of 30-40 each: 500 msec tones; 500 msec words

Condition 1: Figure 3 contains the normal-hearing response to at least 30 repetitions of 500 msec duration, 250 Hz tones. Figure 4 is the topographic representation of these data. Note the fronto-central distribution of the N1 component; P2 centered at Cz; and the absence of a visible P3 component (even

though four of these subjects had a small P3).

Figure 5 is the hearing-impaired response to 500 msec duration, 250 Hz tones. Figure 6 is the topographic representation of these data. Note the similarity in N1 and P2 latency and topography between this and the normal map. Also, notice the more obvious frontal P3 component here.

Figure 7 is a comparison between normal-hearing and hearing-impaired responses to tones recorded at Cz. Mean latency and amplitude data are listed on the figure (as they will be for all such figures in this section). The only significant differences between the groups are: greater N1 amplitude in normal responses; greater P3 amplitude in hearing-impaired responses.

Condition 2: Figure 8 contains the normal-hearing response to at least 30 repetitions of the word "POT" each 500 msec in duration. Figure 9 is the topographic representation of these data. Note the differences in the distributions of N1 and P2 components compared to the normal response to tones. N1 is shifted more to the left hemisphere; P2 more to the right. Also, because all eight of the normal subjects had a P3 in response to "POT," this component is more evident in this map (at the parietal electrodes).

Figure 10 contains the hearing-impaired response to "POT." Figure 11 is the topographic representation of these data. Comparing this to the normal map, note the delay in latency and more frontal distribution of the N1 component. Also note the differences in latency and distribution of the P2 and P3 components.

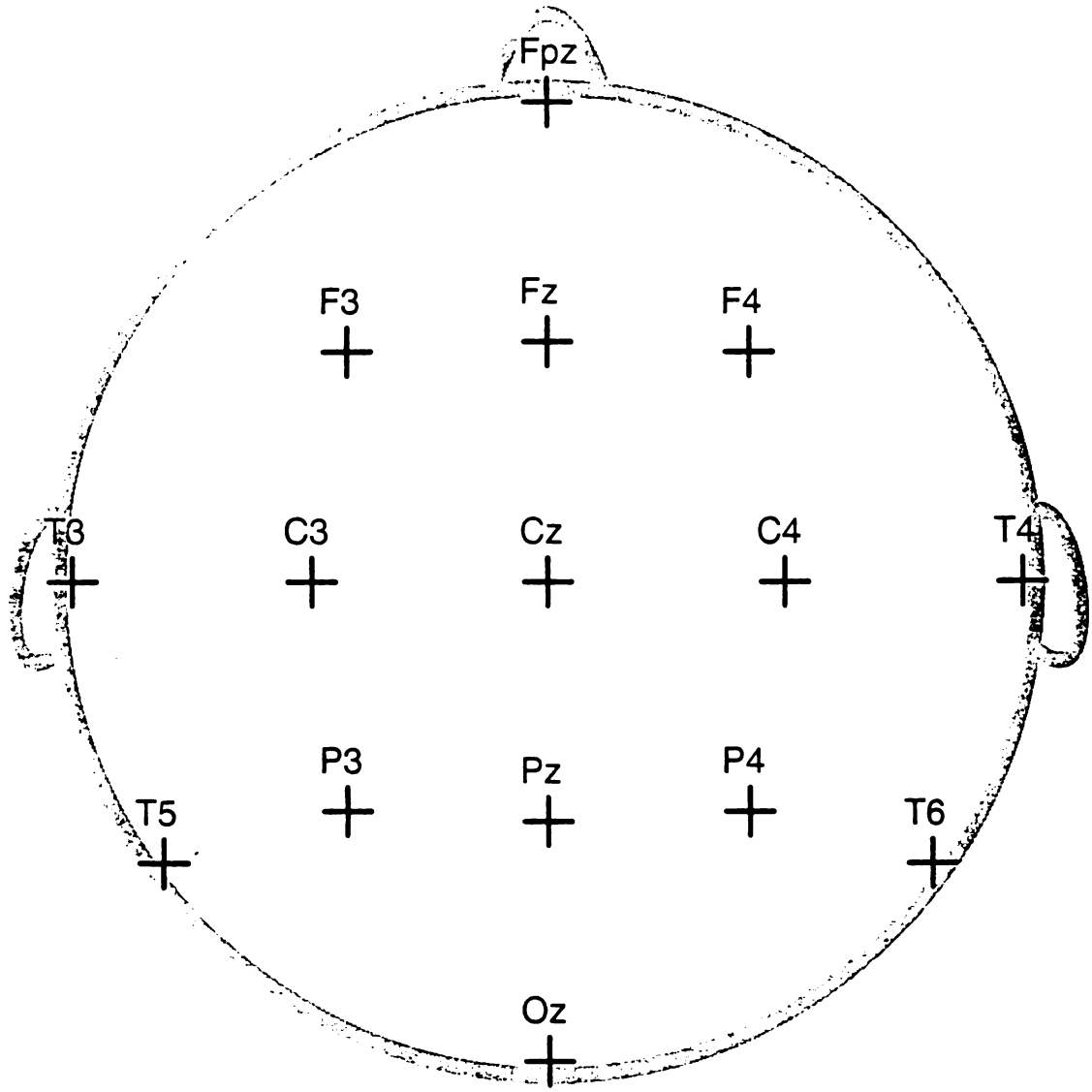


FIGURE 2. Electrode Positions

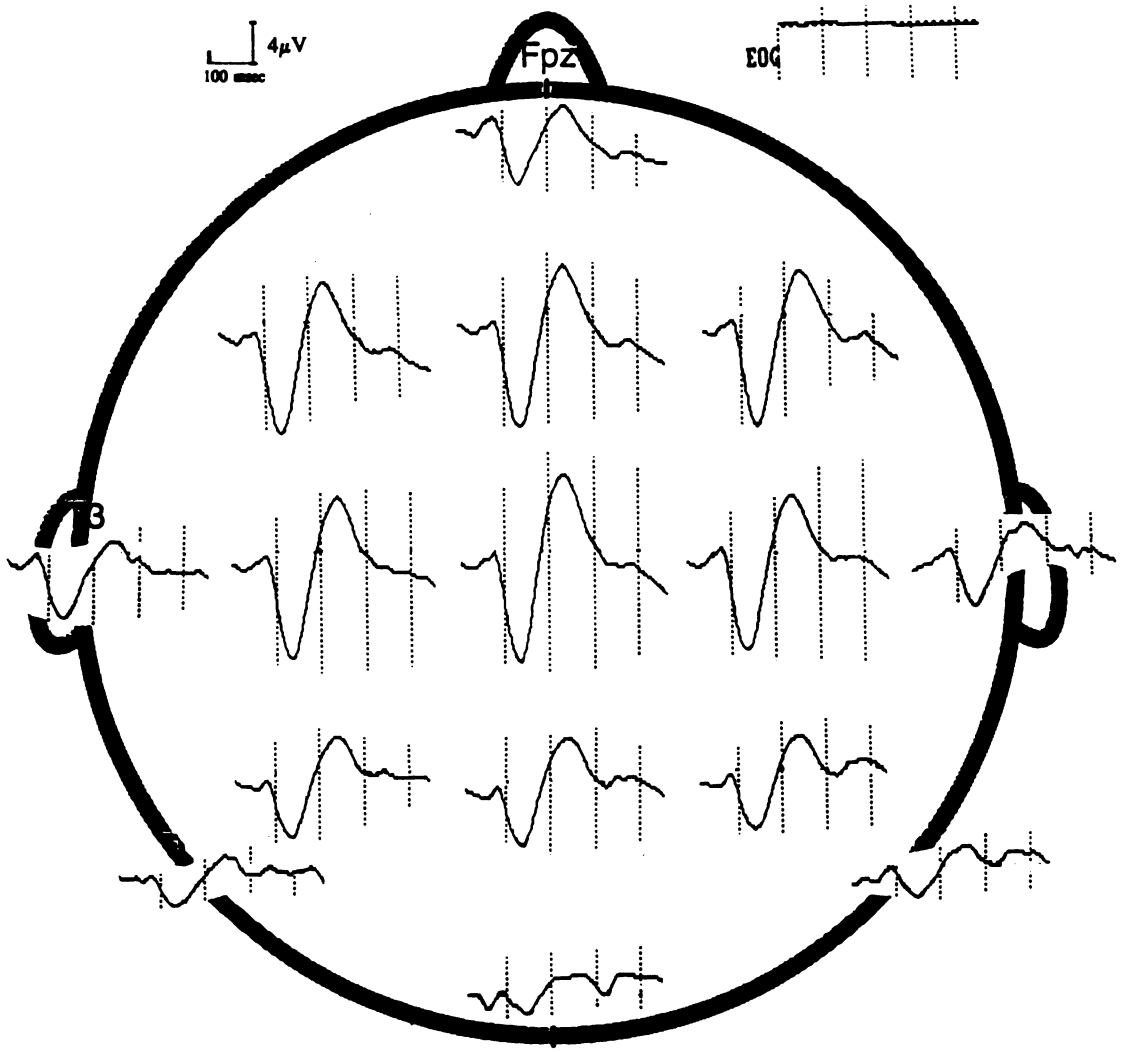
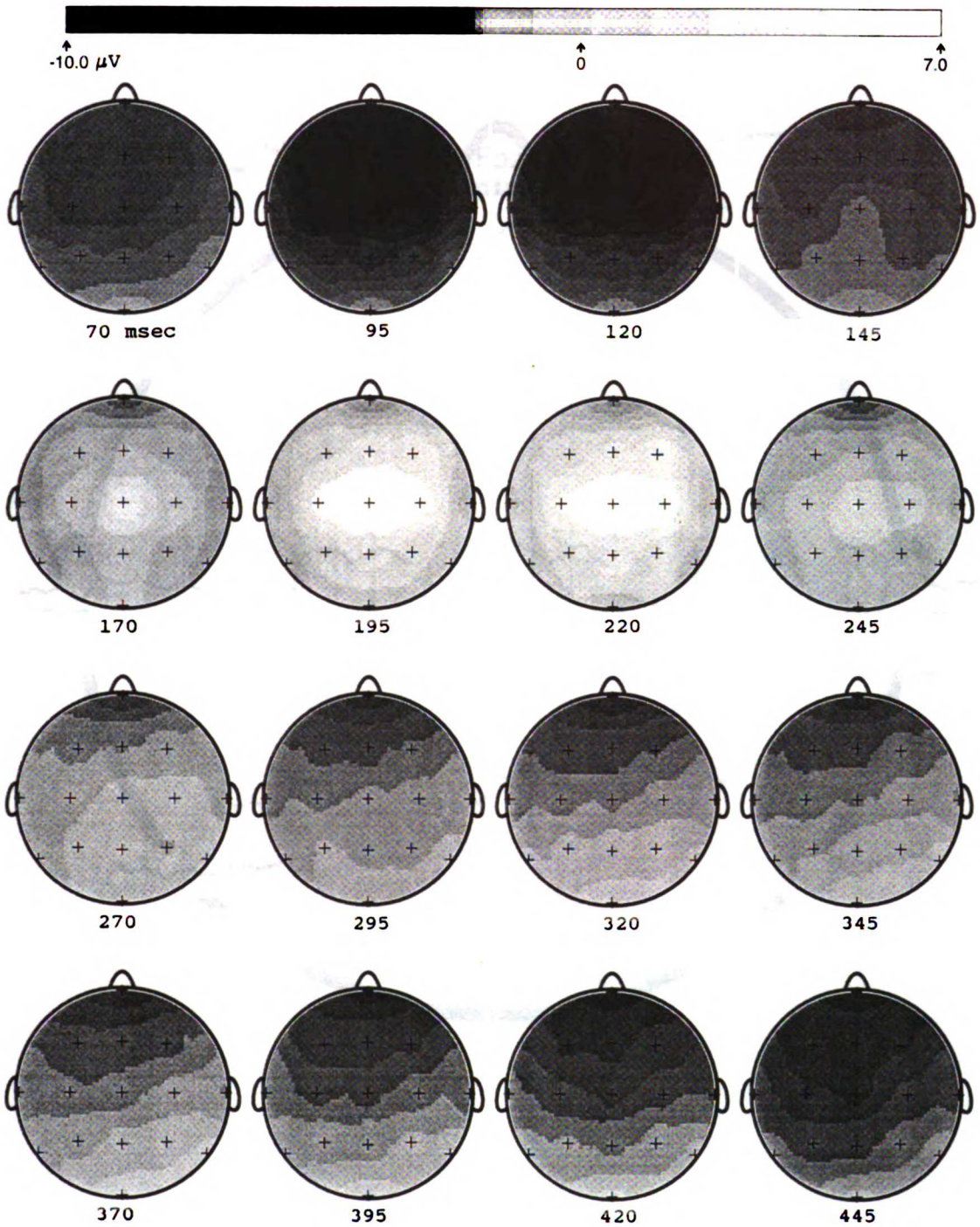


FIGURE 3. Normal Response to Tones (Protocol 1)

FIGURE 4. Normal Response to Tones (Protocol 1)



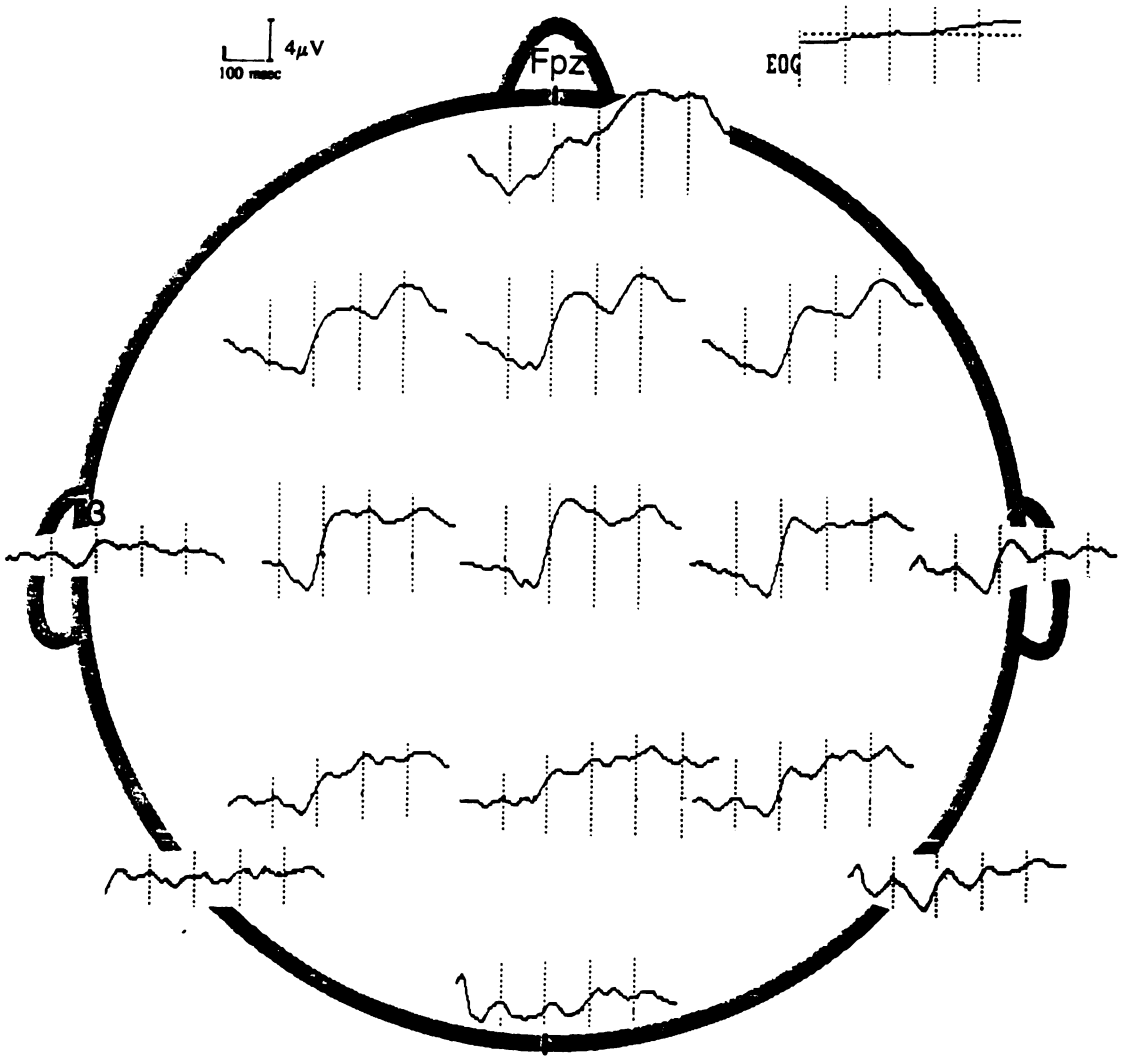
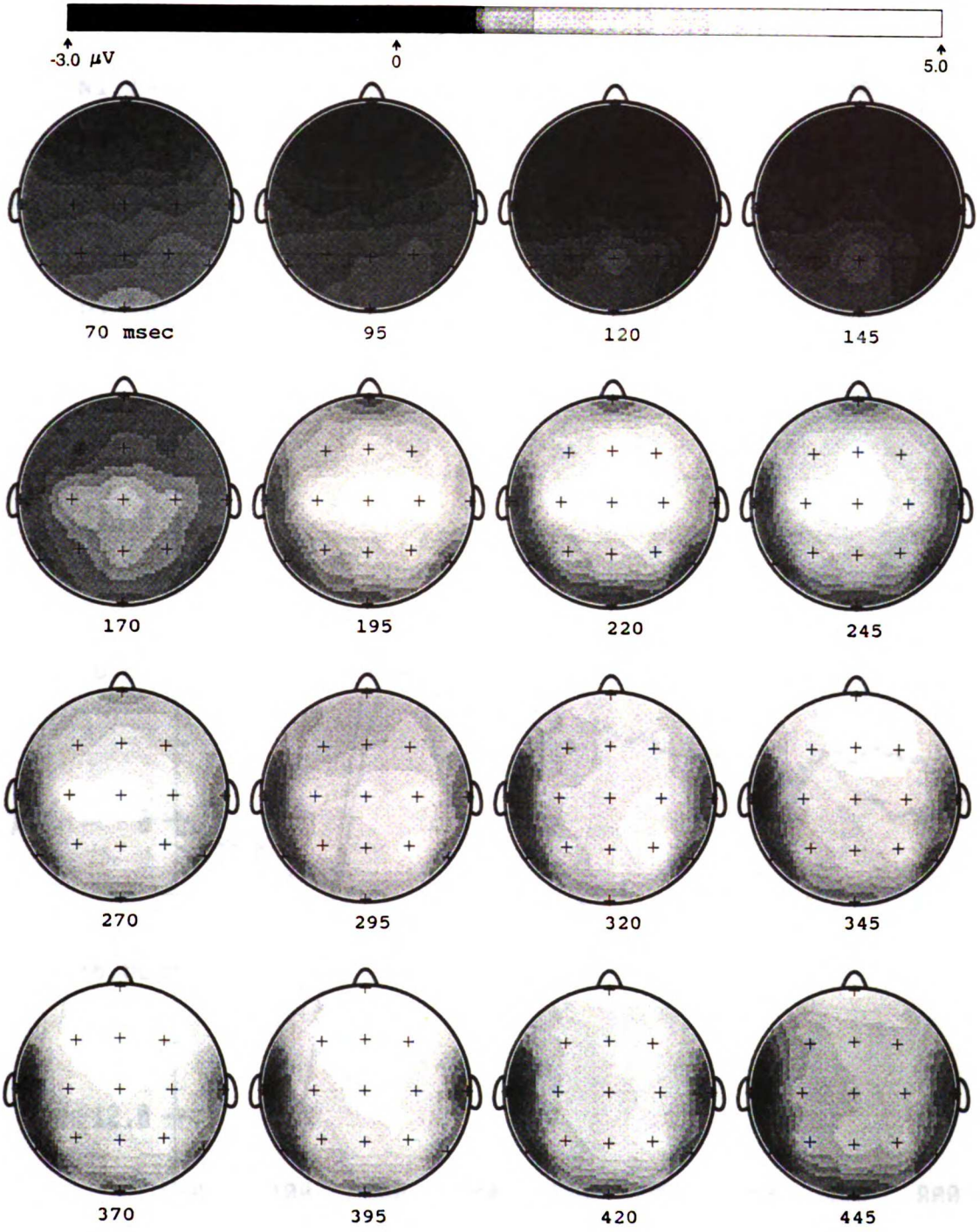


FIGURE 5. Hearing-impaired Response to Tones (Protocol 1)

FIGURE 6. Hearing-impaired Response to Tones (Protocol 1)



	Normal-hearing	Hearing-impaired	p <
N1 lat.	101.8 ± 10.0	119.5 ± 26.7	ns
N1 amp.	-10.1 ± 3.4	-3.8 ± 4.0	.0025
P2 lat.	206.0 ± 21.5	213.5 ± 29.1	ns
P2 amp.	10.4 ± 6.4	8.0 ± 3.3	ns
P3 lat.	360.3 ± 22.8	375.4 ± 25.7	ns
P3 amp.	1.4 ± 2.0	9.3 ± 5.0	.005

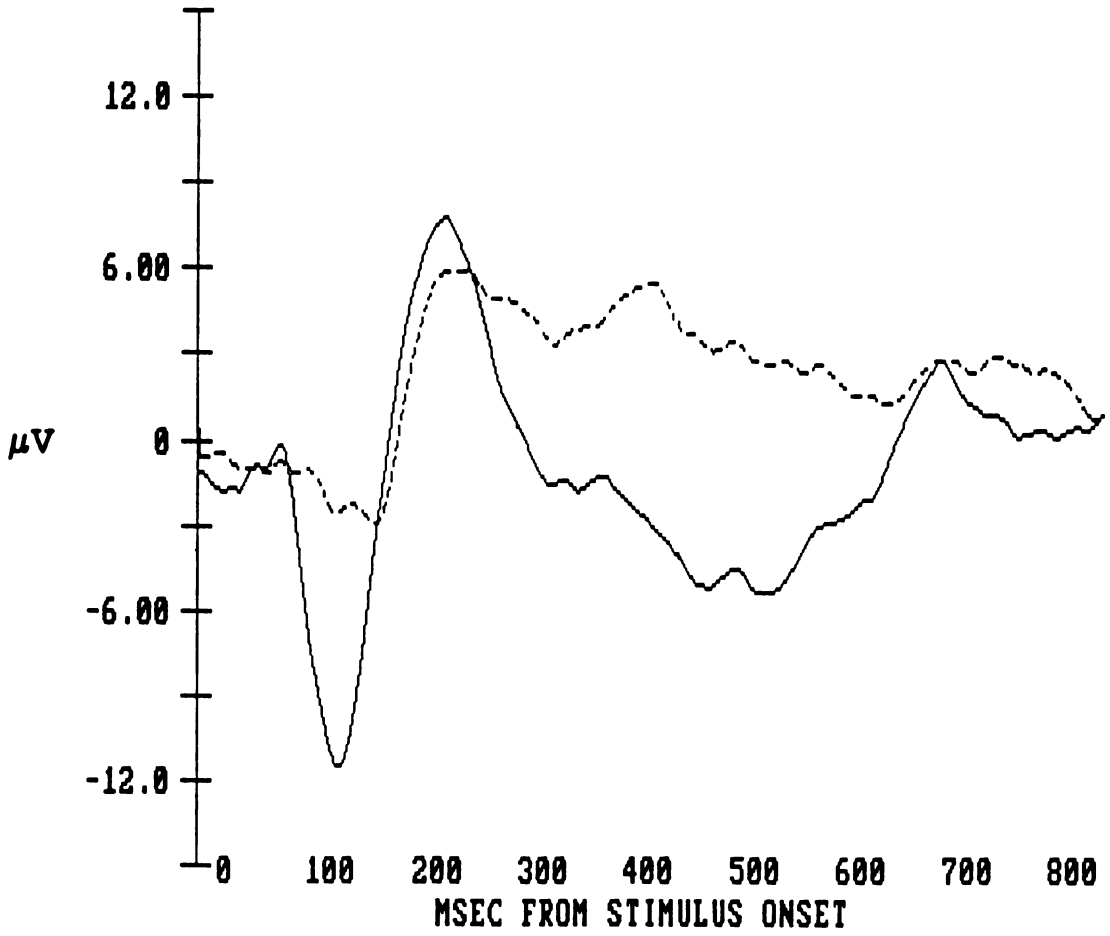


FIGURE 7. Solid = Normal Response to Tones
Dashed = Hearing-impaired Response to Tones

Figure 12 is a comparison between normal-hearing and hearing-impaired responses to "POT" recorded at Cz. Note the significant differences in N1 and P3 latency (greater in hearing-impaired) and N1 amplitude (greater in normals).

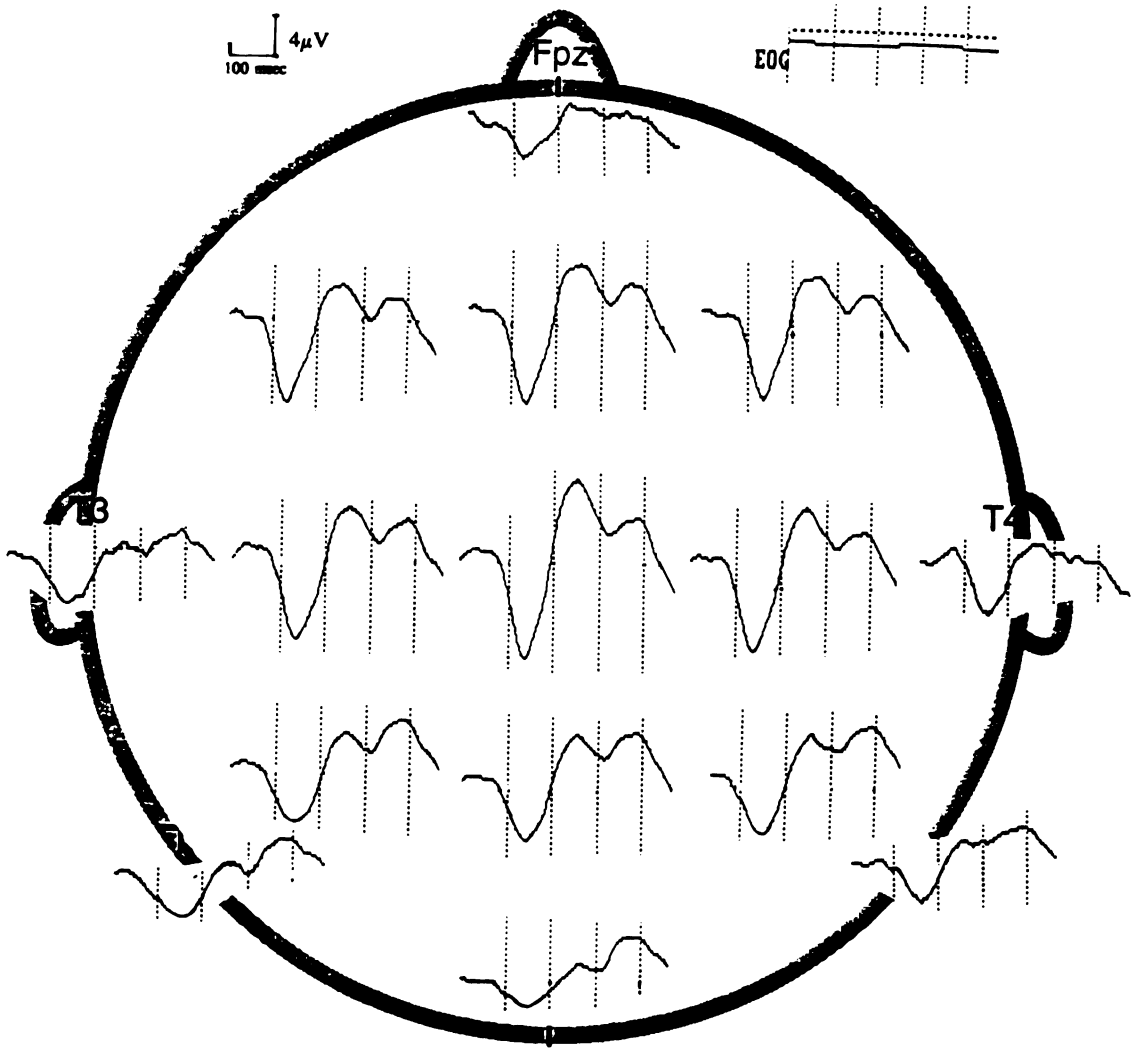
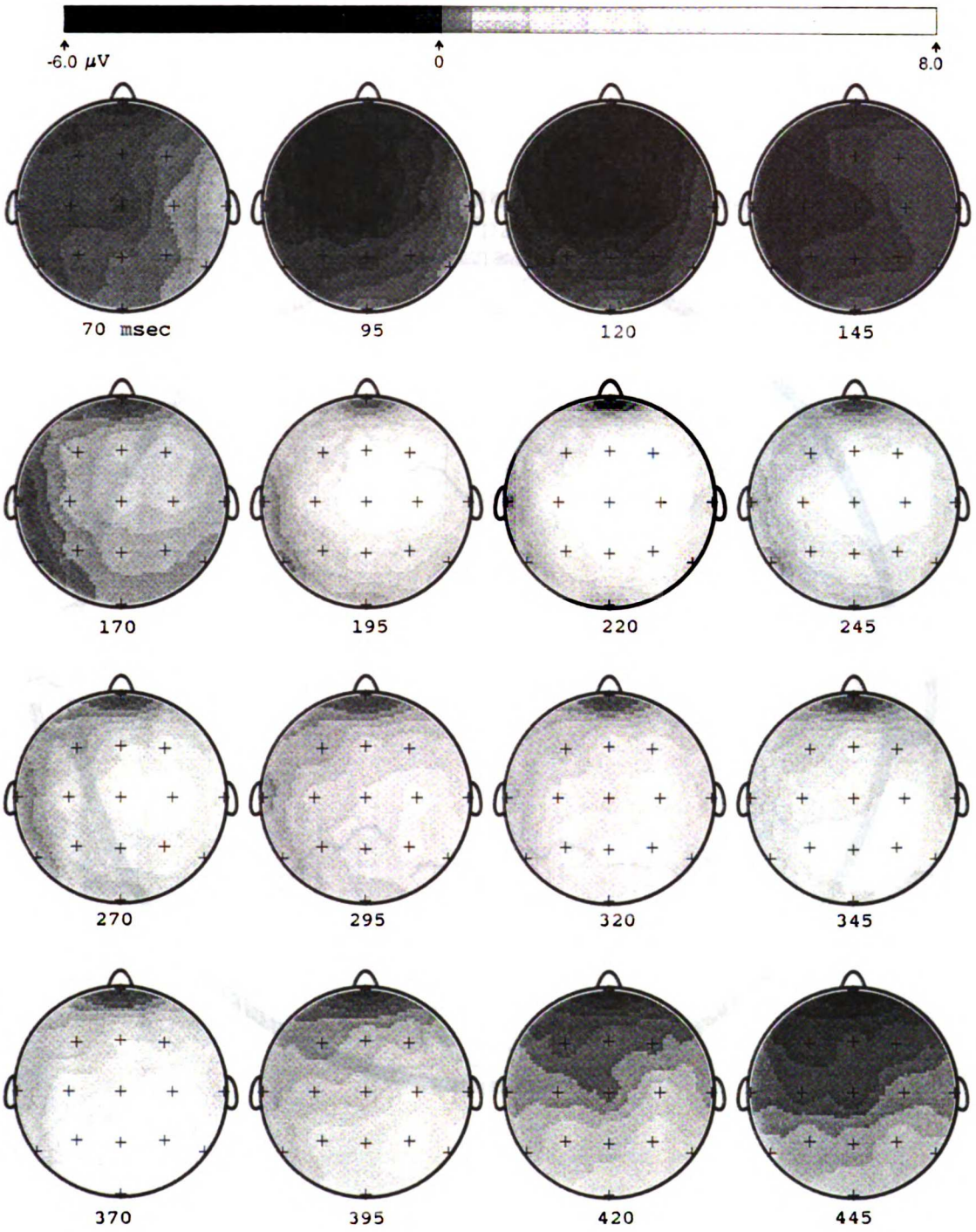


FIGURE 8. Normal Response to "POT" (Protocol 1)

FIGURE 9. Normal Response to "POT" (Protocol 1)



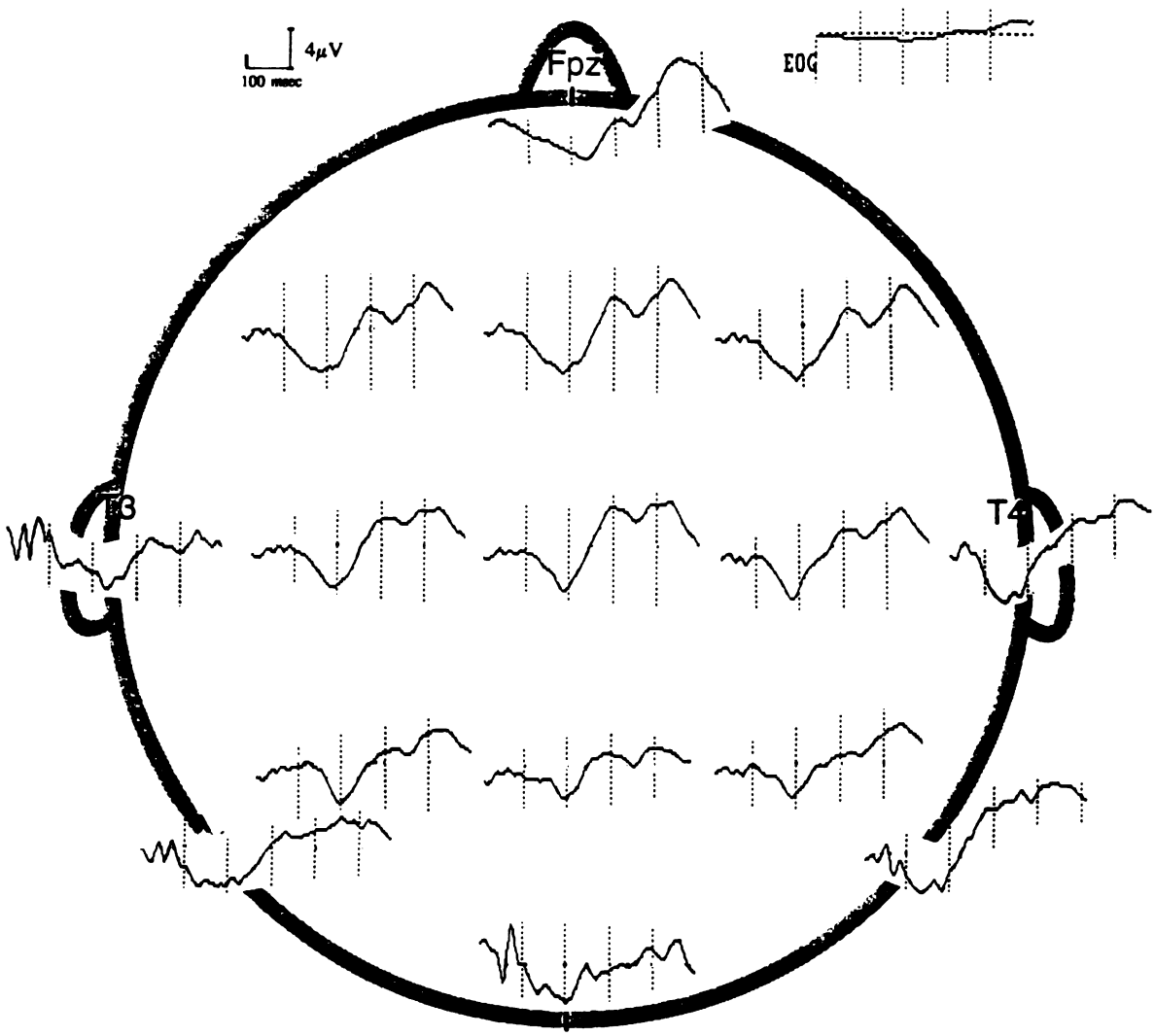
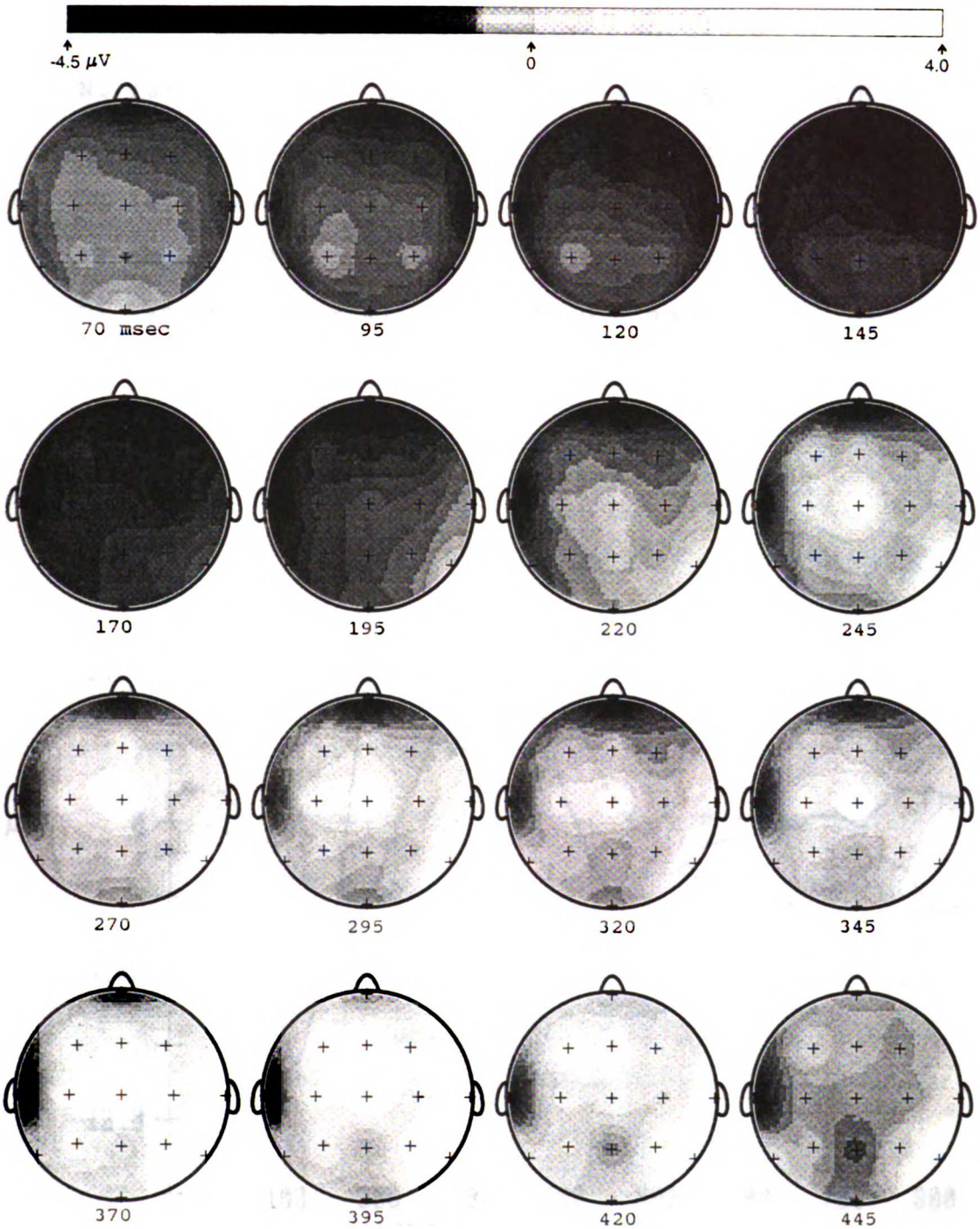


FIGURE 10. Hearing-impaired Response to "POT" (Protocol 1)

FIGURE 11. Hearing-impaired Response to "POT" (Protocol 1)



	Normal-hearing	Hearing-impaired	p <
N1 lat.	106.4 ± 12.0	146.1 ± 26.5	.0025
N1 amp.	-10.7 ± 5.2	-4.8 ± 4.2	.015
P2 lat.	216.3 ± 21.0	236.8 ± 45.1	ns
P2 amp.	9.0 ± 3.7	7.0 ± 6.6	ns
P3 lat.	341.4 ± 41.0	377.0 ± 20.9	.05
P3 amp.	5.5 ± 4.1	6.5 ± 7.3	ns

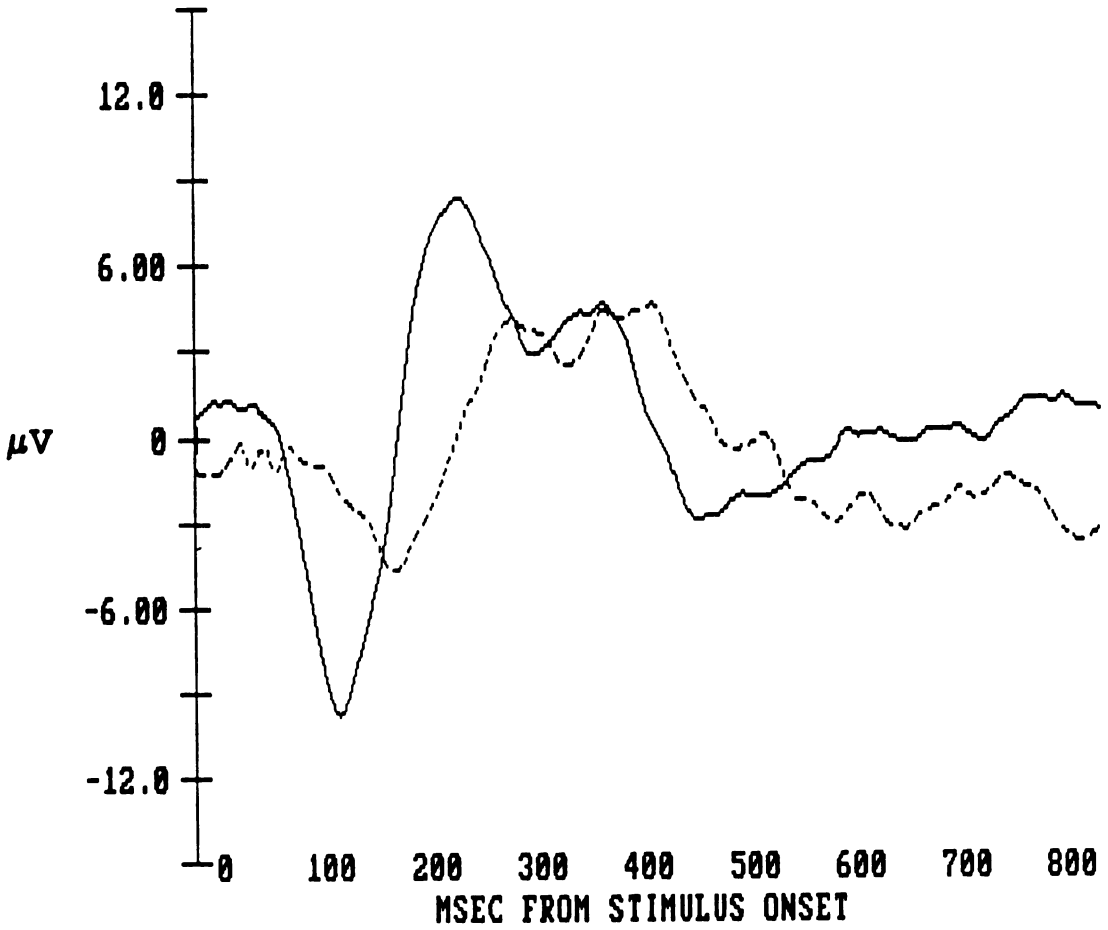


FIGURE 12. Solid = Normal Response to "POT"
Dashed = Hearing-impaired Response to "POT"

PROTOCOL 2 - Normal-hearing Subjects Only: simulated hearing loss

Same stimuli as in Protocol 1; intensity = 34 dB SPL

Condition 1: Figure 13 contains the normal response to tones with a 40 dB reduction of intensity from Protocol 1. Figure 14 is the topographical representation of these data. Compared to normal responses in Protocol 1, the latency of the N1 component increased. P2 remains centered around the Cz electrode. Subjects did not produce P3 components to these stimuli. Compared to normal responses in Protocol 1, Condition 1, N1 and P2 amplitudes are smaller.

Figure 15 compares the normal response to reduced intensity tones in this protocol with the hearing-impaired response to tones in Protocol 1 (recorded from Cz). P2 and P3 amplitudes are significantly greater for the hearing-impaired group. However, N1 amplitude and all latencies are not significantly different.

Condition 2: Figure 16 contains the normal response to reduced intensity "POT." Figure 17 is the topographic representation of these data. Compared to the normal response to "POT" in Protocol 1, the reduced stimulus intensity here resulted in a slight (12.7 msec) but not significant increase in N1 latency and a decrease in N1 amplitude. However, the topography of the N1 component is very similar in both protocols. The reduced amplitude P2 also had a slight increase in latency. The P3 component is no longer evident.

Figure 18 is a comparison of normal responses to 34 dB intensity "POT" (Protocol 2) with hearing-impaired responses to 114 dB "POT" (Protocol 1) recorded

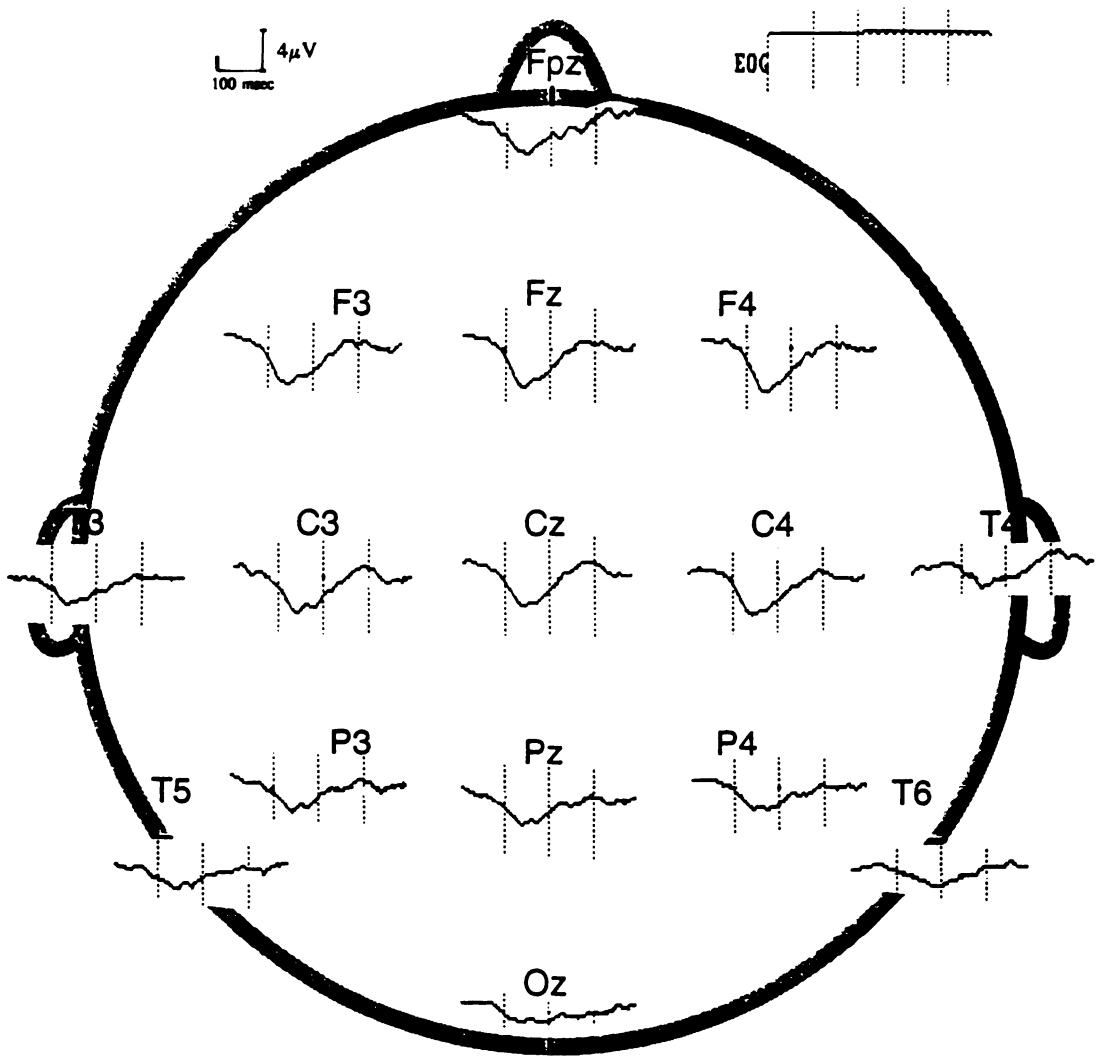
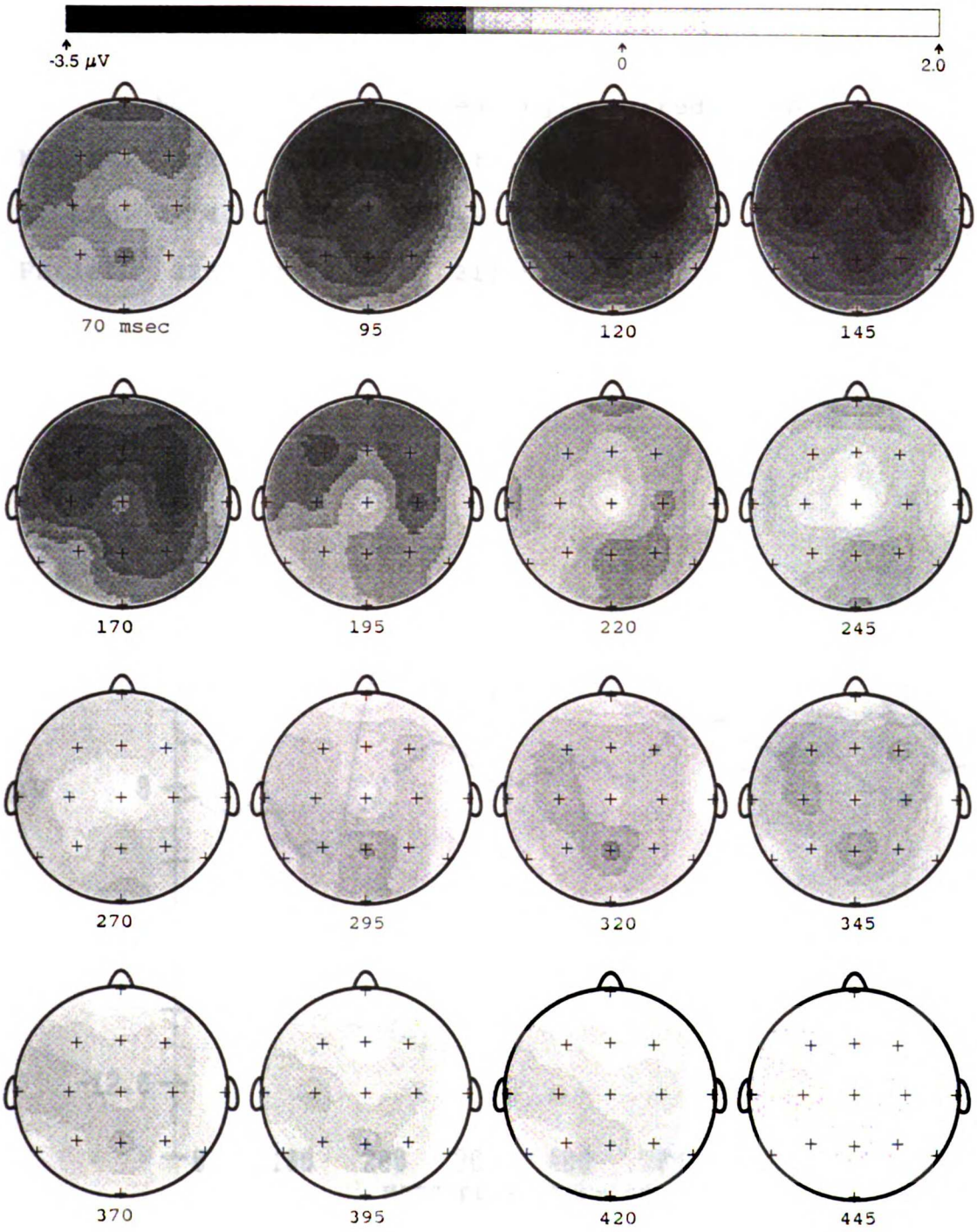


FIGURE 13. Normal Response to Reduced Intensity Tones (Protocol 2)

FIGURE 14. Normal Response to Reduced Intensity Tones (Protocol 2)



	Normal - Tones	Hearing-impaired	p <
N1 lat.	132.8 ± 23.6	119.5 ± 26.7	ns
N1 amp.	- 4.4 ± 2.1	-3.8 ± 4.0	ns
P2 lat.	223.3 ± 41.6	213.5 ± 29.1	ns
P2 amp.	2.5 ± 3.3	8.0 ± 3.3	.0025

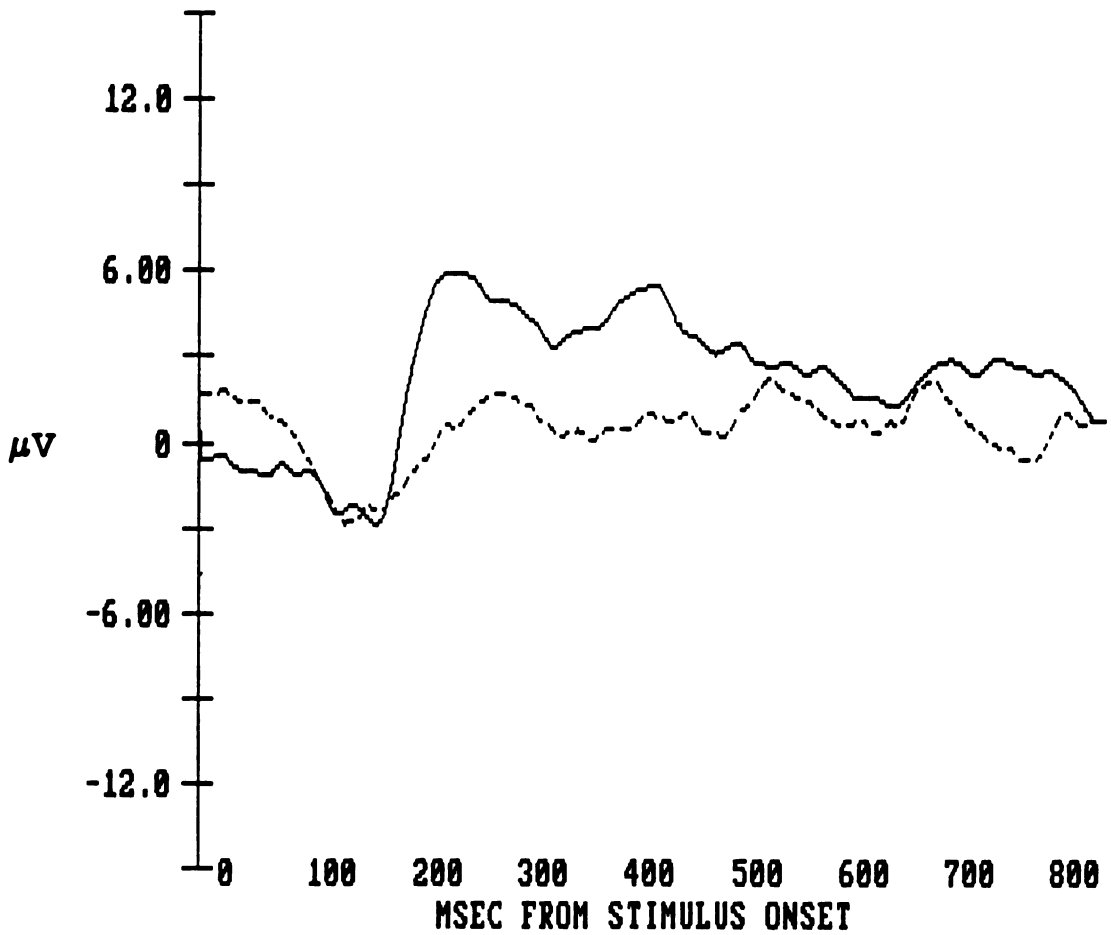


FIGURE 15. Solid = Hearing-impaired Response to Tones (Protocol 1)
Dashed = Normal Response to Tones (Reduced Intensity - Protocol 2)

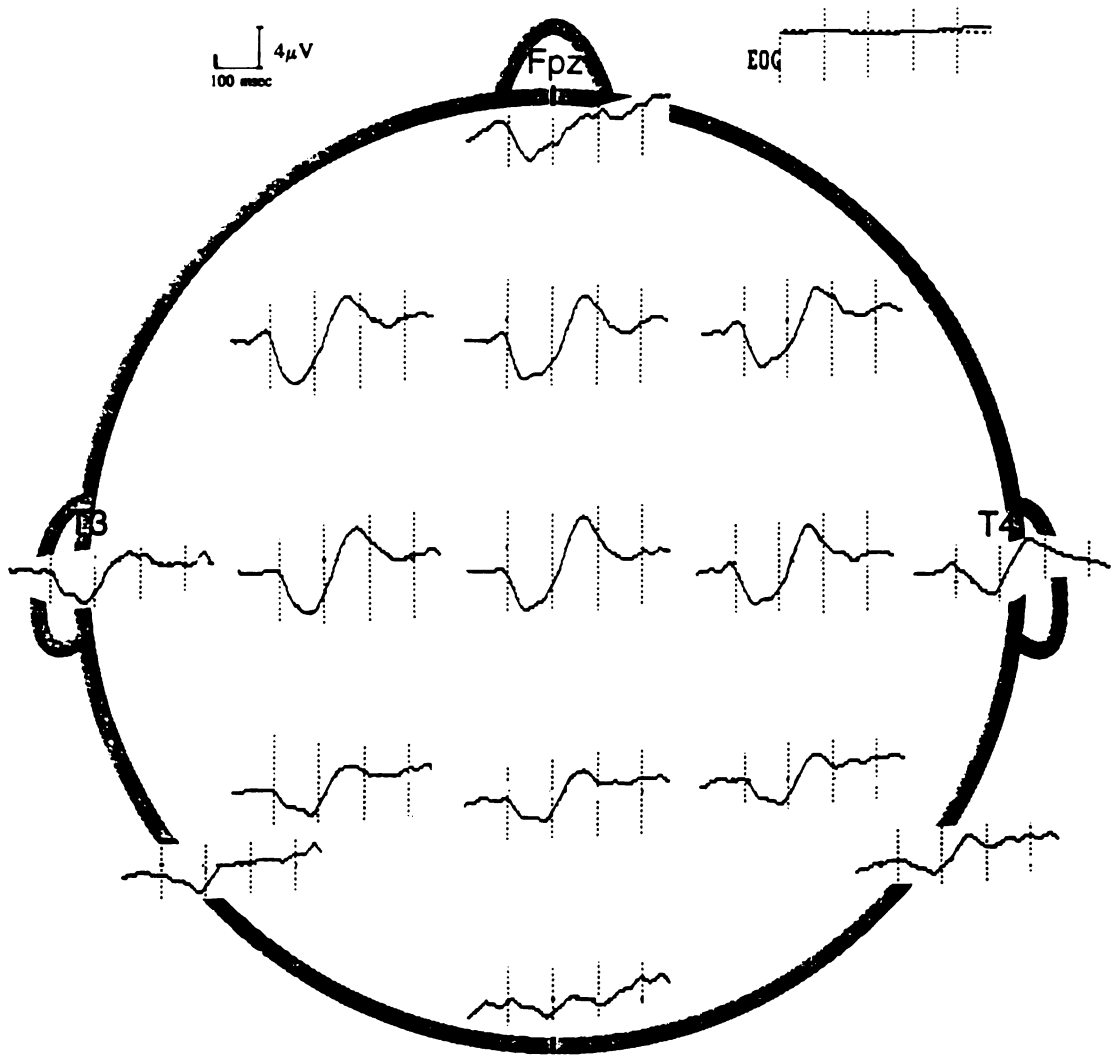
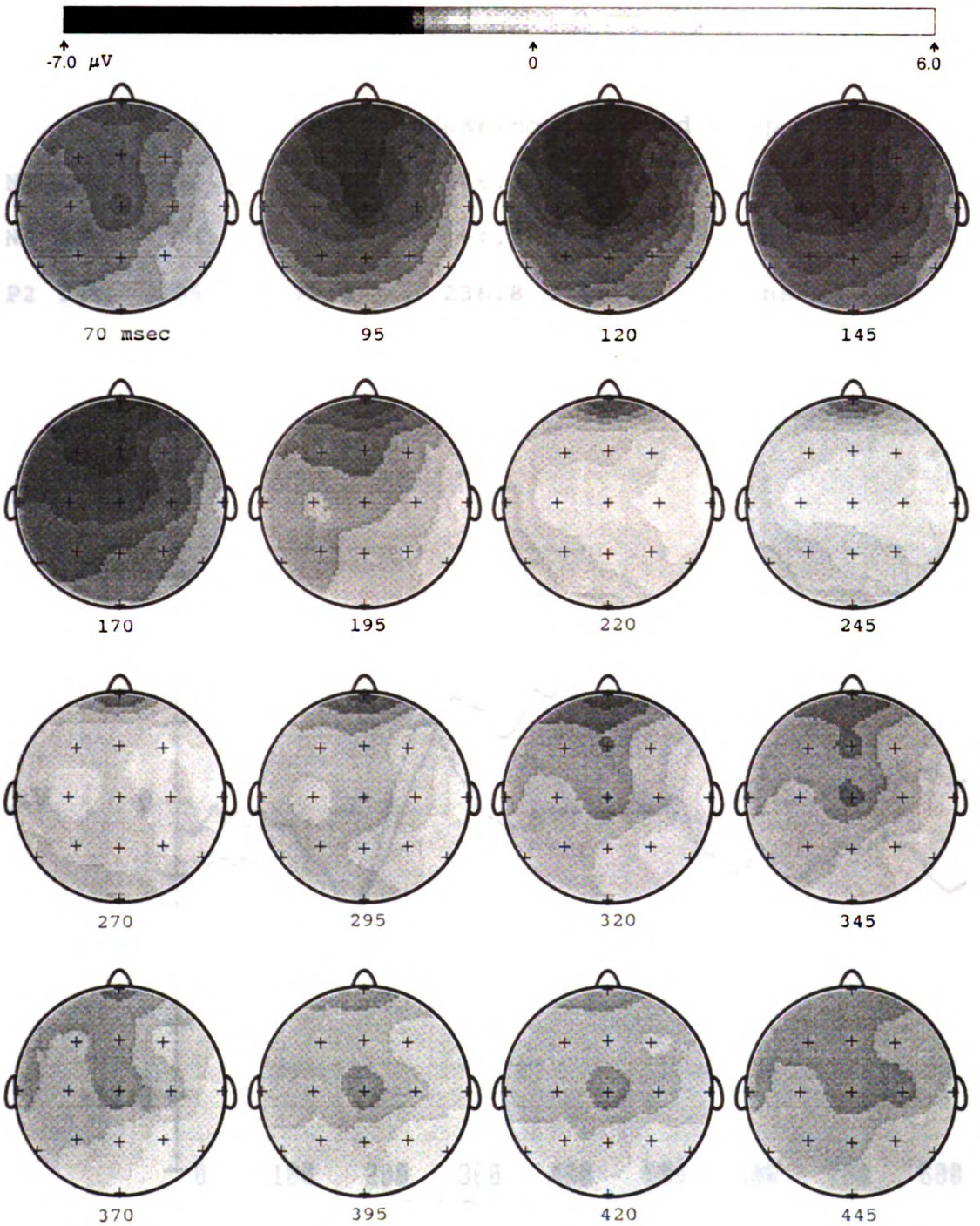


FIGURE 16. Normal Response to Reduced Intensity "POT" (Protocol 2)

FIGURE 17. Normal Response to Reduced Intensity "POT" (Protocol 2)



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	Normal - "POT"	Hearing-impaired	p <
N1 lat.	119.1 ± 18.5	146.1 ± 26.5	.02
N1 amp.	-4.7 ± 3.2	-4.8 ± 4.2	ns
P2 lat.	208.5 ± 39.5	236.8 ± 45.1	ns
P2 amp.	5.6 ± 4.0	7.0 ± 6.6	ns

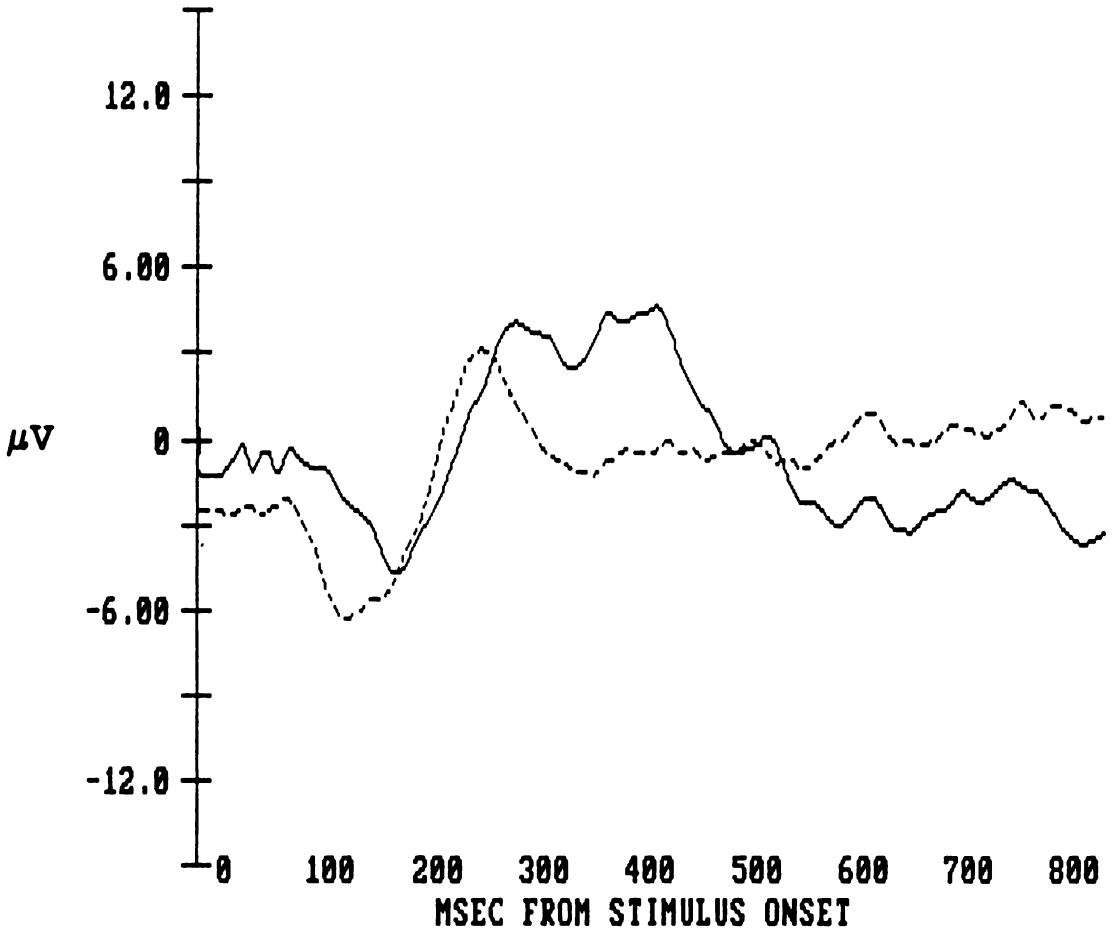


FIGURE 18. Solid = Hearing-impaired Response to "POT" (Protocol 1)
Dashed = Normal Response to "POT" (Reduced Intensity - Protocol 2)

from Cz. Differences in N1 latency remain significant. However, P2 latency, N1 and P2 amplitudes are not significantly different. P3 is present in the hearing-impaired response, not in the normal response.

PROTOCOL 3 - Normal-hearing Subjects Only: simulated hearing loss

Same stimuli as in Protocol 1; with masking noise

Condition 1: Figure 19 contains the normal response to 74 dB tones + masking noise (which raised audiometric thresholds by 40 dB). Figure 20 is the topographic representation of these data. Compared to the normal response to tones in Protocol 1, the N1 latency increased slightly and its amplitude decreased, but its distribution remains frontocentral. P2 is greatly reduced in amplitude, but there is no significant change in its latency. P3 is not evident.

Figure 21 is a comparison between normal responses to tones + masking noise (Protocol 3) and hearing-impaired responses to tones (Protocol 1) recorded from Cz. There are no significant differences in N1 or P2 latency or N1 amplitude. P2 and P3 components have greater amplitude in the hearing-impaired response. The topographic distribution of the N1 component is similar in both groups.

Condition 2: Figure 22 is the normal response to 74 dB "POT" + masking noise. Figure 23 is the topographic representation of these data. Compared to the normal response to "POT" in Protocol 1, N1 and P2 latencies are significantly increased; N1 and P2 amplitudes are decreased. However, the topographical distributions of N1 and P2 here are similar to those in Protocol 1. The P3 component is not evident here.

Figure 24 is a comparison between the normal response to "POT" + masking noise (Protocol 3) and the hearing-impaired response to "POT" (Protocol 1) recorded from Cz. Notice that there are no significant differences in N1 or P2 latency or amplitude. P3 is evident in the hearing-impaired response, not in the normal response.

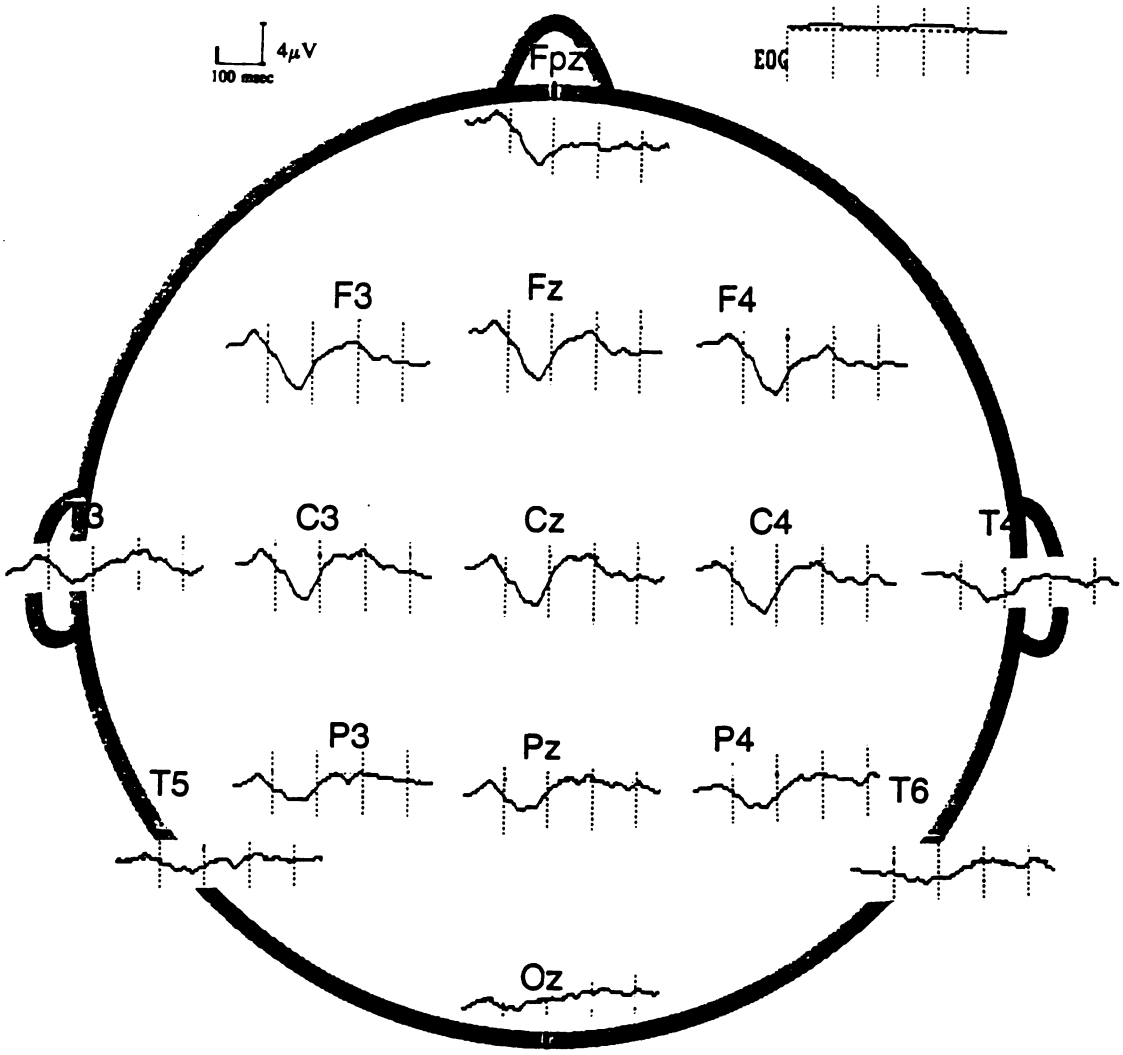
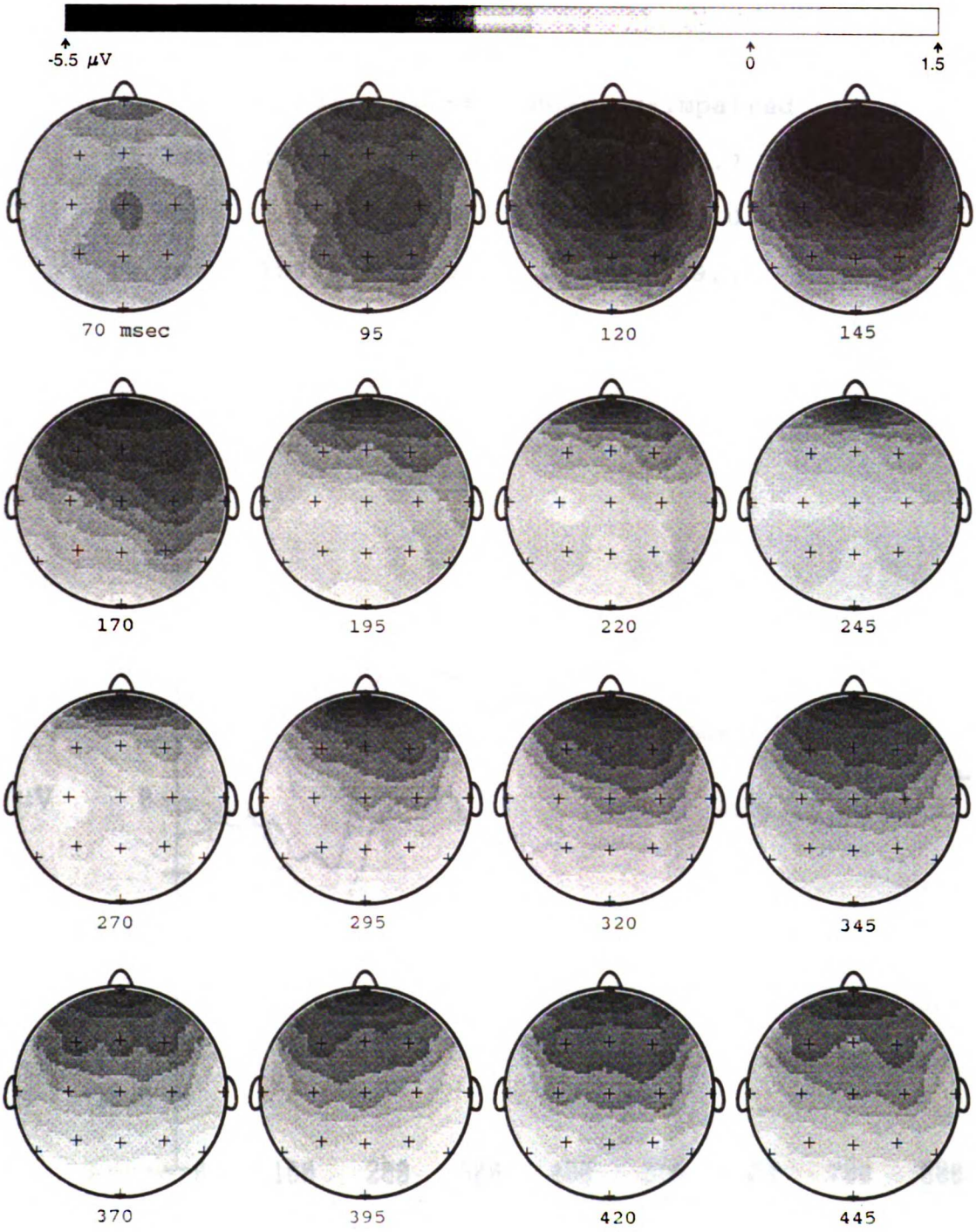


FIGURE 19. Normal Response to Tones + Noise (Protocol 3)

FIGURE 20. Normal Response to Tones + Noise (Protocol 3)



	Normal - Tones	Hearing-impaired	p <
N1 lat.	124.1 ± 16.4	119.5 ± 26.7	ns
N1 amp.	- 5.4 ± 3.8	-3.8 ± 4.0	ns
P2 lat.	189.1 ± 48.5	213.5 ± 29.1	ns
P2 amp.	2.9 ± 2.4	8.0 ± 3.3	.0025

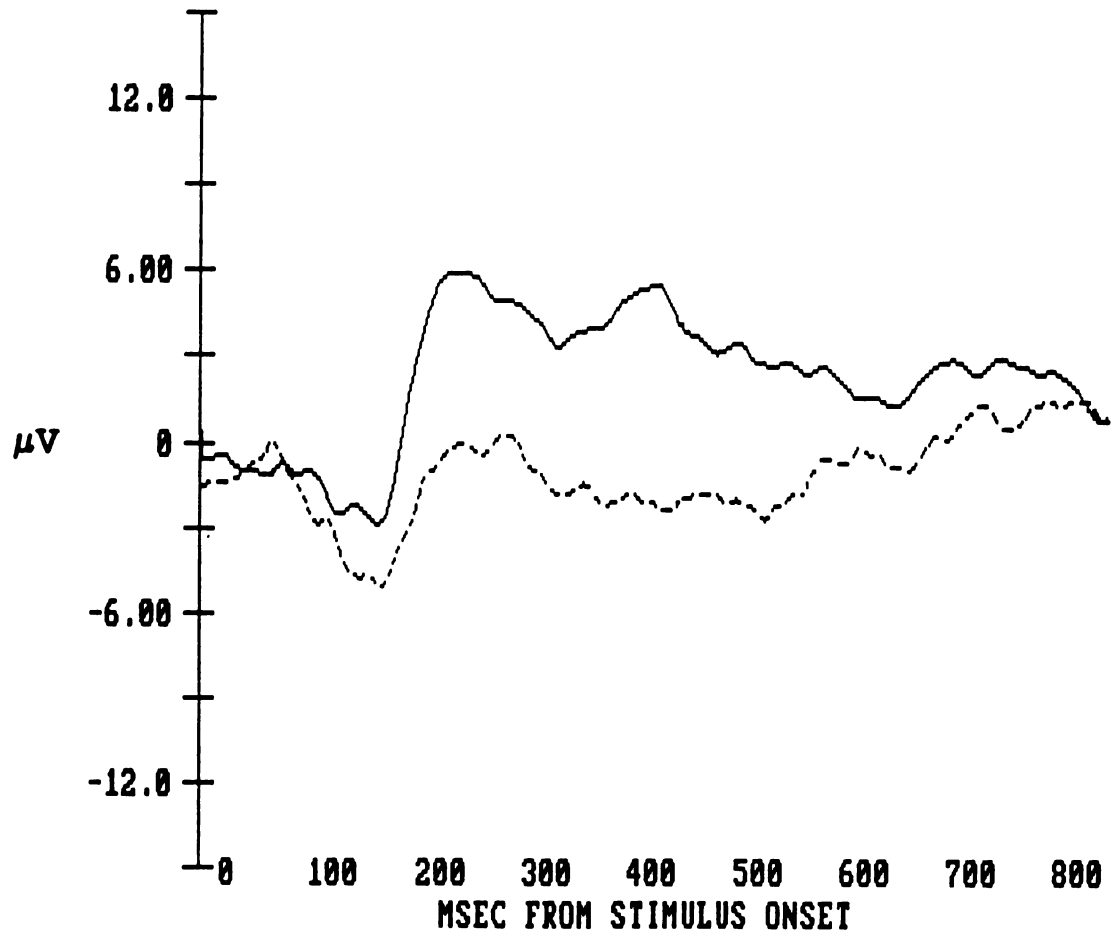


FIGURE 21. Solid = Hearing-impaired Response to Tones (Protocol 1)
Dashed = Normal Response to Tones + Masking Noise (Protocol 3)

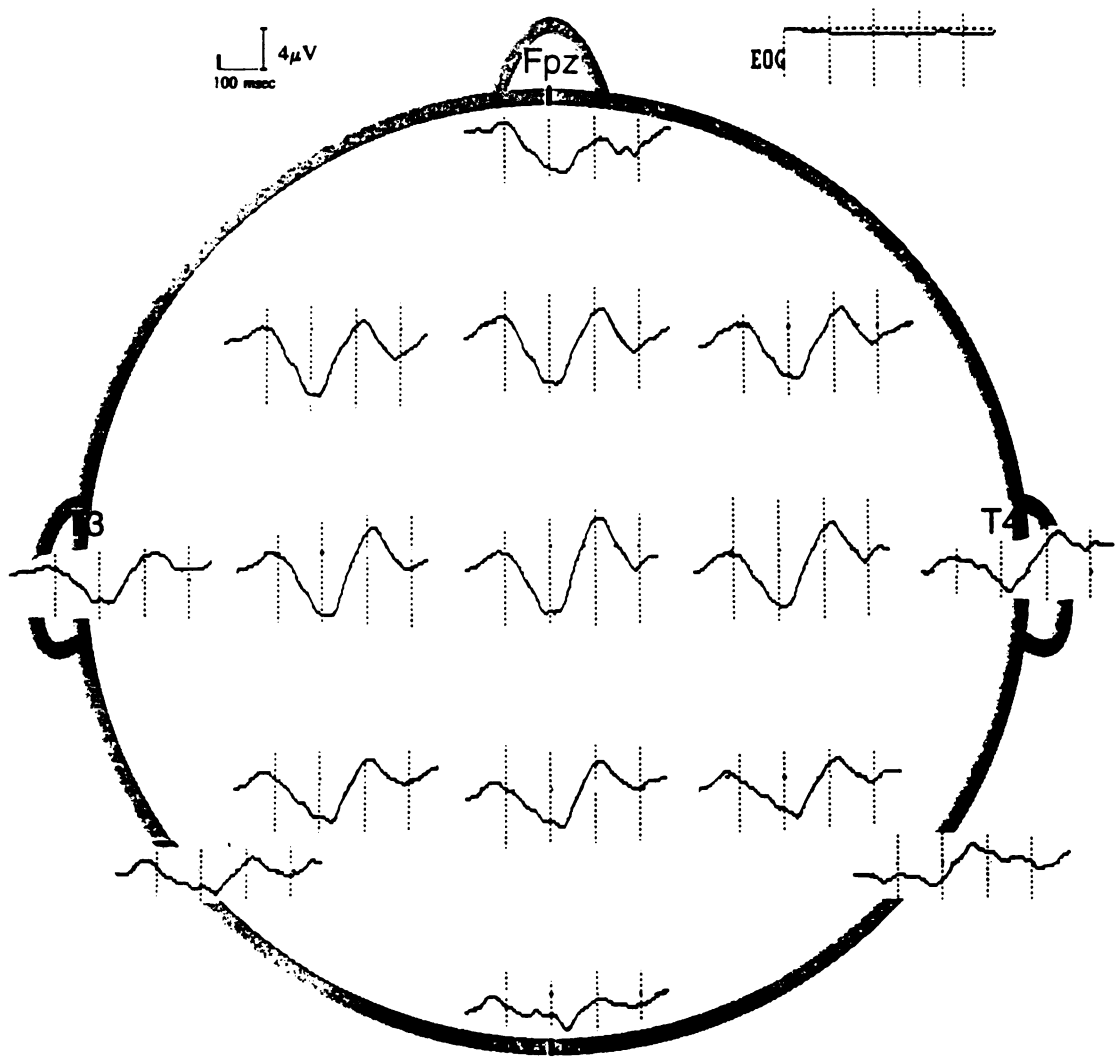
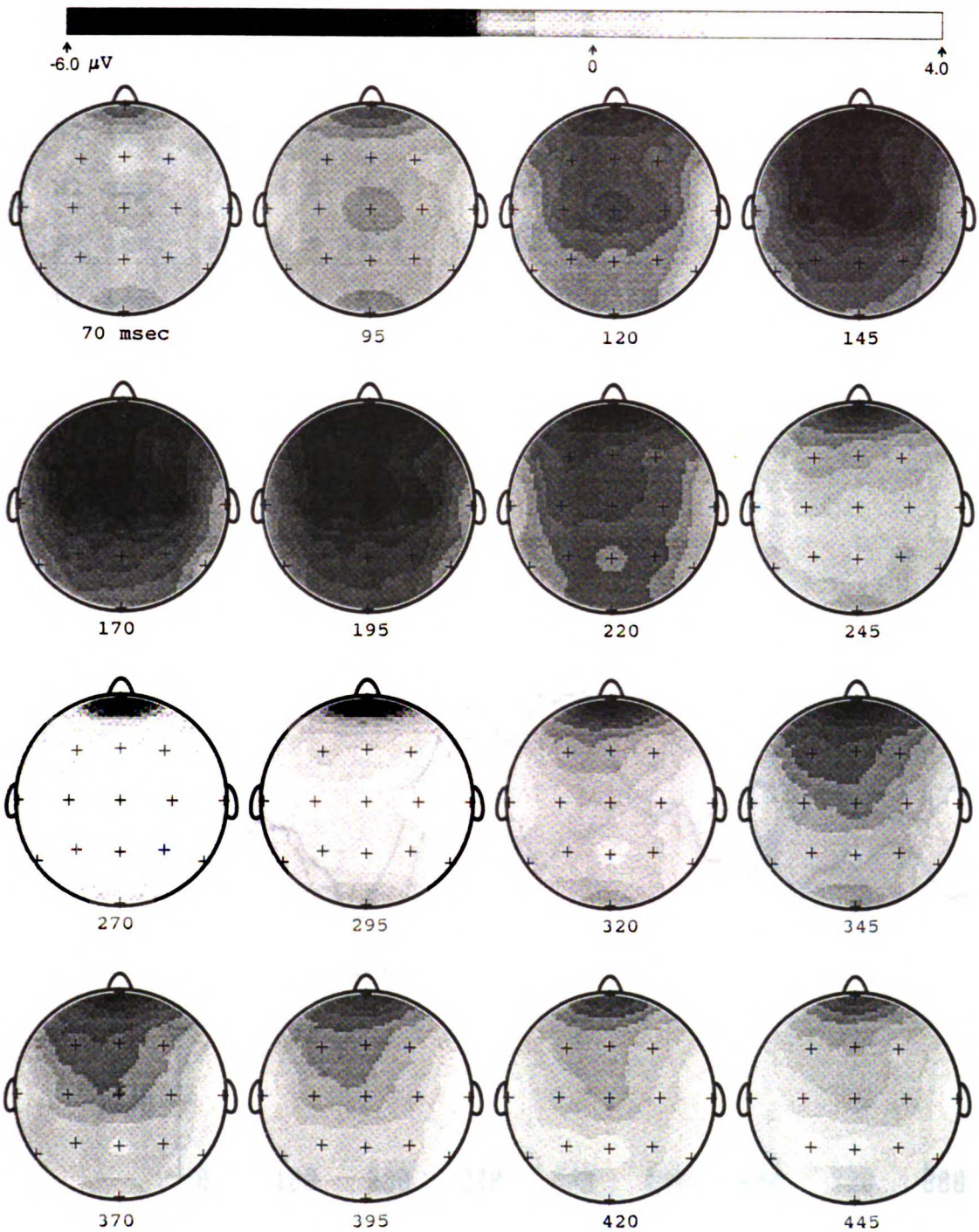


FIGURE 22. Normal Response to "POT" + Noise (Protocol 3)

FIGURE 23. Normal Response to "POT" + Noise (Protocol 3)



	Normal - "POT"	Hearing-impaired	p <
N1 lat.	169.5 ± 24.8	146.1 ± 26.5	ns
N1 amp.	-5.6 ± 3.8	-4.8 ± 4.2	ns
P2 lat.	261.4 ± 38.2	236.8 ± 45.1	ns
P2 amp.	5.7 ± 5.0	7.0 ± 6.6	ns

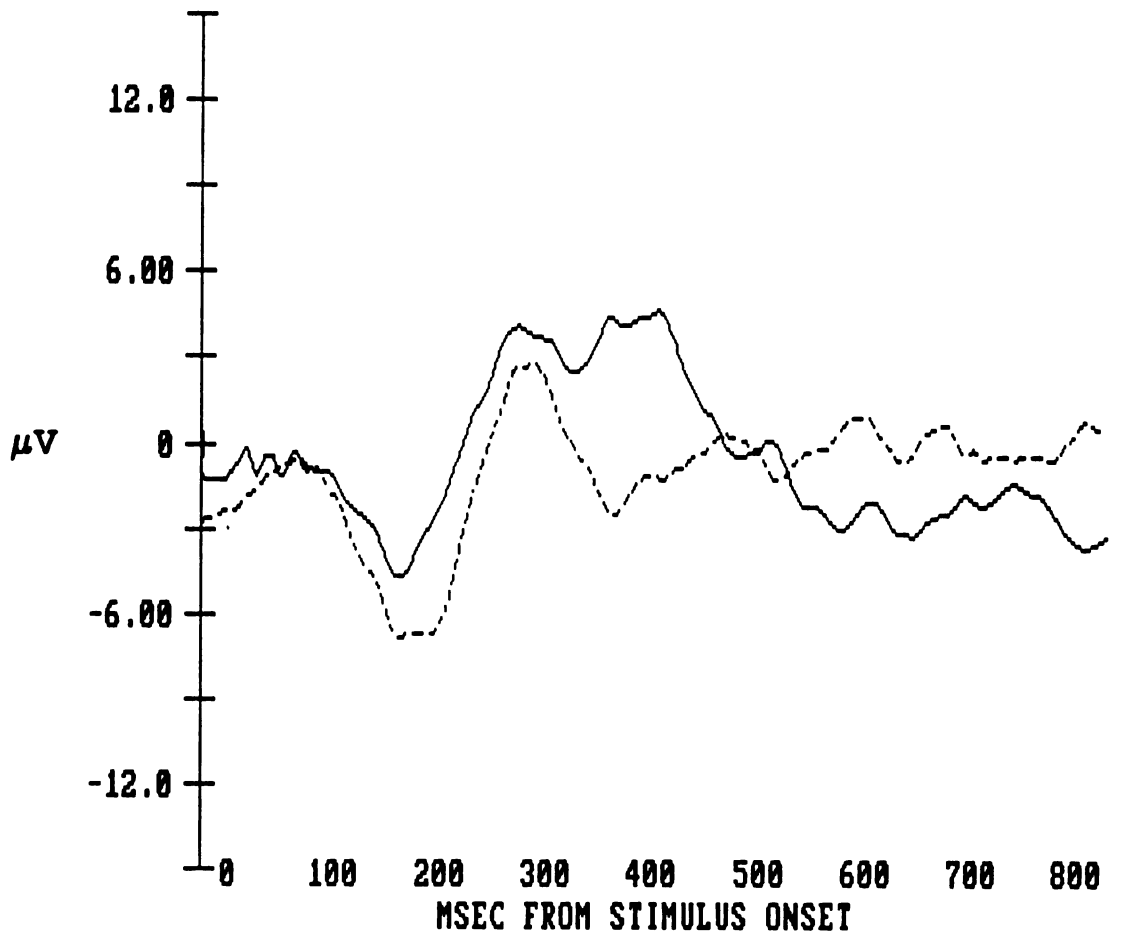


FIGURE 24. Solid = Hearing-impaired Response to "POT" (Protocol 1)
Dashed = Normal Response to "POT" + Masking Noise (Protocol 3)

	Protocol 1	Protocol 2	Protocol 3
N1 lat.	101.8 ± 10.0	132.8 ± 23.6	124.1 ± 16.4
N1 amp.	-10.1 ± 3.4	- 4.4 ± 2.1	- 5.4 ± 3.8
P2 lat.	206.0 ± 21.5	223.3 ± 41.6	189.1 ± 48.5
P2 amp.	10.4 ± 6.4	2.5 ± 3.3	2.9 ± 2.4

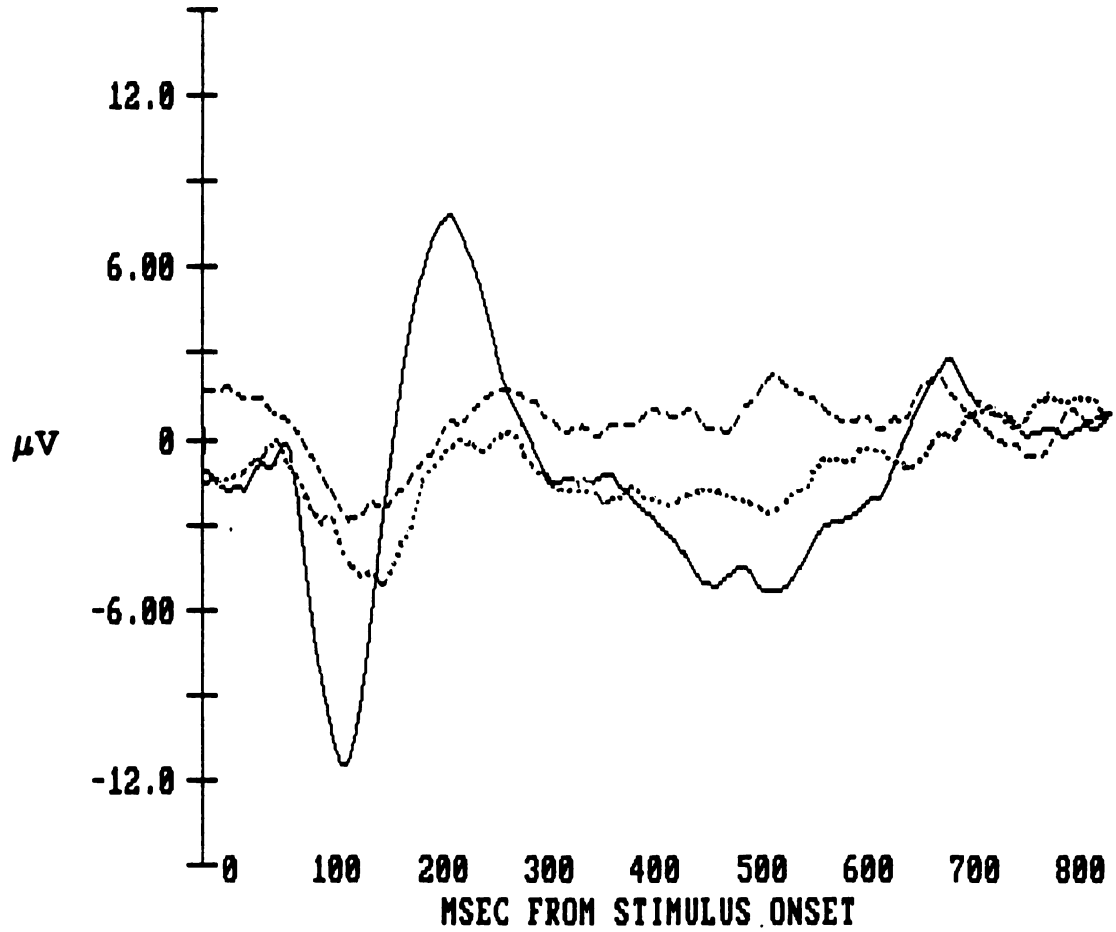


FIGURE 25. Solid = Normal Response to Tones (Protocol 1)
Dashed = Normal Response to Tones (Reduced Intensity - Protocol 2)
Dotted = Normal Response to Tones + Masking Noise (Protocol 3)

	Protocol 1	Protocol 2	Protocol 3
N1 lat.	106.4 ± 12.0	119.1 ± 18.5	169.5 ± 24.8
N1 amp.	-10.7 ± 5.2	-4.7 ± 3.2	-5.6 ± 3.8
P2 lat.	216.3 ± 21.0	208.5 ± 39.5	261.4 ± 38.2
P2 amp.	9.0 ± 3.7	5.6 ± 4.0	5.7 ± 5.0

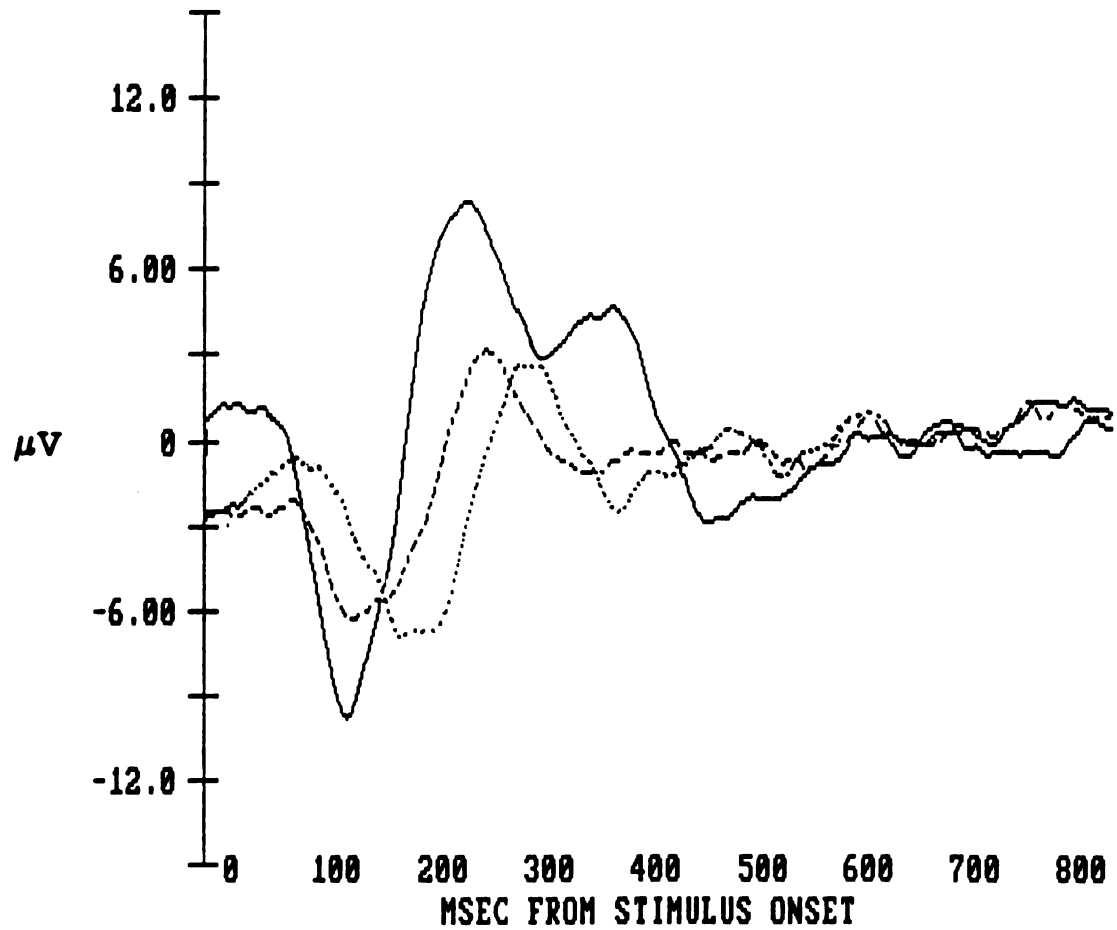


FIGURE 26. Solid = Normal Response to "POT" (Protocol 1)
Dashed = Normal Response to "POT"
(Reduced Intensity - Protocol 2)
Dotted = Normal Response to "POT" + Masking Noise
(Protocol 3)

The topographical distribution of the N1 component is more frontal in the hearing-impaired response. P2 distribution is similar in both groups: centered around Cz.

Figure 25 is a comparison of normal responses to tones in Protocols 1, 2, and 3 (recorded from Cz). Note the similar reductions of N1 and P2 amplitude in both Protocols 2 and 3 compared to Protocol 1. Notice also the significant increase in N1 latency in Protocols 2 and 3, but no significant difference in P2 latency.

Figure 26 is a comparison of normal responses to "POT" in Protocols 1, 2, and 3 (recorded from Cz). Note the reductions of N1 and P2 amplitude in Protocols 2 and 3 compared to Protocol 1. However, N1 and P2 latencies are not significantly different in Protocols 1 (74 dB stimulus) and 2 (34 dB stimulus). N1 and P2 latencies are significantly greater in Protocol 3 (stimulus + masking noise). The P3 component is evident in Protocol 1, not in 2 or 3.

In summary, AEP data from Protocols 1, 2, and 3 indicate the following:

1) Comparing normal and hearing-impaired responses to tones in Protocol 1, there are no significant differences in latencies of N1, P2, or P3 components. Greater N1 amplitude in the normal response is probably a consequence of greater subjective stimulus intensity. Greater P3 amplitude in the hearing-impaired response might be a reflection of the uncharacteristically intense stimulus (114 dB) presented to this group. The topography of AEPs in response to tones is similar in both groups.

2) Comparing normal and hearing-impaired responses to "POT" in Protocol 1, there are significant differences in N1 and P3 latency (greater in hearing-impaired) and also in N1 amplitude (greater in normal responses). Again, greater N1 amplitude

is probably caused by greater subjective stimulus intensity. Increased AEP latencies in hearing-impaired responses could be a result of lower subjective stimulus intensity, but they might also be a reflection of increased stimulus complexity. The fact that the hearing-impaired subjects could not perceive the higher frequency components of "POT" might cause delays in auditory processing. Differences in topography of N1, P2, and P3 components in response to "POT" could be indications of differences in organization of auditory pathways between the normal and hearing-impaired groups.

3) Reduced intensity tones in Protocol 2 resulted in AEPs recorded from normals that matched the N1, P2, and P3 latencies recorded from hearing-impaired subjects in Protocol 1. Greater P2 and P3 amplitudes were recorded from hearing-impaired subjects. Topography of normal AEPs remained similar to those in Protocol 1.

Reduced intensity "POT" in Protocol 2 caused a reduction in normal N1 and P2 amplitudes which were then similar to hearing-impaired N1 and P2 amplitudes in Protocol 1. Normal P3 amplitude was also reduced in Protocol 2. However, normal N1 and P2 latencies were not significantly affected by reduced intensity "POT." The topography of the normal N1 in response to "POT" was similar in Protocols 1 and 2.

4) Adding masking noise to tones in Protocol 3 resulted in N1 and P2 latencies from normals that are not significantly different from hearing-impaired responses. Hearing-impaired P2 and P3 amplitudes were greater than normal responses to tones in Protocol 3. Normal N1 topography remained stable even with the addition of masking noise.

Adding masking noise to "POT" in Protocol 3 resulted in N1 and P2 latencies and amplitudes from normals that are not significantly different from hearing-impaired responses. Hearing-impaired P3 amplitude remained significantly greater. Even though the latencies changed, the topographies of normal N1 and P2 components in response to "POT" + noise remained similar to normal topographies in Protocols 1 and 2. That is, normal AEP topographies in response to "POT" were not transformed to hearing-impaired patterns by reducing stimulus intensity nor by adding masking noise to simulate hearing loss. This finding supports the hypothesis that AEP topography is a reflection of neural organization and is not necessarily determined by stimulus parameters alone.

PROTOCOL 4 - A random presentation
of 30-40 each:
Condition 1: 50 msec 250 Hz tone burst
Condition 2: 100 msec 250 Hz tone burst
Condition 3: 500 msec 250 Hz tone burst
Condition 4: 50 msec pause between noise bursts
Condition 5: 100 msec pause between noise bursts
Condition 6: silence (an indicator of background EEG activity)
Condition 7: 250 msec vowel ("AH")
Condition 8: 250 msec consonant-vowel ("PA")
Condition 9: 500 msec word ("POT")

Normal-hearing Subjects, Conditions 1-3: Figure 27 contains normal responses to 250 Hz tones of different durations. N1 and P2 latencies and amplitudes are not significantly different between conditions 1 and 2 (50 msec and 100 msec duration tones). However, N1 latencies in condition 3 (500 msec duration tones) are greater than N1 latencies in condition 1 ($p < .015$) and condition 2 ($p < .05$). P2 latencies are not significantly different across the three conditions. N1 and P2 amplitudes are not significantly different between conditions 1 and 2. However, N1 amplitudes are smaller in condition 3 than in condition 1 ($p < .05$). Also, P2 amplitudes are smaller in condition 3 than in condition 1 ($p < .0075$) and condition 2 ($p < .01$).

Figure 28 is a topographical map of normal responses to 500 msec duration tones. The topography of AEPs here is similar to normal responses to tones in Protocol 1. Figure 29 is a comparison of normal responses to 500 msec duration tones in Protocols 1 and 4. Note the decreases in N1 and P2 amplitudes and the increase in N1 latency in Protocol 4. These differences are probably caused by variations in subject attention between the protocols.

Hearing-impaired Subjects, Conditions 1-3: Figure 30 contains hearing-impaired

	50 msec Tones	100 msec Tones	500 msec Tones
N1 lat.	100.4 ± 9.6	106.9 ± 9.6	122.0 ± 20.2
N1 amp.	-11.8 ± 5.5	-9.3 ± 4.5	-7.5 ± 3.5
P2 lat.	194.1 ± 25.2	213.3 ± 20.9	216.5 ± 20.5
P2 amp.	7.5 ± 3.8	6.8 ± 3.4	2.9 ± 2.2

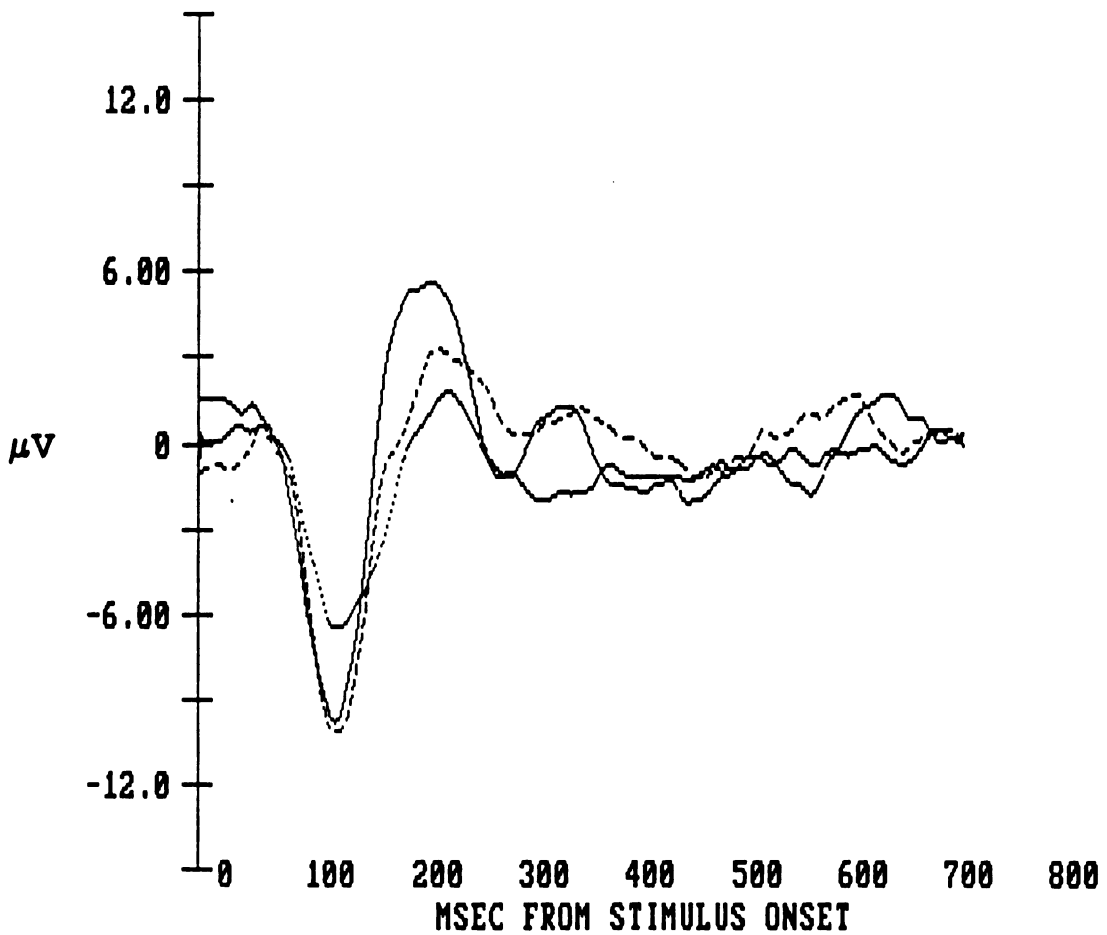
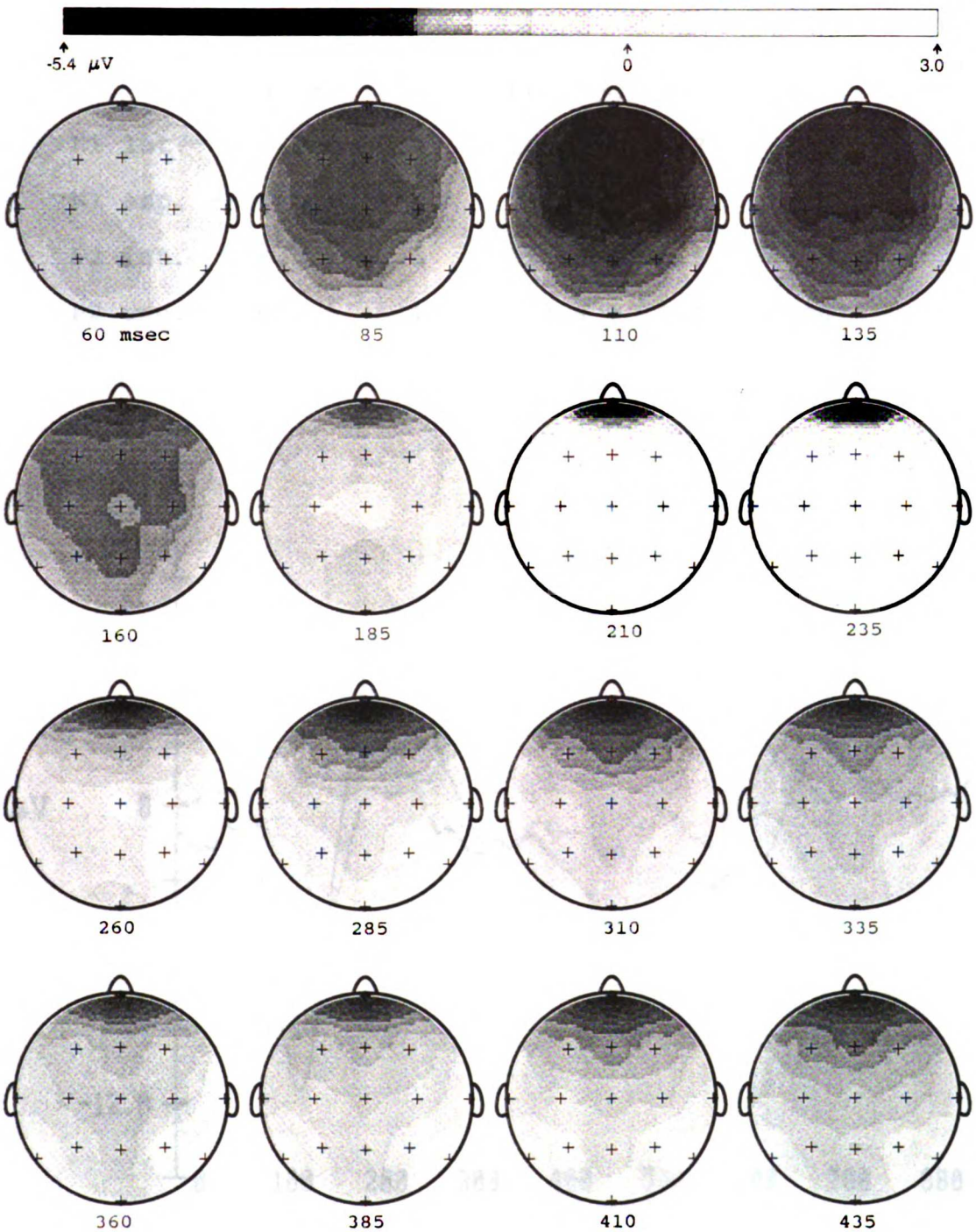


FIGURE 27. Solid = Normal Response to Tones (50 msec)
Dashed = Normal Response to Tones (100 msec)
Dotted = Normal Response to Tones (500 msec)

FIGURE 28. Normal Response to 500 msec Tones (Protocol 4)



	Attending (Protocol 1)	Reading (Protocol 4)	p <
N1 lat.	101.8 ± 10.0	122.0 ± 20.2	.015
N1 amp.	-10.1 ± 3.4	-7.5 ± 3.5	ns
P2 lat.	206.0 ± 21.5	216.5 ± 20.5	ns
P2 amp.	10.4 ± 6.4	2.9 ± 2.2	.0075

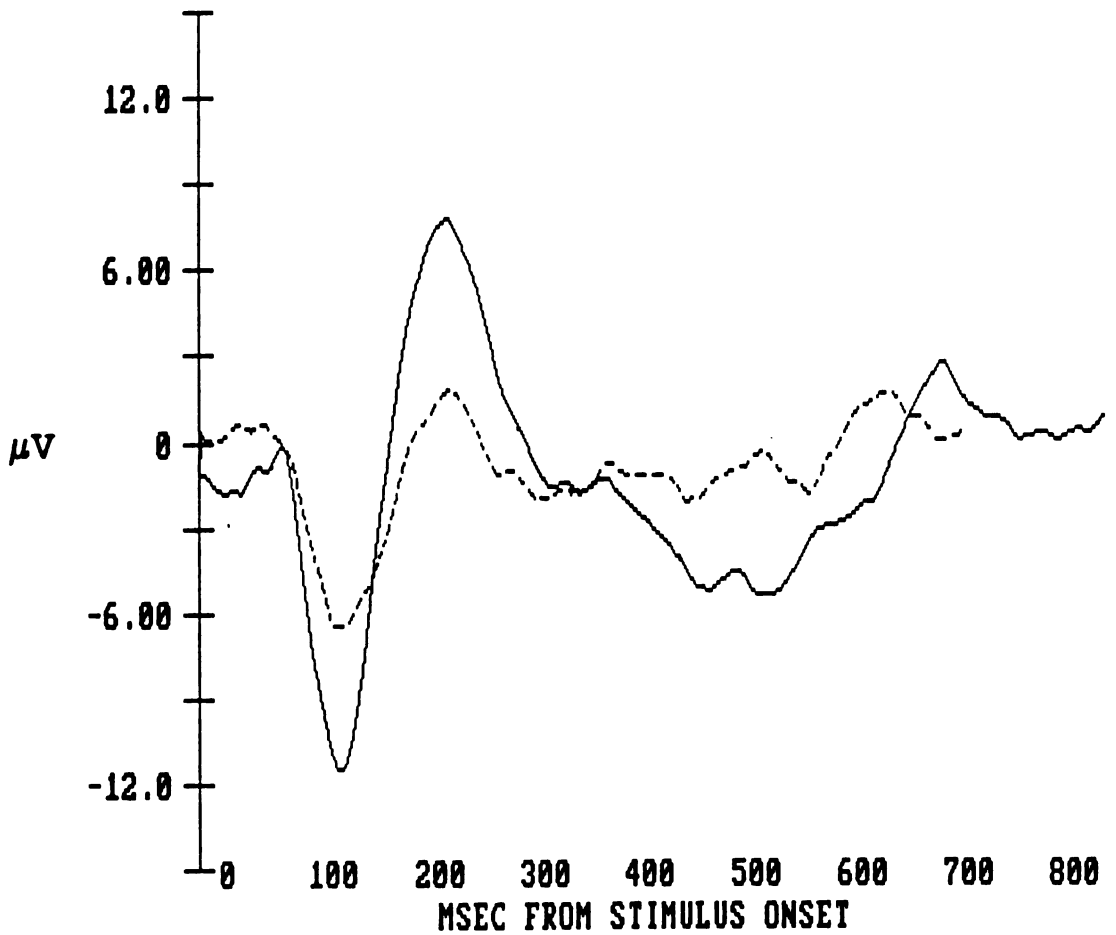


FIGURE 29. Solid = Normal Response to Tones (500 msec)
(Protocol 1, Condition 1 - Attending)
Dashed = Normal Response to Tones (500 msec)
(Protocol 4, Condition 3 - Reading)

	50 msec Tones	100 msec Tones	500 msec Tones
N1 lat.	106.9 ± 31.3	130.8 ± 25.5	130.1 ± 22.9
N1 amp.	-3.6 ± 3.2	-2.8 ± 2.2	-2.9 ± 2.5
P2 lat.	191.3 ± 17.3	191.6 ± 27.2	206.8 ± 42.7
P2 amp.	5.0 ± 4.8	4.3 ± 4.1	3.4 ± 2.4

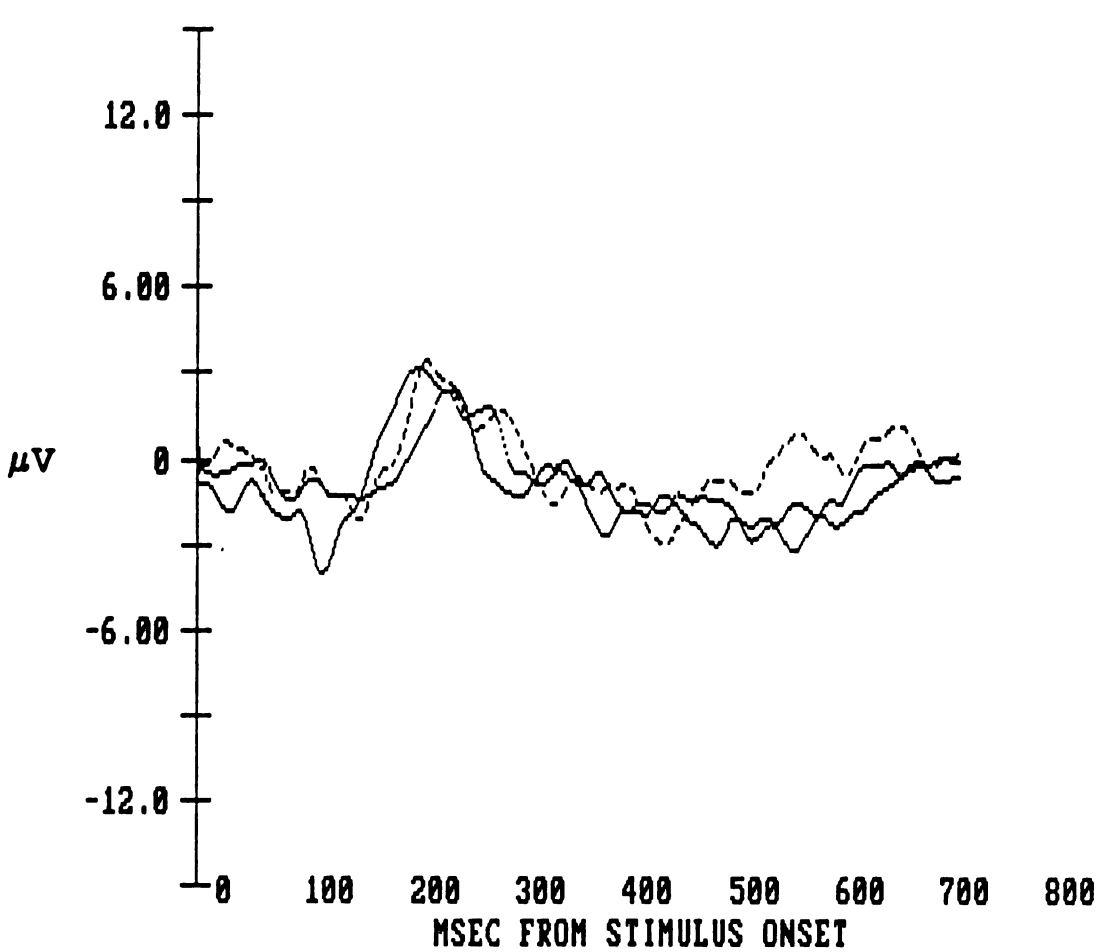
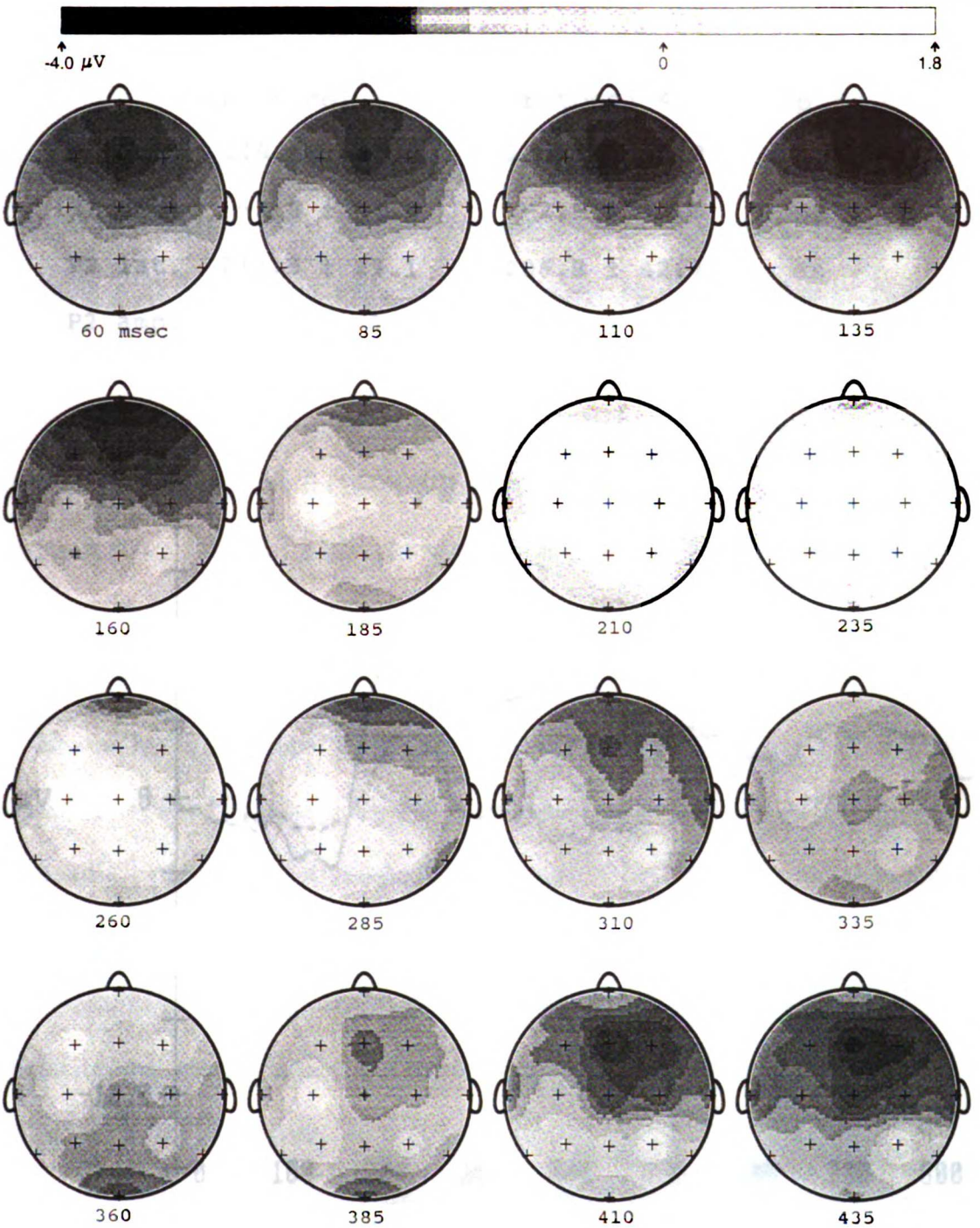


FIGURE 30. Solid = Hearing-impaired Response to Tones (50 msec)
Dashed = Hearing-impaired Response to Tones (100 msec)
Dotted = Hearing-impaired Response to Tones (500 msec)

FIGURE 31. Hearing-impaired Response to 500 msec Tones (Protocol 4)



	Attending (Protocol 1)	Reading (Protocol 4)	p <
N1 lat.	119.5 ± 26.7	130.1 ± 22.9	ns
N1 amp.	-3.8 ± 4.0	-2.9 ± 2.5	ns
P2 lat.	213.5 ± 29.1	206.8 ± 42.7	ns
P2 amp.	8.0 ± 3.3	3.4 ± 2.4	.005

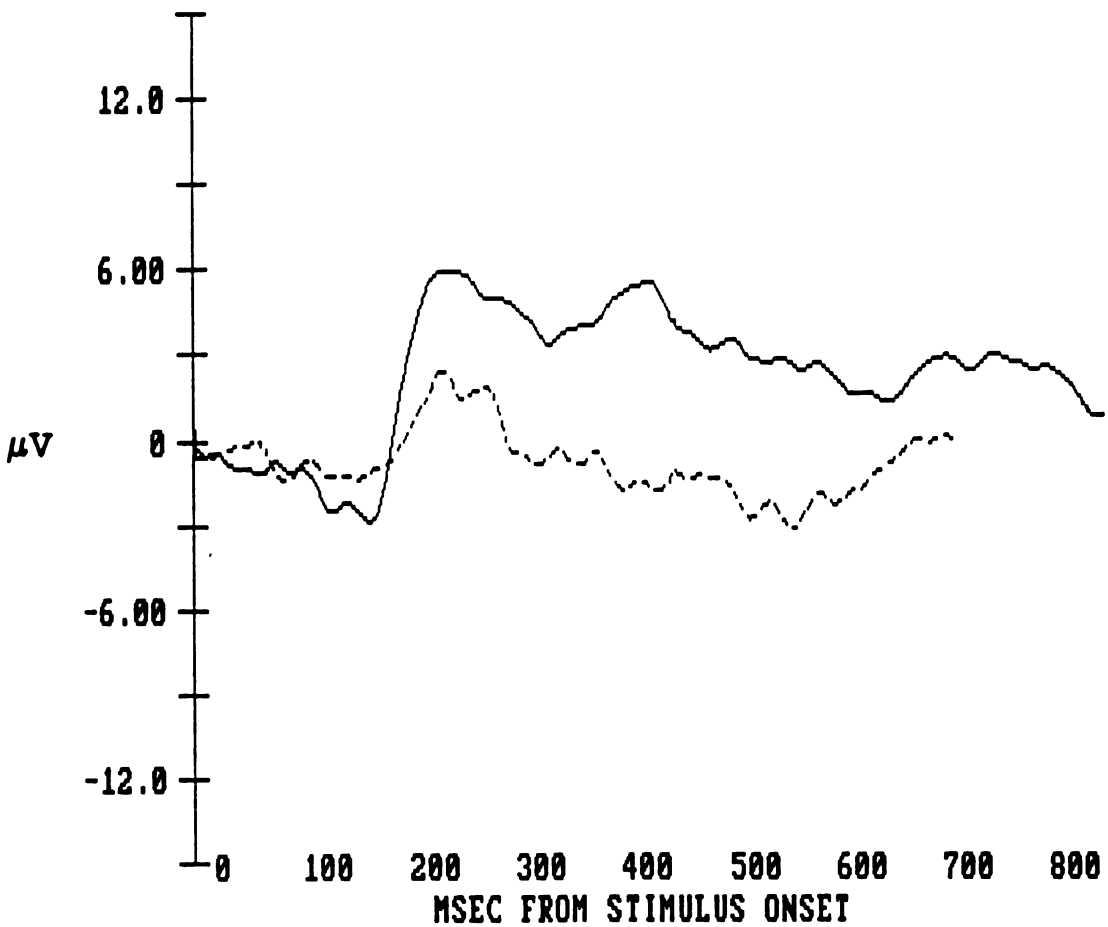


FIGURE 32. Solid = Hearing-impaired Response to Tones (500 msec) (Protocol 1, Condition 1 - Attending)
Dashed = Hearing-impaired Response to Tones (500 msec) (Protocol 4, Condition 3 - Reading)

responses to 250 Hz tones of different durations. N1 and P2 latencies and amplitudes are not significantly different across the three conditions. Figure 31 is a topographic map of hearing-impaired responses to 500 msec duration tones in Protocol 4. The distribution of components is similar to hearing-impaired responses in Protocol 1. Figure 32 is a comparison between hearing-impaired responses to 500 msec duration tones in Protocols 1 and 4. Latencies of components are not significantly different. However, P2 and P3 amplitudes are greater in Protocol 1. Again, variations in subject attention probably account for these differences. Compared to responses from normal-hearing subjects, N1 and P2 latencies are not significantly different. Hearing-impaired N1 amplitudes are smaller in each condition; P2 amplitudes are not significantly different.

Normal-hearing Subjects, Conditions 4 and 5: Figure 33 contains normal responses to continuous white noise after a pause (50 or 100 msec in duration) in continuous white noise. There are no significant differences in N1 or P2 latencies or amplitudes in these onset responses (when the 50 msec onset delay is taken into account).

Hearing-impaired Subjects, Conditions 4 and 5: Figure 34 contains hearing-impaired responses to continuous noise after a pause. There are no significant differences in N1 or P2 latencies or amplitudes within this subject population. Compared to the normal responses, hearing-impaired N1 latencies are greater in both conditions ($p < .0025$ for both). P2 latencies are also greater in the hearing-impaired responses ($p < .005$ and $p < .02$ for conditions 4 and 5 respectively). N1 and P2

	Onset After 50 msec Pause	Onset After 100 msec Pause
N1 lat.	123.9 ± 35.7	120.3 ± 15.5
N1 amp.	-3.4 ± 1.7	-3.9 ± 2.3
P2 lat.	176.3 ± 24.1	189.3 ± 20.1
P2 amp.	1.3 ± 1.7	3.0 ± 2.5

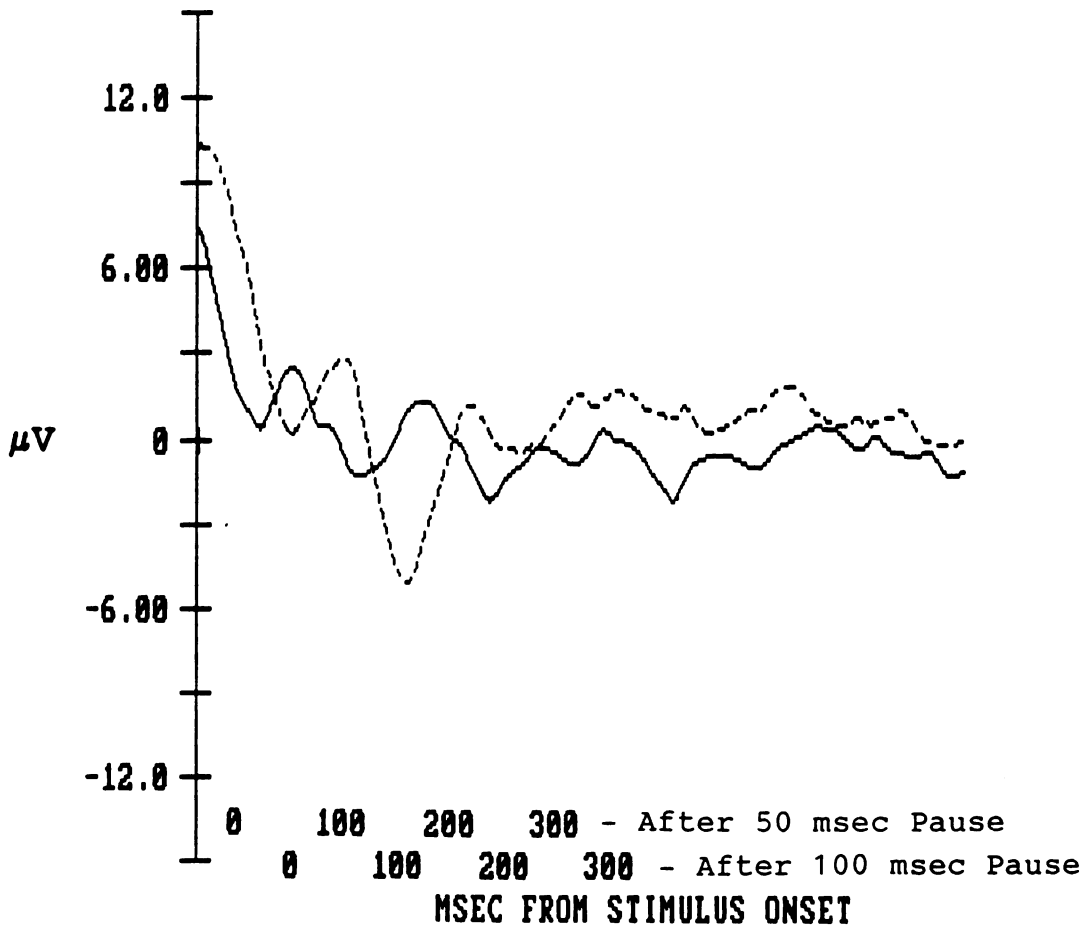


FIGURE 33. Solid = Normal Response to Noise After 50 msec Pause
Dashed = Normal Response to Noise After 100 msec Pause

	Onset After 50 msec Pause	Onset After 100 msec Pause
N1 lat.	176.3 ± 15.8	171.4 ± 24.8
N1 amp.	-4.9 ± 1.9	-3.6 ± 1.2
P2 lat.	225.1 ± 32.5	258.0 ± 66.1
P2 amp.	1.0 ± 1.6	2.2 ± 1.6

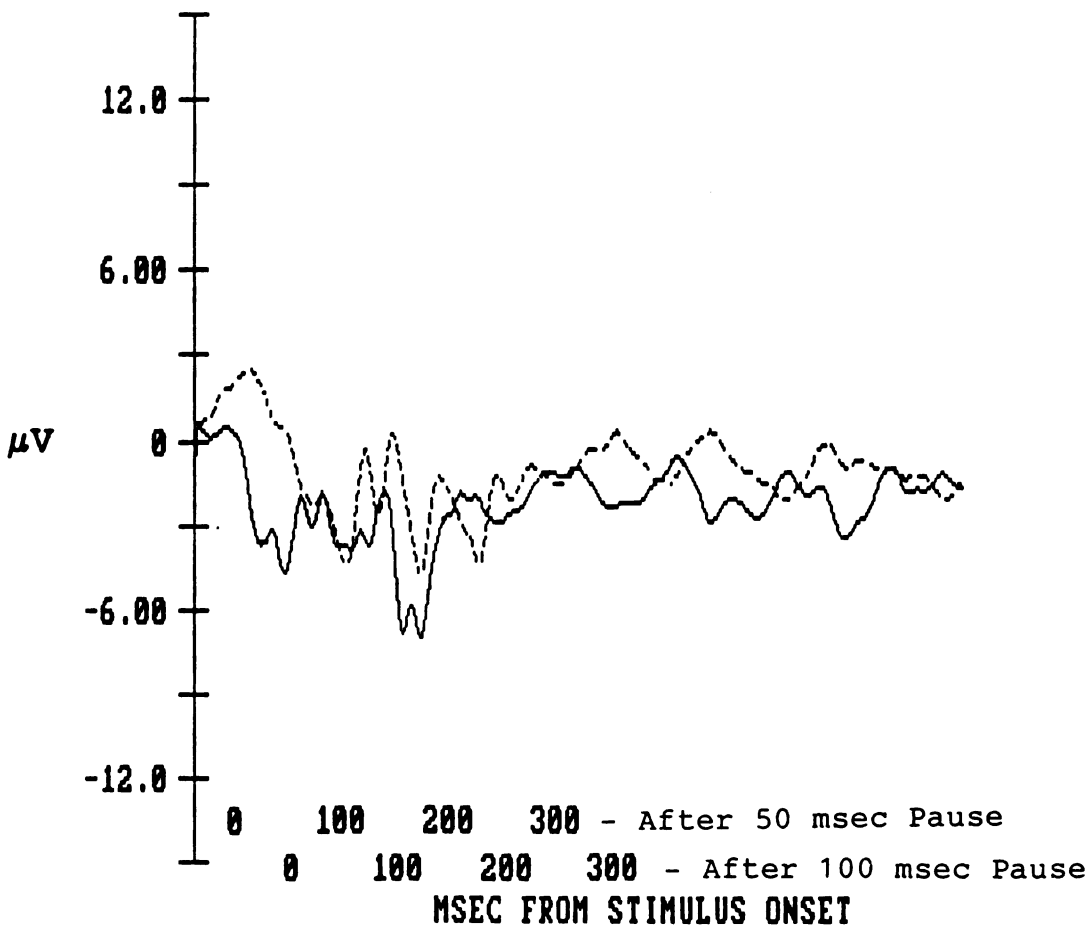


FIGURE 34. Solid = Hearing-impaired Response to Noise After 50 msec Pause
Dashed = Hearing-impaired Response to Noise After 100 msec Pause

amplitudes are not significantly different.

Normal-hearing Subjects, Condition 6: Figure 35 contains the normal response to silence. This is an indicator of background noise during this protocol.

Hearing-impaired Subjects, Condition 6: Figure 35 also contains the hearing-impaired response to silence.

Normal-hearing Subjects, Condition 7: Figure 36 is a topographical map of the normal response to the vowel "AH" (250 msec duration).

Hearing-impaired Subjects, Condition 7: Figure 37 is a topographical map of the hearing-impaired response to "AH" (250 msec duration). Compared to the normal map, the topography of N1 is shifted more to the left hemisphere. The distribution of P2 is slightly more frontal in the hearing-impaired map.

Figure 38 is a comparison of normal and hearing-impaired responses to "AH" recorded from Cz. Hearing-impaired N1 and P2 latencies are significantly greater than the normals. N1 amplitude is greater in normal responses. P2 amplitude is not significantly different. These group differences are probably caused by a combination of 1) differences in subjective stimulus intensity; 2) differences in group ability to perceive the high frequency components of speech; and 3) differences in organization of central auditory pathways.

Normal-hearing Subjects, Condition 8: Figure 39 is a topographic map of the normal response to the consonant-vowel "PA" (250 msec duration). Note the P3 component in the parietal/occipital region which was not evident in the normal response to "AH."

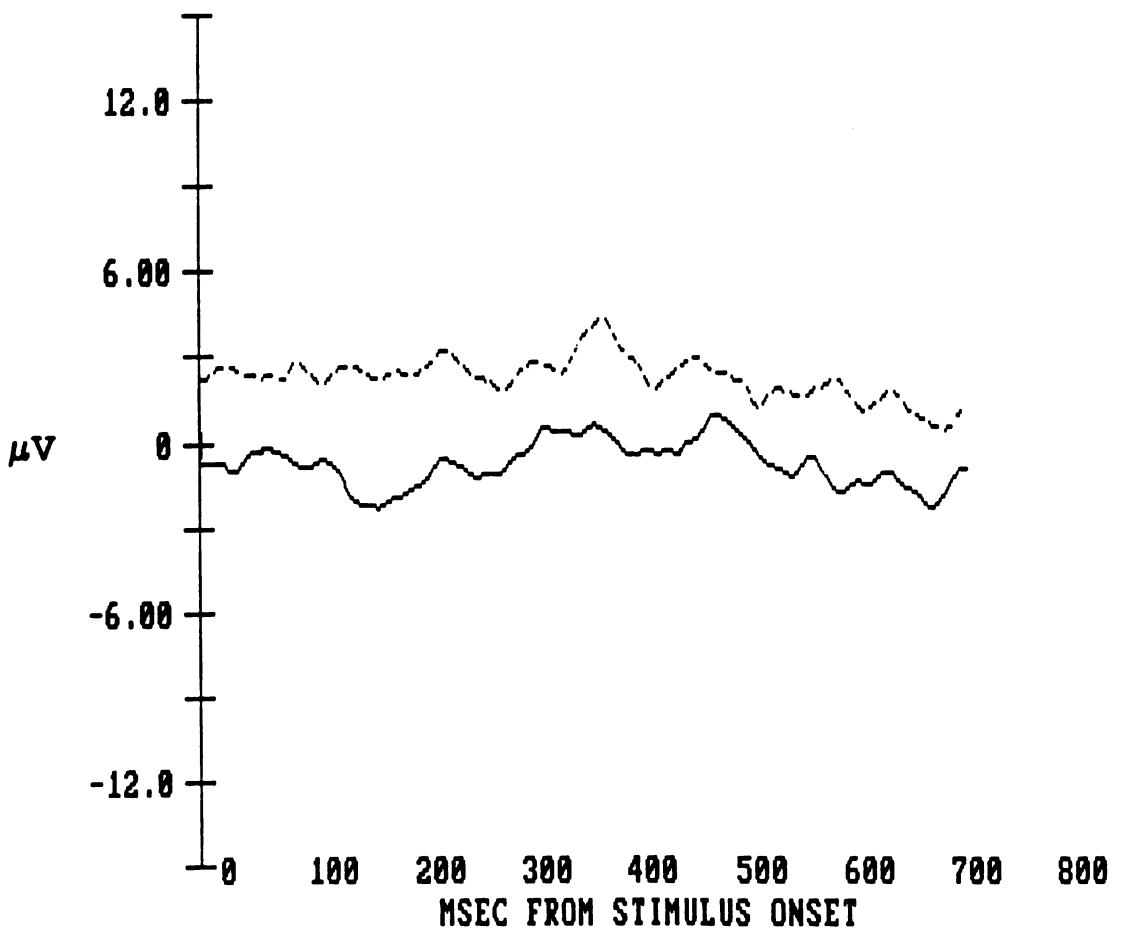


FIGURE 35. Solid = Normal Response to Silence
Dashed = Hearing-impaired Response to Silence

FIGURE 36. Normal Response to "AH" (Protocol 4)

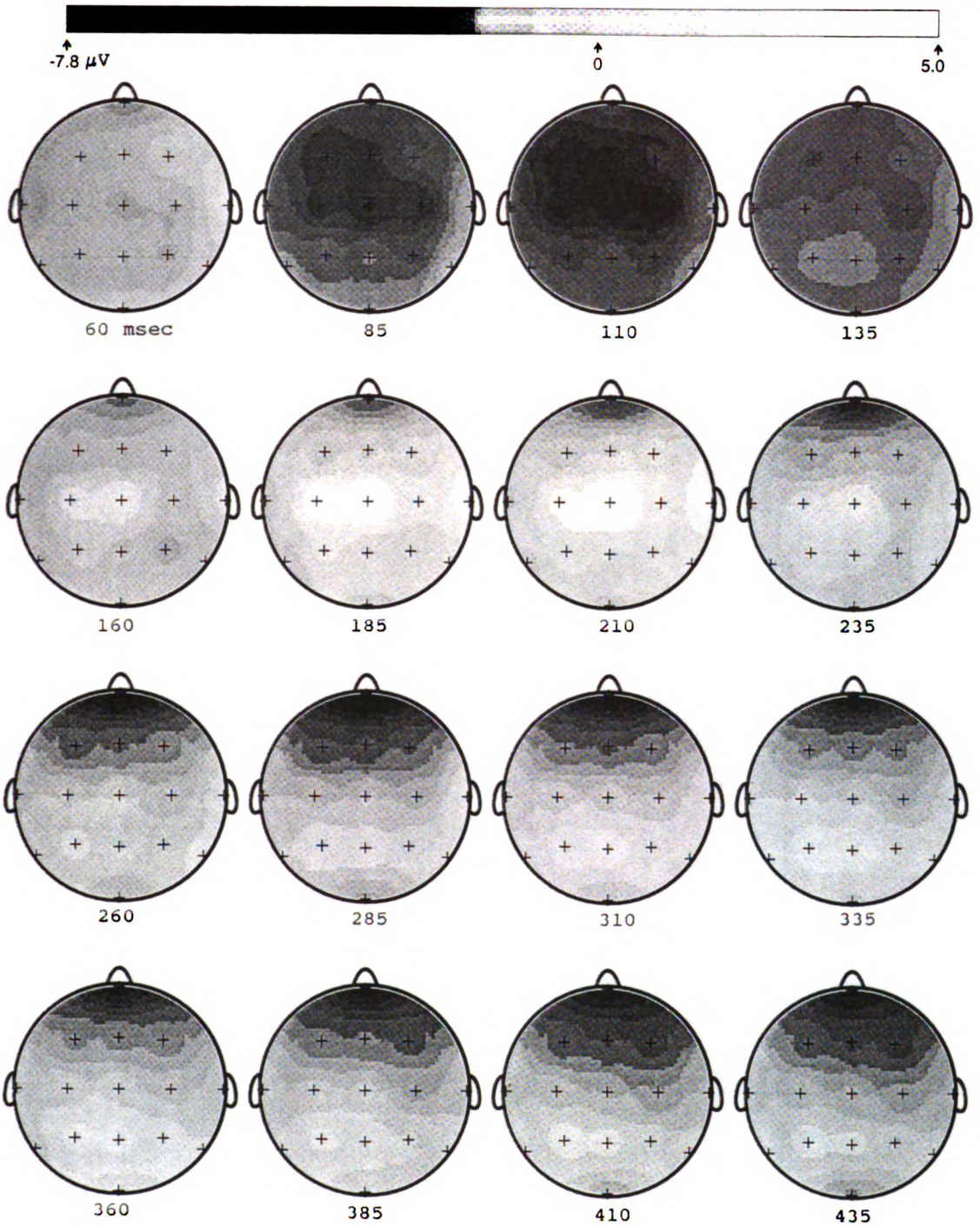


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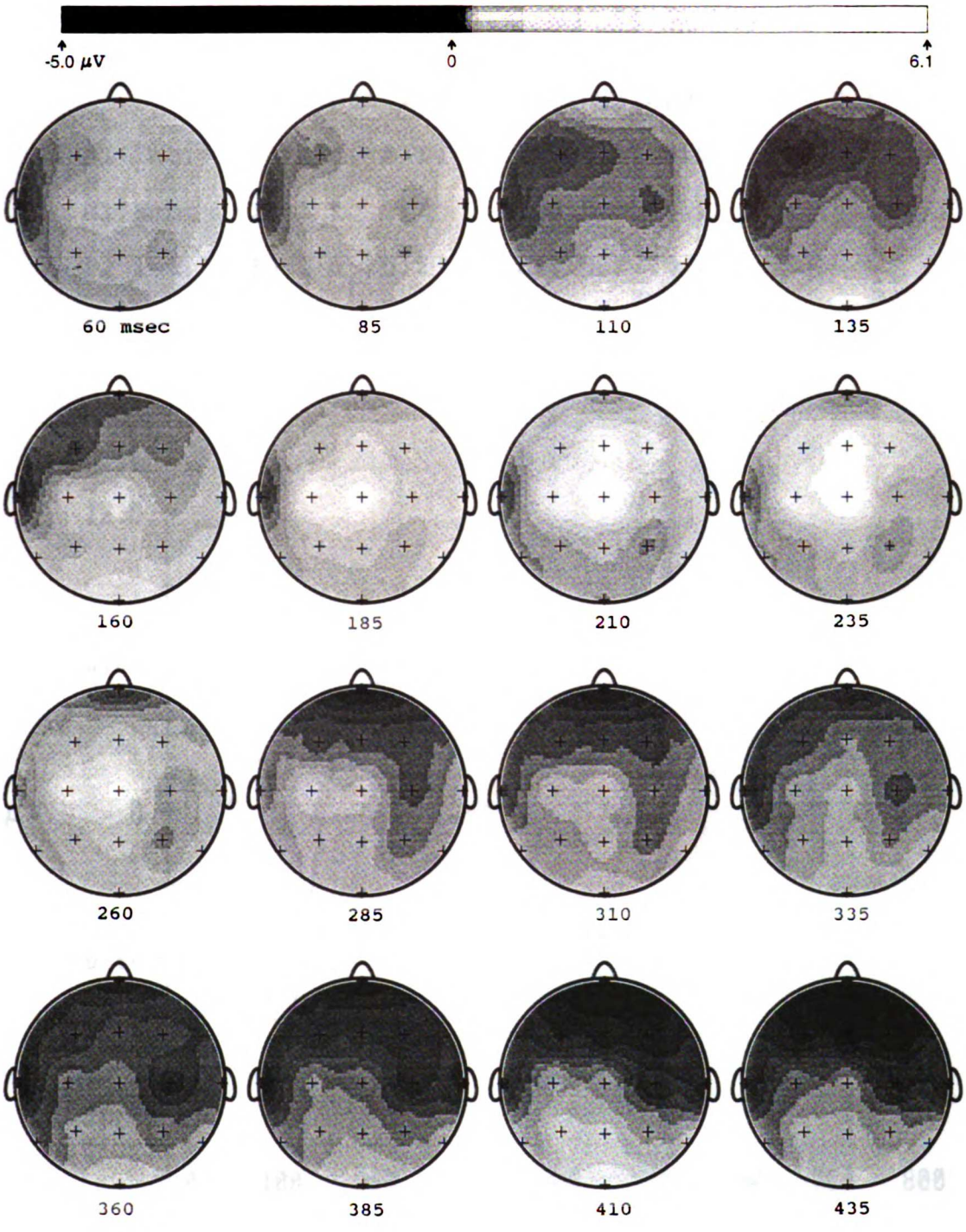
FIGURE 37. Hearing-impaired Response to "AH" (Protocol 4)



	Normal-hearing	Hearing-impaired	p <
N1 lat.	103.1 ± 8.1	123.5 ± 29.9	.05
N1 amp.	-11.5 ± 3.7	-6.5 ± 4.2	.05
P2 lat.	188.1 ± 20.2	224.0 ± 38.2	.025
P2 amp.	4.2 ± 4.2	6.1 ± 4.3	ns

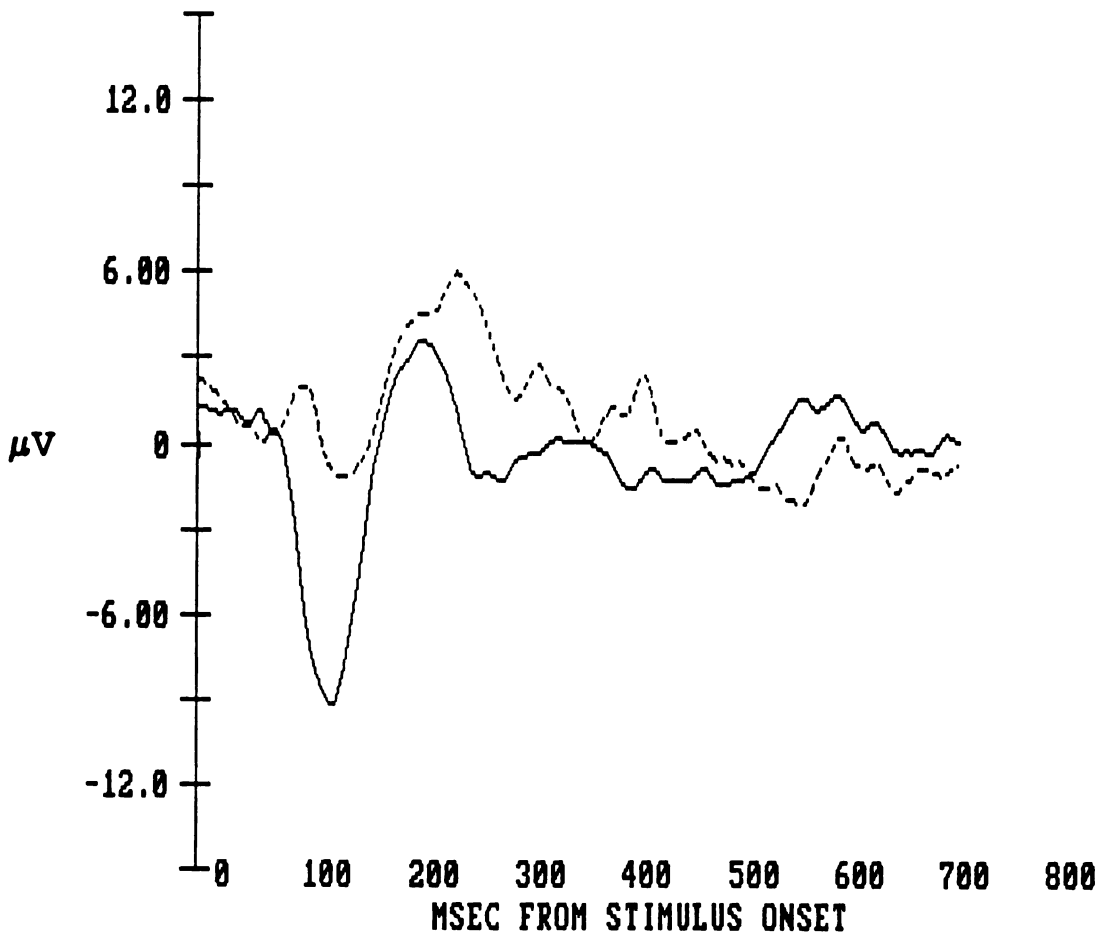


FIGURE 38. Solid = Normal Response to "AH" (250 msec)
Dashed = Hearing-impaired Response to "AH" (250 msec)

FIGURE 39. Normal Response to "PA" (Protocol 4)

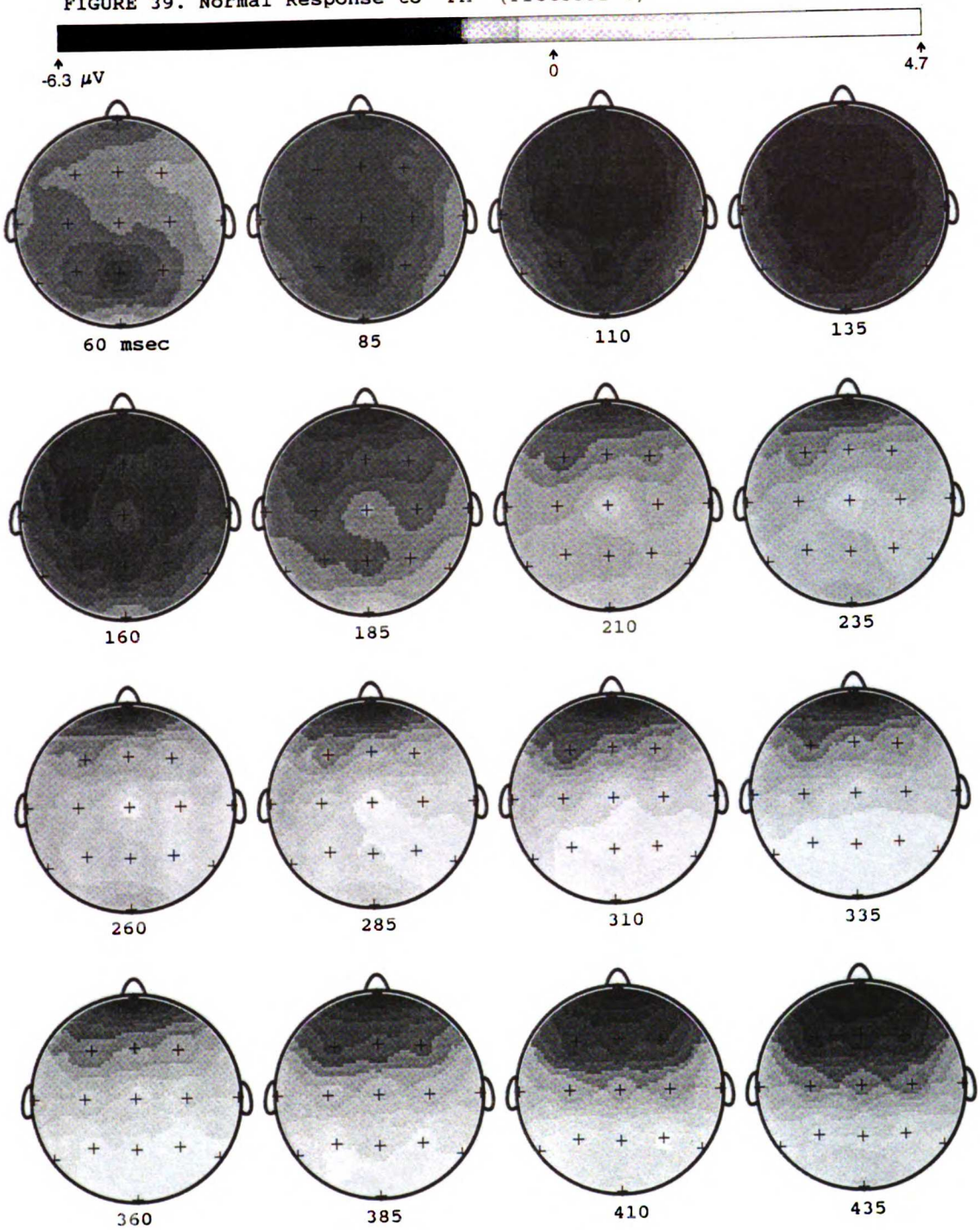
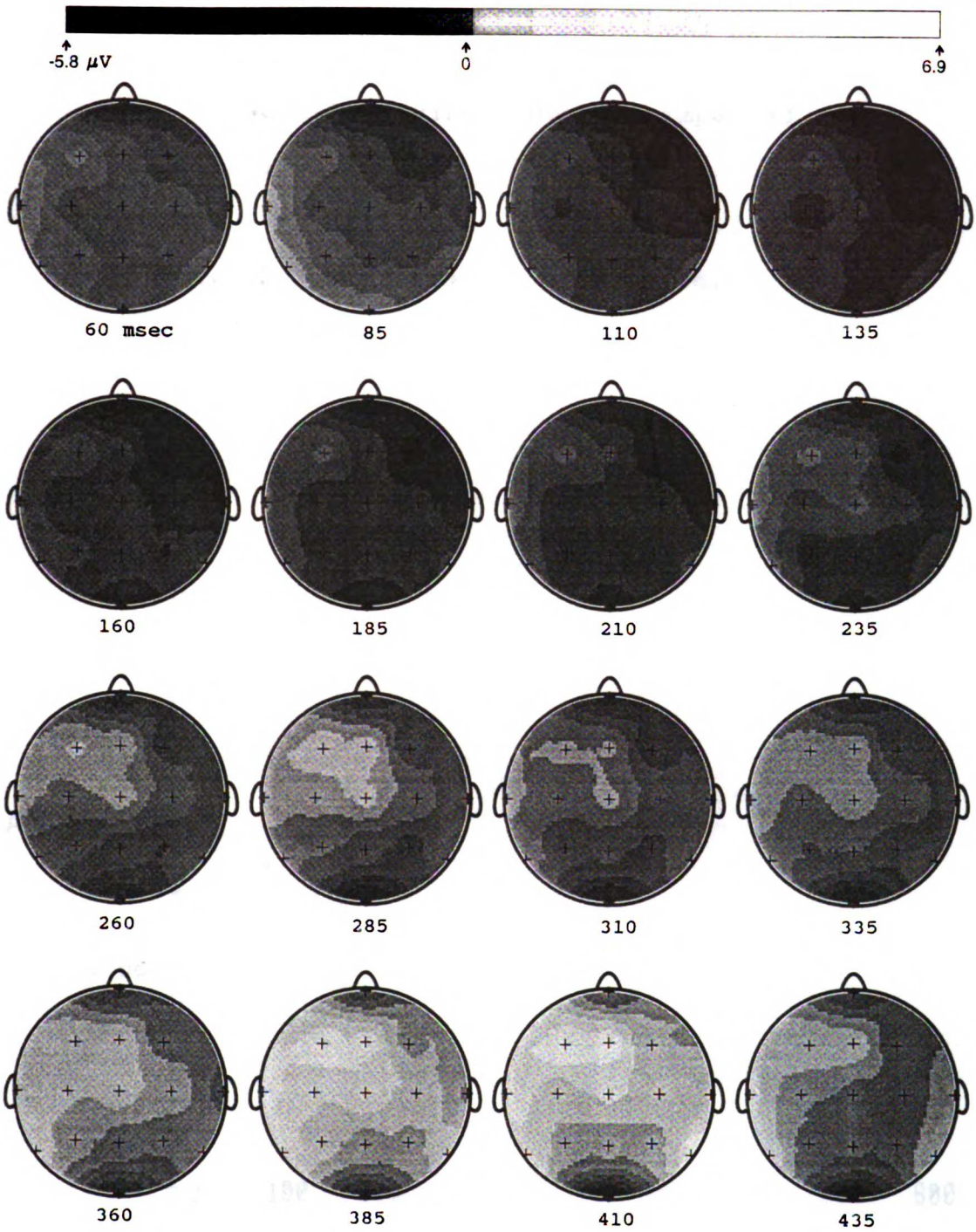


FIGURE 40. Hearing-impaired Response to "PA" (Protocol 4)



	Normal-hearing	Hearing-impaired	p <
N1 lat.	120.9 ± 13.5	150.8 ± 36.6	.05
N1 amp.	-5.6 ± 3.2	-3.4 ± 1.7	ns
P2 lat.	223.6 ± 28.9	273.3 ± 86.8	ns
P2 amp.	5.2 ± 3.9	5.1 ± 4.6	ns

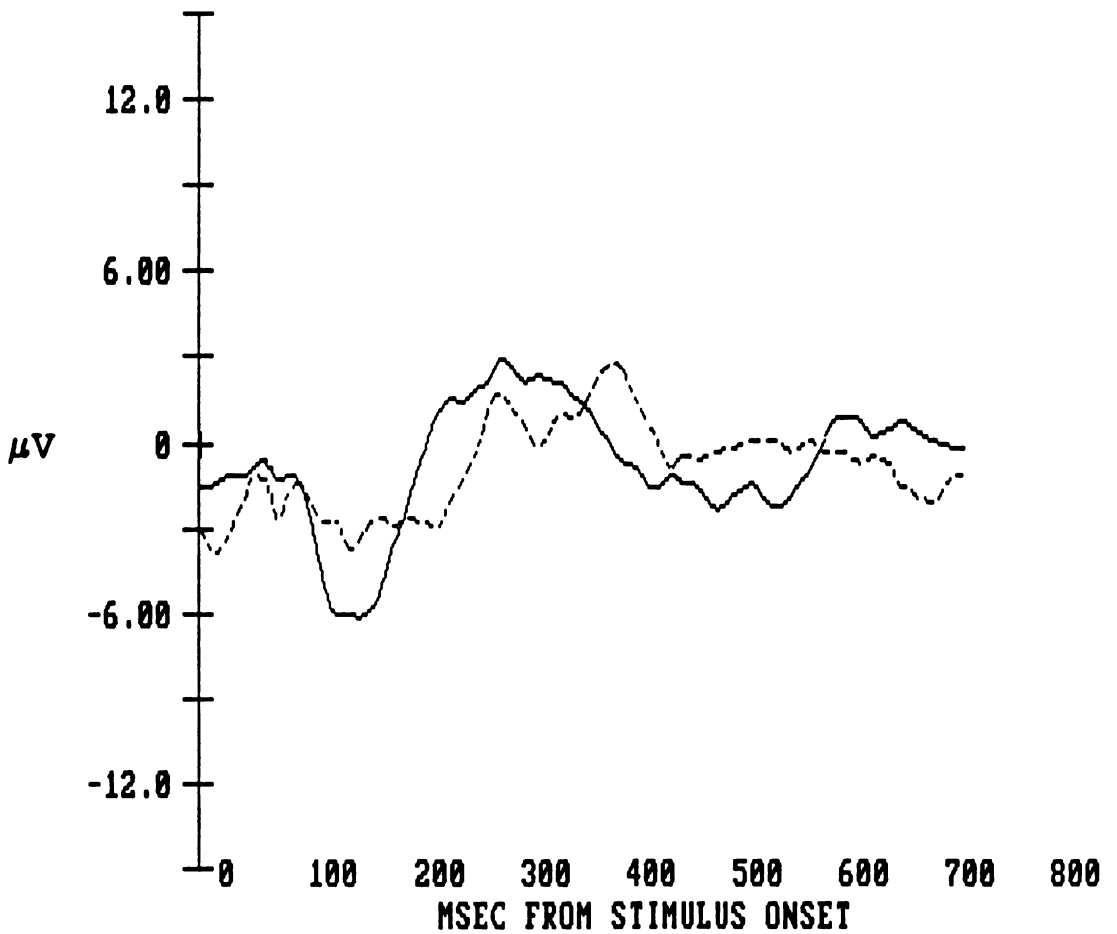
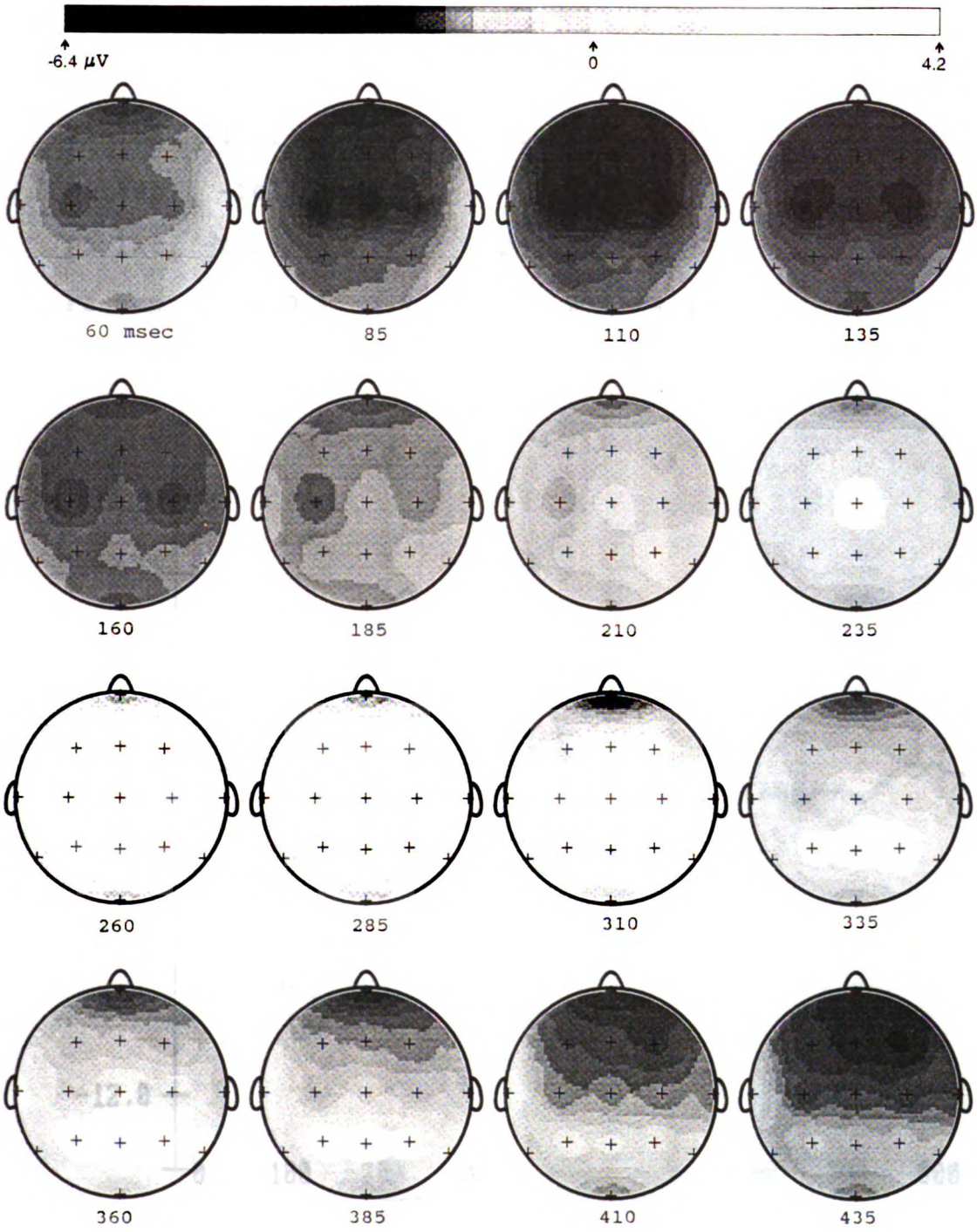


FIGURE 41. Solid = Normal Response to "PA" (250 msec)
Dashed = Hearing-impaired Response to "PA" (250 msec)

FIGURE 42. Normal Response to "POT" (Protocol 4)



	Attending (Protocol 1)	Reading (Protocol 4)
N1 lat.	106.4 ± 12.0	99.0 ± 17.1
N1 amp.	-10.7 ± 5.2	-7.0 ± 3.8
P2 lat.	216.3 ± 21.0	242.3 ± 37.9
P2 amp.	9.0 ± 3.7	6.2 ± 4.1

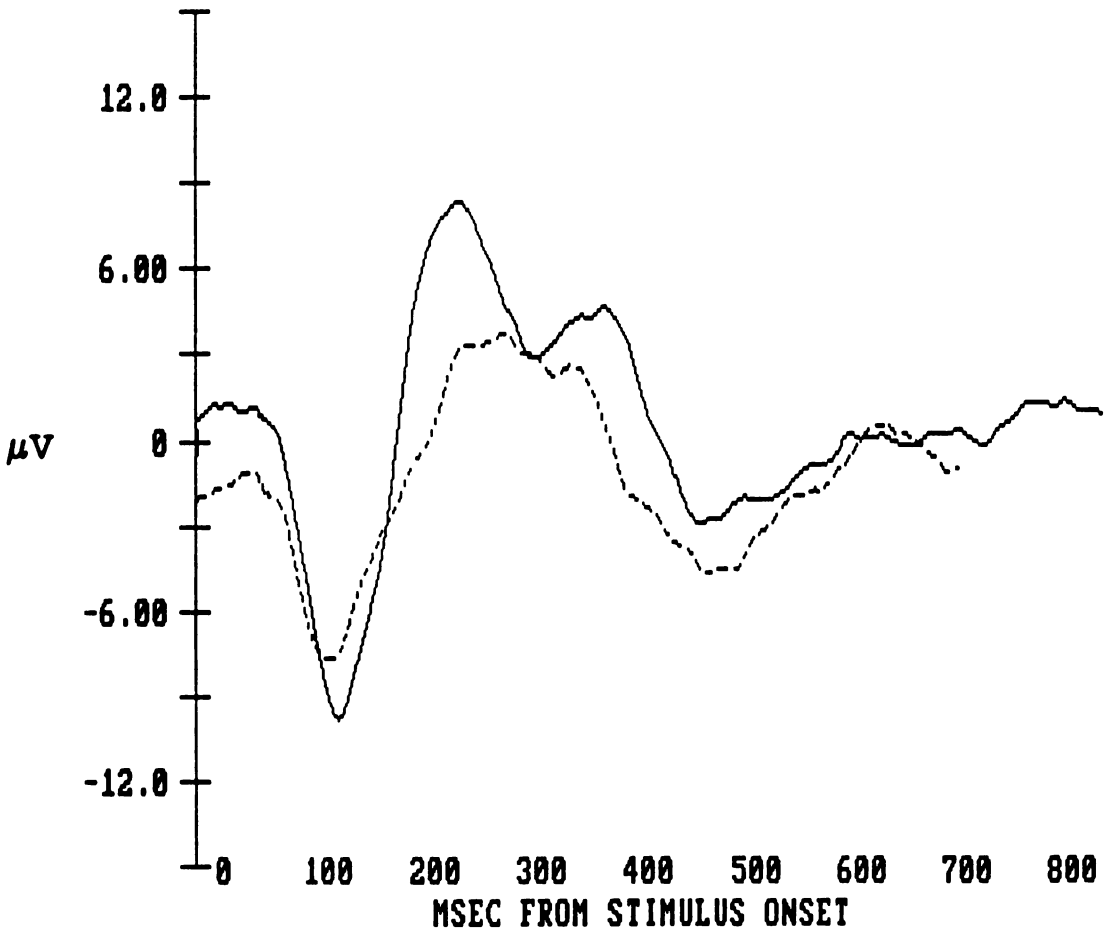
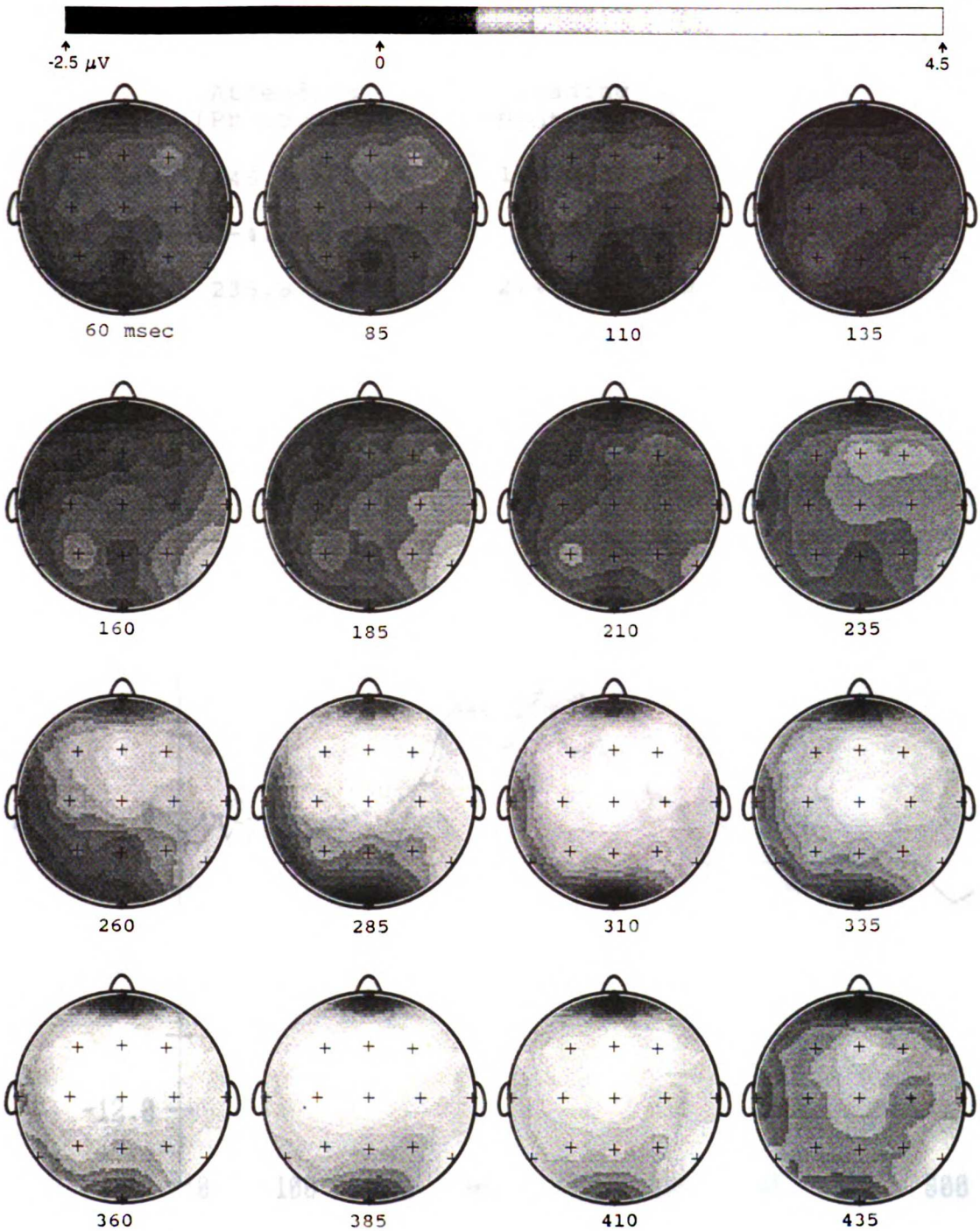


FIGURE 43. Solid = Normal Response to "POT"
(Protocol 1, Condition 2 - Attending)
Dashed = Normal Response to "POT"
(Protocol 4, Condition 9 - Reading)

FIGURE 44. Hearing-impaired Response to "POT" (Protocol 4)



	Attending (Protocol 1)	Reading (Protocol 4)
N1 lat.	146.1 ± 26.5	146.0 ± 39.5
N1 amp.	-4.8 ± 4.2	-2.6 ± 1.7
P2 lat.	236.8 ± 45.1	274.5 ± 47.3
P2 amp.	7.0 ± 6.6	4.5 ± 3.6

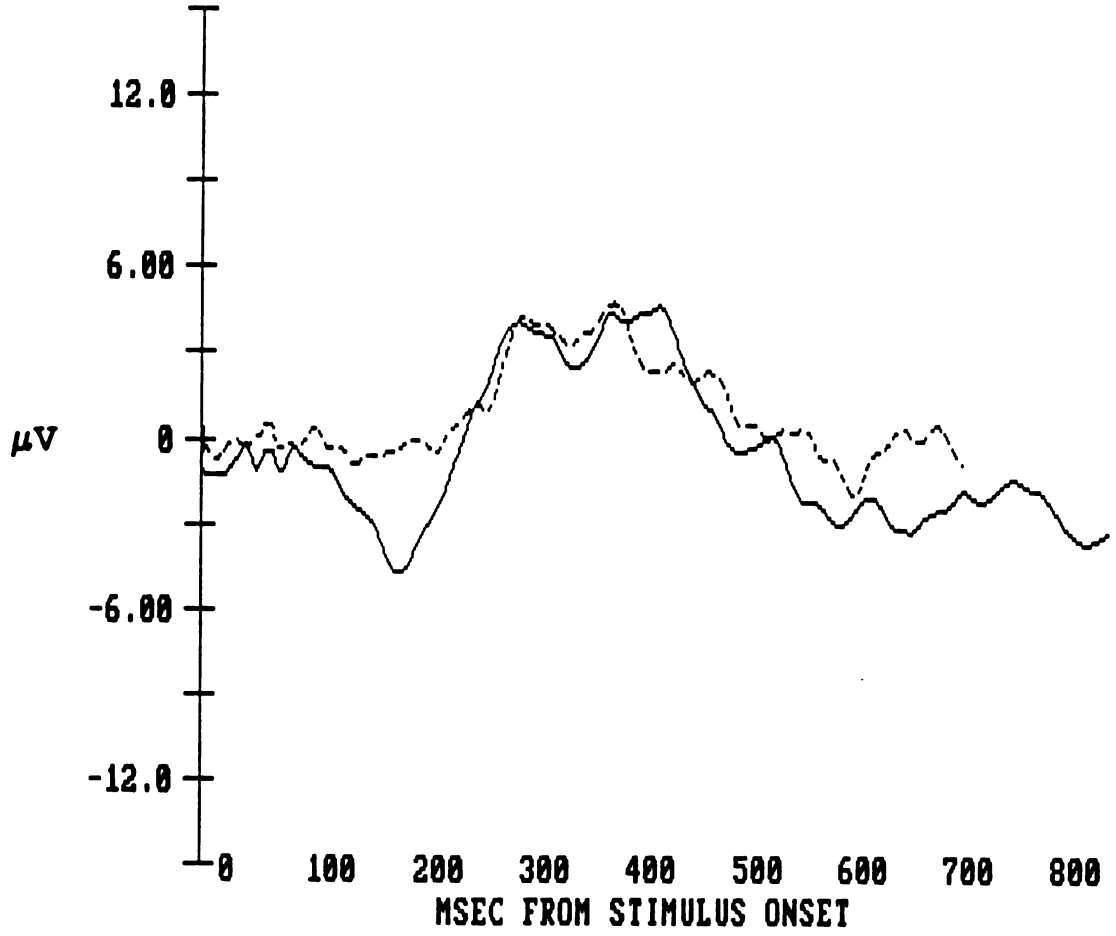


FIGURE 45. Solid = Hearing-impaired Response to "POT" (Protocol 1, Condition 2 - Attending)
Dashed = Hearing-impaired Response to "POT" (Protocol 4, Condition 9 - Reading)

	Normal-hearing	Hearing-impaired	p <
N1 lat.	99.0 ± 17.1	146.0 ± 39.5	.0075
N1 amp.	-7.0 ± 3.8	-2.6 ± 1.7	.05
P2 lat.	242.3 ± 37.9	274.5 ± 47.3	ns
P2 amp.	6.2 ± 4.1	4.5 ± 3.6	ns

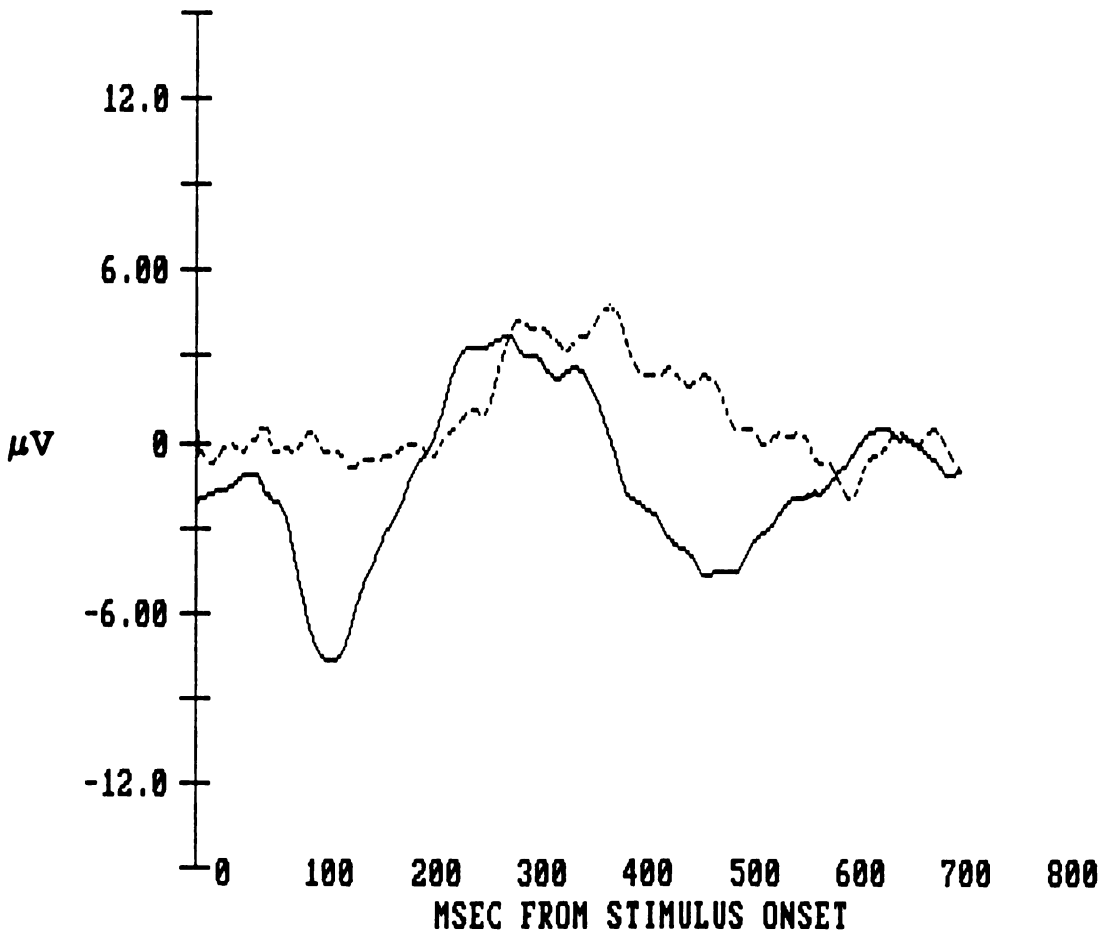


FIGURE 46. Solid = Normal Response to "POT"
Dashed = Hearing-impaired Response to "POT"
(Protocol 4)

Hearing-impaired Subjects, Condition 8: Figure 40 is a topographic map of the hearing-impaired response to "PA" (250 msec duration). Compared to the normal map, distributions of N1, P2, and P3 components are more frontal.

Figure 41 is a comparison of normal and hearing-impaired responses to "PA" recorded from Cz. The only significant difference is increased N1 latency in the hearing-impaired response.

Normal-hearing Subjects, Condition 9: Figure 42 is a topographic map of the normal response to the word "POT" (500 msec duration). Compared to the normal response to "POT" in Protocol 1, distributions of N1, P2, and P3 components are similar. Comparing Figure 42 to maps of normal responses to "AH" and "PA," distributions of N1 and P2 components are similar. The most striking difference among these maps is the presence of a parietal P3 component in normal responses to consonant-vowels ("PA" and "POT") and the absence of P3 in response to the vowel "AH."

Figure 43 is a comparison of normal responses to "POT" in Protocols 1 and 4 (recorded from Cz). There are no significant differences in component latencies. There are slight (but not significant) reductions of N1, P2, and P3 amplitudes during Protocol 4 (when subjects were reading).

Hearing-impaired Subjects, Condition 9: Figure 44 is a topographic map of hearing-impaired responses to "POT." Compared to the normal map, the distributions of N1 and P3 components are more frontal.

Figure 45 is a comparison of hearing-impaired responses to "POT" in Protocols 1

and 4. Component latencies are not significantly different. Even though N1 amplitude appears smaller in Protocol 4, the difference is not statistically significant.

Figure 46 is a comparison of normal and hearing-impaired responses to "POT" in Protocol 4 (recorded from Cz). Hearing-impaired N1 latency is significantly greater; as is normal N1 amplitude. Other components are not significantly different.

In summary, the results from Protocol 4 are as follows:

1) Normal N1 latency increases; N1 and P2 amplitudes decrease in responses to 250 Hz tones of increasing duration. Hearing-impaired N1 latency also increased in response to longer duration 250 Hz tones. AEP component amplitude reductions were seen in responses to tones from both groups when the task changed from attentive listening (Protocol 1) to reading (Protocol 4).

2) Hearing-impaired N1 and P2 latencies were significantly greater than normals in response to noise bursts after pauses of both 50 and 100 msec. These delays in temporal processing probably result from a combination of a) the inability of profoundly hearing-impaired subjects to perceive the high frequency components of white noise; b) differences in subjective stimulus intensity; and c) differences in abilities to process gaps in complex acoustic stimuli.

3) Normal subjects produced P3 components in response to consonant-vowels ("PA" or "POT"), but not in response to the vowel "AH." This might be a reflection of enhanced attention to consonant-vowel sounds or it could be an indicator of differential neural processing of consonant-vowel vs. vowel sounds.

4) Compared to normals, hearing-impaired subjects had delayed N1 latencies to all

speech sounds ("AH," "PA," and "POT"). Again, this could be a reflection of a) lower subjective stimulus intensities; b) differences in ability to perceive and process the high frequency components of speech; or c) differences in the organization of central auditory pathways.

5) Topographic distributions of AEP components in response to speech sounds are different in normal vs. hearing-impaired maps. This supports the hypothesis that differences in organization of central auditory pathways exist between the groups. These differences are most evident in responses to complex acoustic stimuli.

PROTOCOL 5 - P3 "Oddball" Paradigm Using Low Frequency Tones

	A random presentation of the following:
Condition 1:	80 each 100 msec 125 Hz tones
Condition 2:	30 each 100 msec 250 Hz tones ("rare")
Condition 3:	30 each 100 msec 125 Hz tones ("pre-rare")
Condition 4:	30 each 100 msec 125 Hz tones ("post-rare")

Normal-hearing Subjects, Condition 2: Figure 47 contains the normal response to at least 30 repetitions of the 250 Hz "rare" tone. Mean latency and amplitude values are given on the figure for N1 and P3 components. Figure 48 is the topographical map of these data. Note the presence of a P3 and the absence of a clear P2 component. Comparing N1 components in this condition with those in Protocol 4, Condition 2 (also 100 msec duration, 250 Hz tones), there are no significant differences in N1 latency, amplitude, or topography between the conditions.

Normal-hearing Subjects, Condition 3: Figure 47 also contains the normal response to at least 30 repetitions of the 125 Hz "common" tone which preceded each of the "rare" tones in the stimulus sequence. Figure 49 is the topographical map of these data. Note the absence of the P3 and the presence of a clear P2 component. N1 latencies, amplitudes, and topographies are not significantly different between conditions 2 and 3 in this protocol.

Hearing-impaired Subjects, Condition 2: Figure 50 contains the hearing-impaired response to the 250 Hz "rare" tone. Compared to the normal response, hearing-impaired N1 latency is greater ($p < .05$); however, P3 latencies are not different. Normal N1 amplitude is greater ($p < .02$); P3 amplitudes are not significantly different. Figure 51 is the topographical representation of these data. Compared to

	250 Hz "Rare"	125 Hz "Common"
N1 lat.	107.0 ± 13.4	108.4 ± 8.8
N1 amp.	-11.4 ± 5.4	-11.0 ± 4.4
P2 lat.	-	195.9 ± 22.9
P2 amp.	-	8.9 ± 4.2
P3 lat.	331.3 ± 25.7	-
P3 amp.	11.3 ± 8.8	-

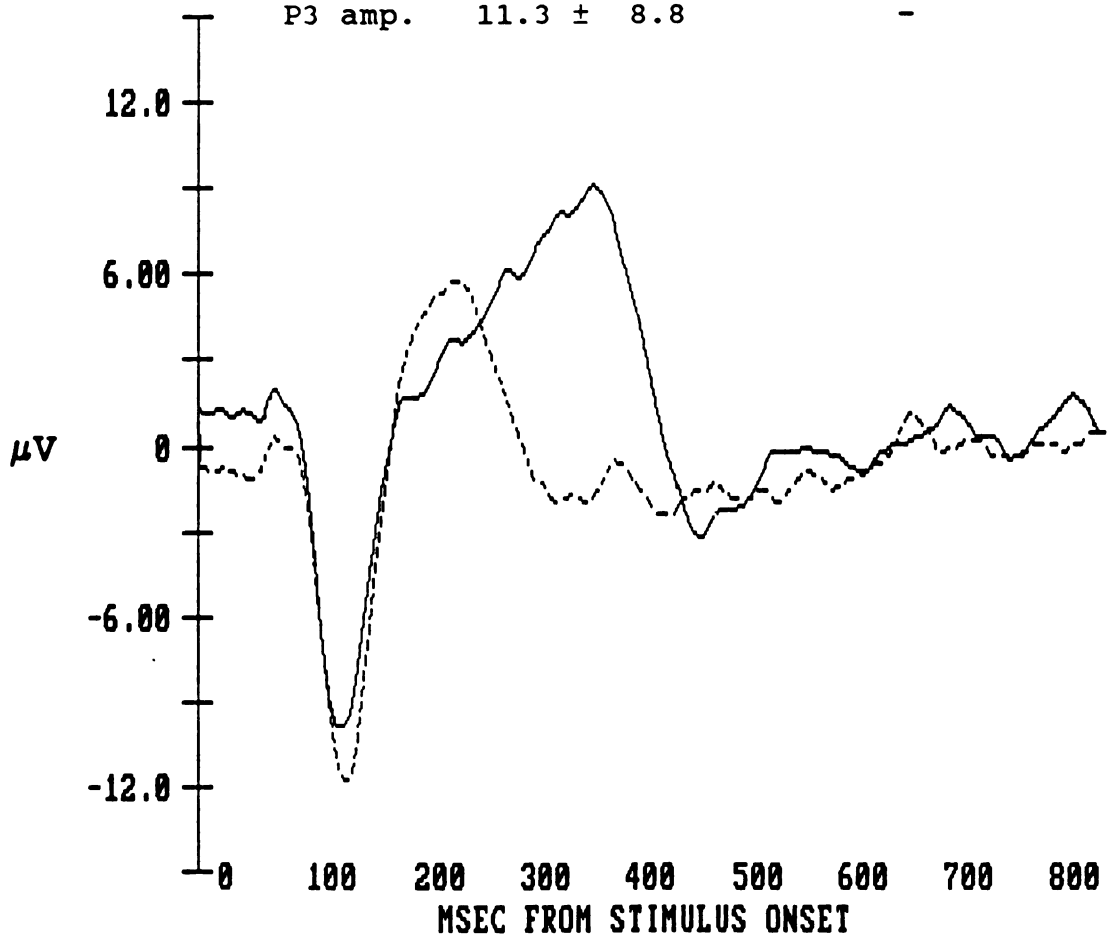
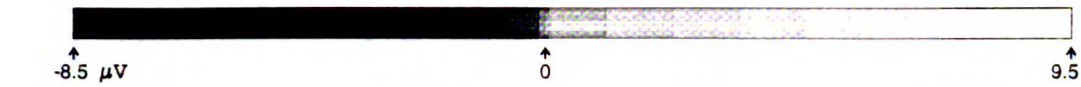
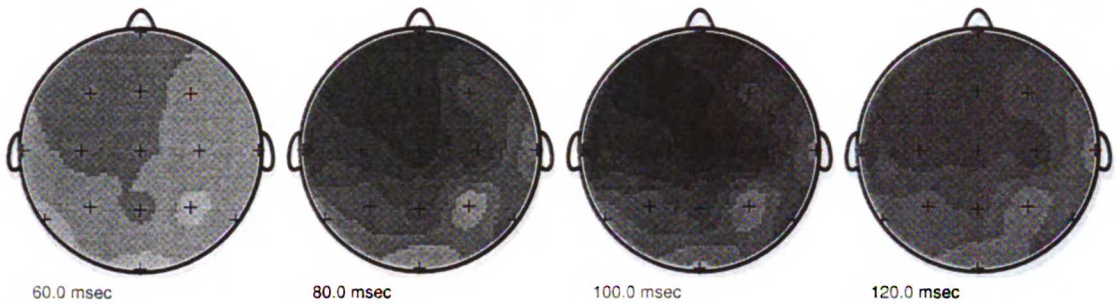
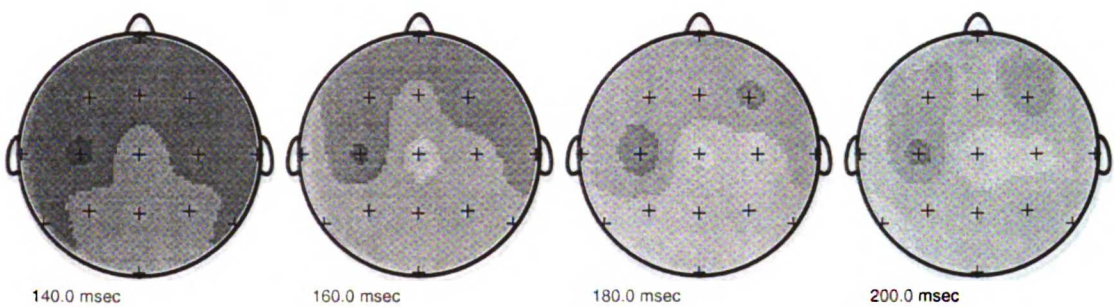


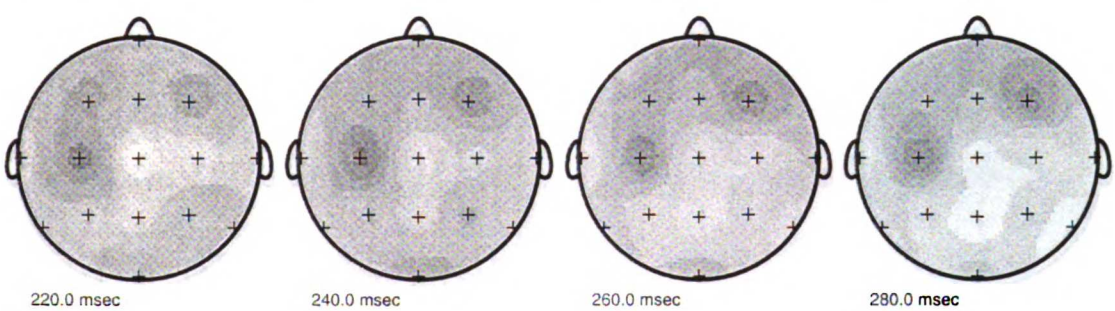
FIGURE 47. Solid = Normal Response to 250 Hz (Rare) Tones
Dashed = Normal Response to 125 Hz (Common) Tones

FIGURE 48. Normal Response to "Rare" (250 Hz) Tones (Protocol 5)









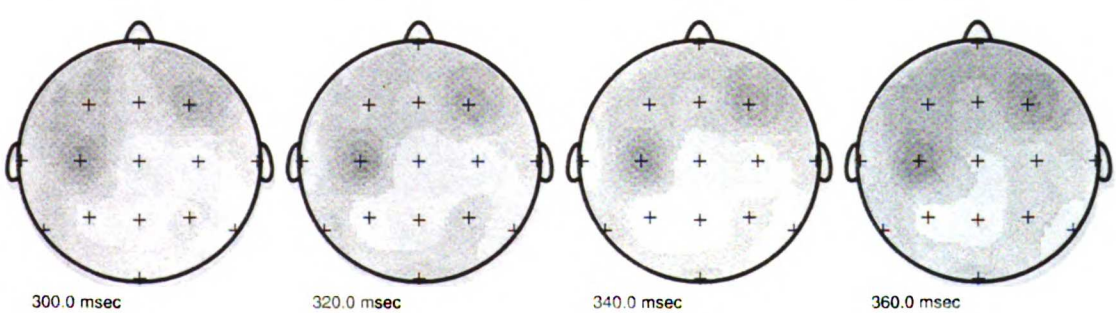
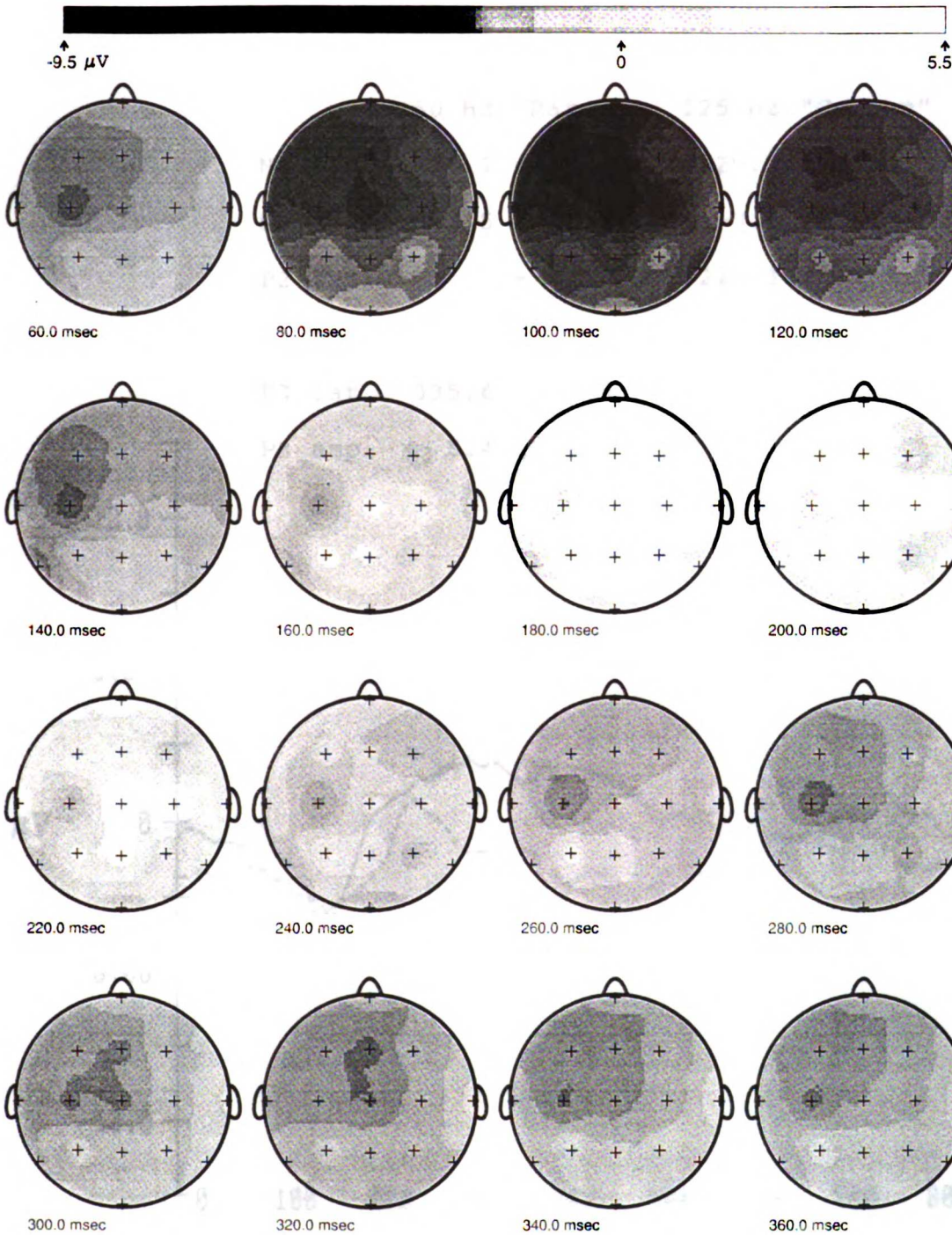


FIGURE 49. Normal Response to "Common" (125 Hz) Tones (Protocol 5)



	250 Hz "Rare"	125 Hz "Common"
N1 lat.	134.1 ± 35.6	129.7 ± 23.4
N1 amp.	-3.5 ± 1.7	-3.8 ± 1.0
P2 lat.	-	220.1 ± 28.3
P2 amp.	-	3.4 ± 2.9
P3 lat.	335.6 ± 60.9	-
P3 amp.	5.4 ± 2.6	-

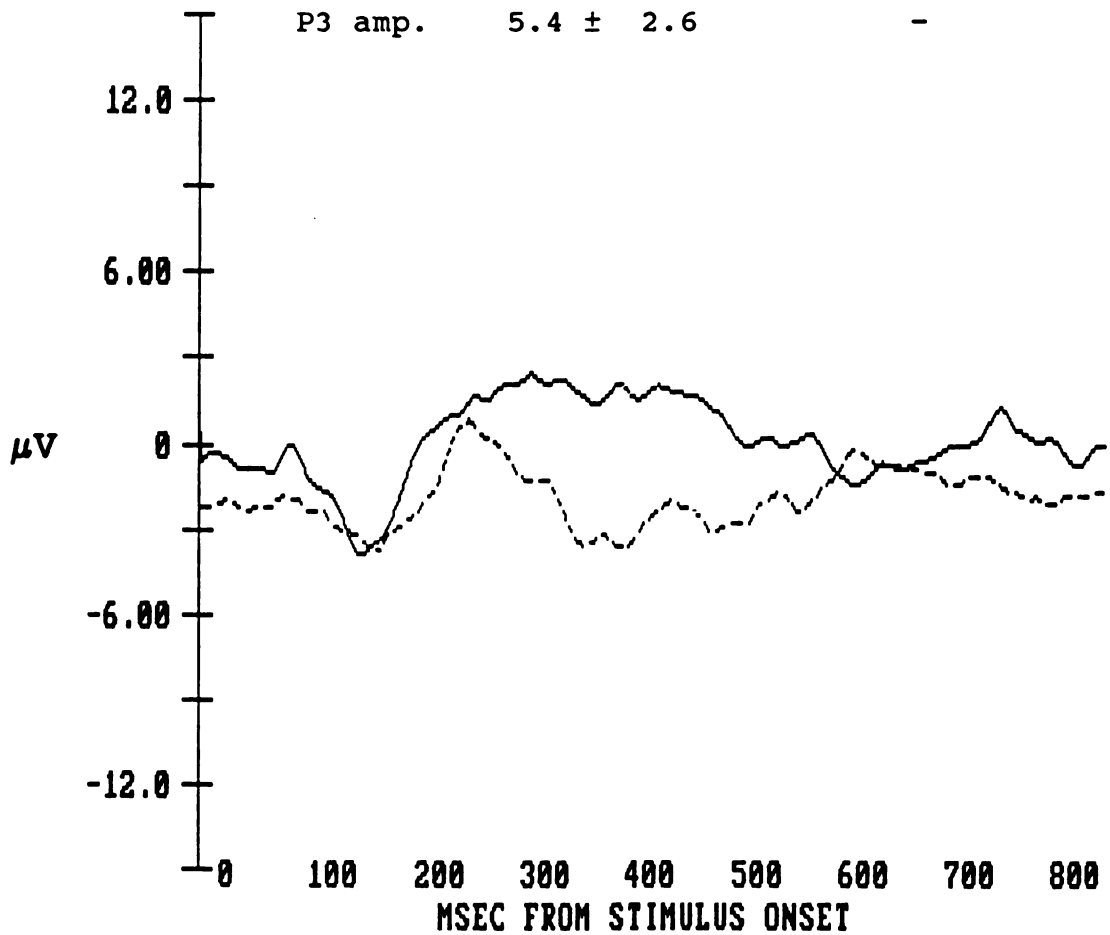


FIGURE 50. Solid = Hearing-impaired Response to 250 Hz (Rare) Tones
Dashed = Hearing-impaired Response to 125 Hz (Common) Tones

FIGURE 51. Hearing-impaired Response to "Rare" Tones (Protocol 5) 84

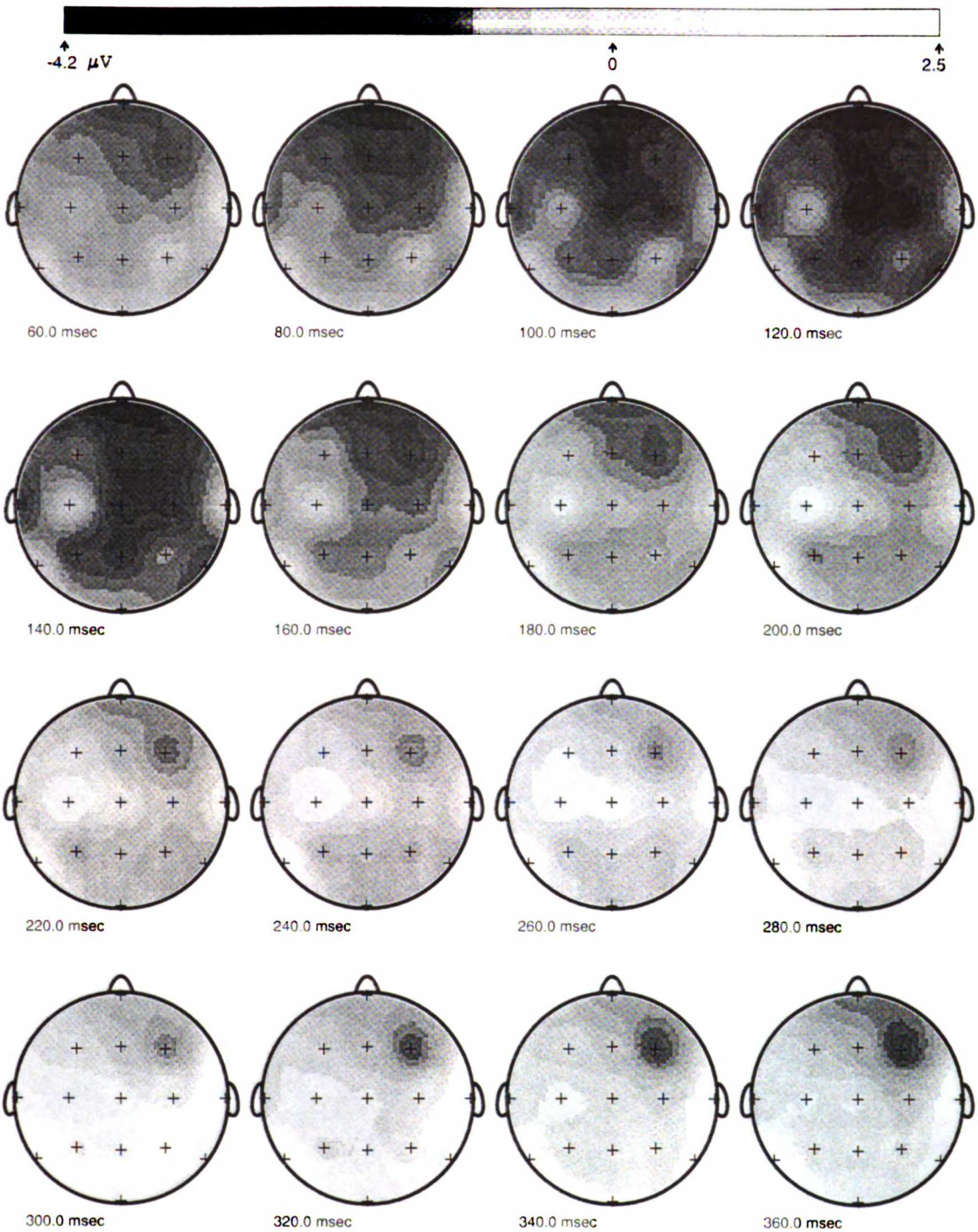
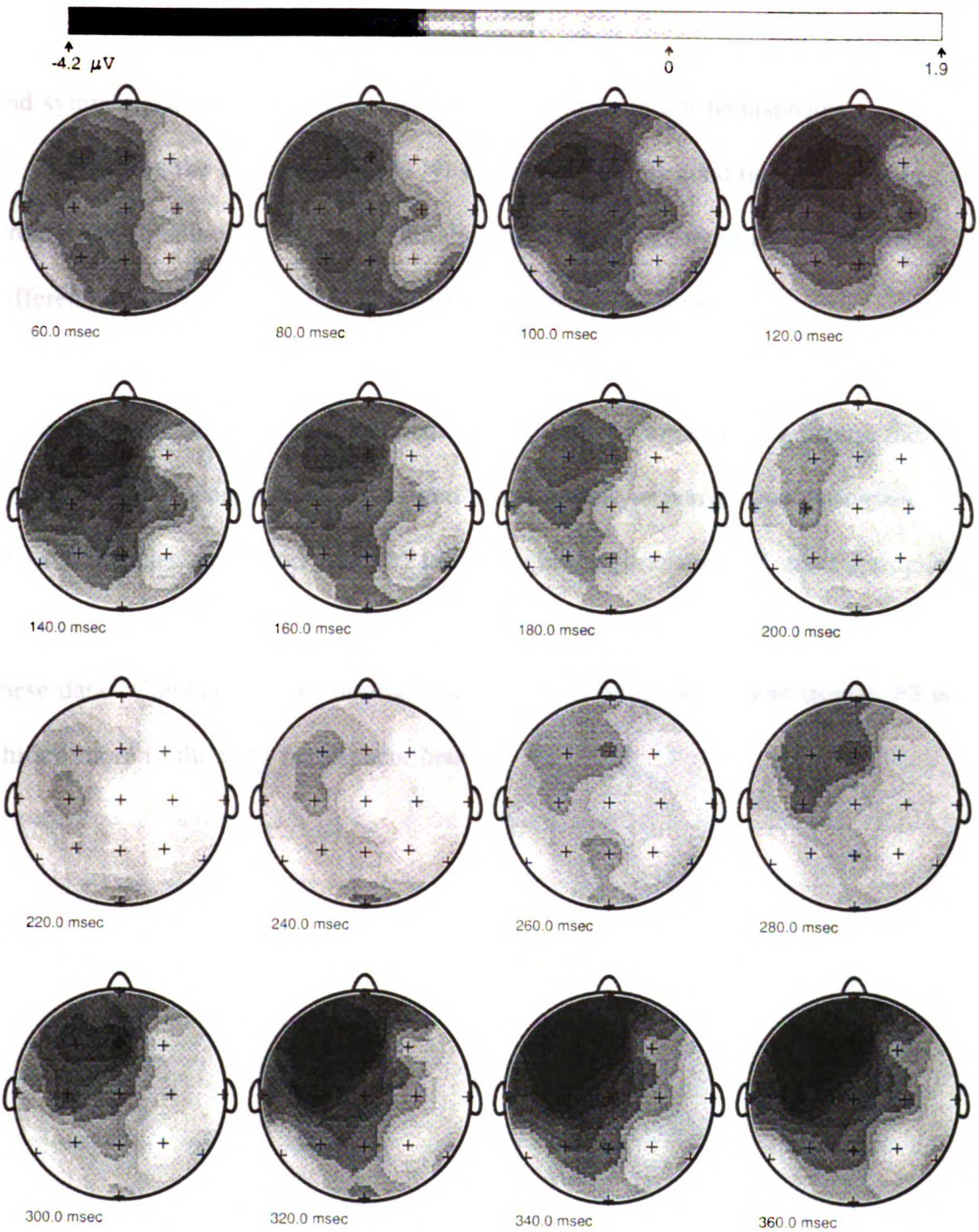


FIGURE 52. Hearing-impaired Response to "Common" Tones (Protocol 5) 85



the normal topography, the hearing-impaired N1 distribution is somewhat more frontal and symmetrical; the P3 is more asymmetrical (shifted to left hemisphere).

Comparing the hearing-impaired N1 component in this condition with that in Protocol 4, Condition 2 (100 msec duration, 250 Hz tones), there are no significant differences in N1 latency or amplitude between the conditions.

Hearing-impaired Subjects, Condition 3: Figure 50 contains the hearing-impaired response to the 125 Hz "pre-rare" tone. Again, note the absence of a P3 and the presence of a P2 component. Compared to the normal response, hearing-impaired N1 and P2 latencies are greater ($p < .05$ for both); hearing-impaired N1 and P2 amplitudes are smaller ($p < .02$ and $p < .05$ respectively). Figure 52 is the topographic map of these data. Compared to the normal map, the N1 distribution is more frontal; P2 is shifted more to the right hemisphere here.

PROTOCOL 6 - Responses To First Names

A random presentation
of the following:
Condition 1: 80 first names other than the subject's first name (matched with subject's first name for gender, # of syllables, intensity, and duration)
Condition 2: 30 repetitions of the subject's first name

Normal-hearing Subjects, Condition 1: Figure 53 contains the normal response to a variety of 80 first names (other than the subject's first name) matched for gender, number of syllables, duration and intensity with the subject's first name in Condition 2. Figure 54 is the topographic map of these data. Note the distributions of the N1 and P2 components, and the absence of a P3.

Normal-hearing Subjects, Condition 2: Figure 53 also contains the normal response to at least 30 repetitions of the subject's first name. Note the large P3 component not present in Condition 1. Figure 55 is the topographic map of these data. Comparing this map with Figure 54, the N1 here has a more frontal, symmetrical distribution. Comparing this map with Figure 48 (normal response to rare tones), the P3 here has a more parietal (less central) distribution.

Hearing-impaired Subjects, Condition 1: Figure 56 contains the hearing-impaired response to 80 first names other than the subject's first name. Compared to the normal response, N1 and P2 latencies are not significantly different. Hearing-impaired N1 amplitude is smaller ($p < .015$); P2 amplitudes are not significantly different. Figure 57 is the topographic map of these data. Compared to Figure 54 (normal response to the same condition), the N1 and P2 components have more

	Other Names	Own Name
N1 lat.	118.8 ± 16.7	139.2 ± 22.7
N1 amp.	-5.7 ± 2.0	-6.8 ± 2.9
P2 lat.	229.2 ± 35.6	226.8 ± 29.2
P2 amp.	2.1 ± 2.5	4.4 ± 5.0
P3 lat.	-	375.5 ± 38.5
P3 amp.	-	9.5 ± 6.2

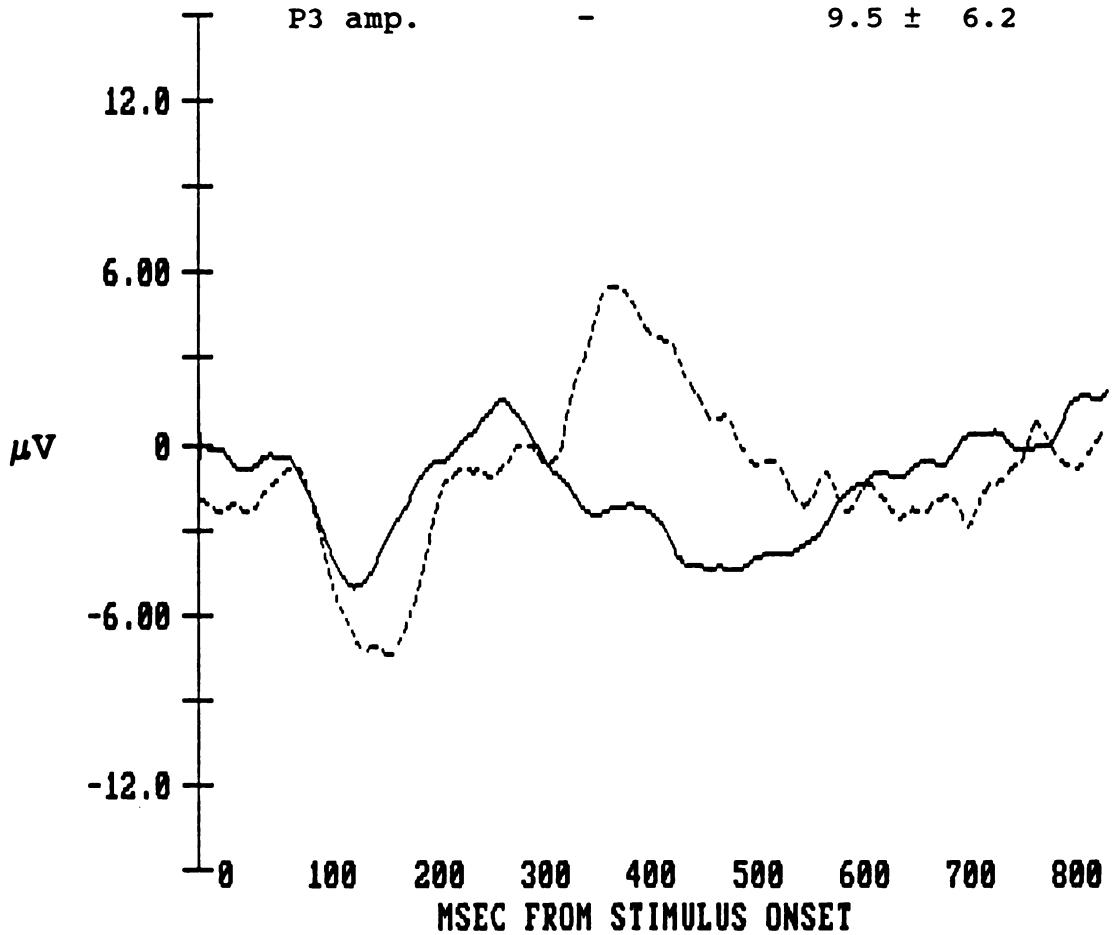


FIGURE 53. Solid = Normal Response to Other First Names
Dashed = Normal Response to Subjects' Own First Name
(Protocol 6)

FIGURE 54. Normal Response to Other Names (Protocol 6)

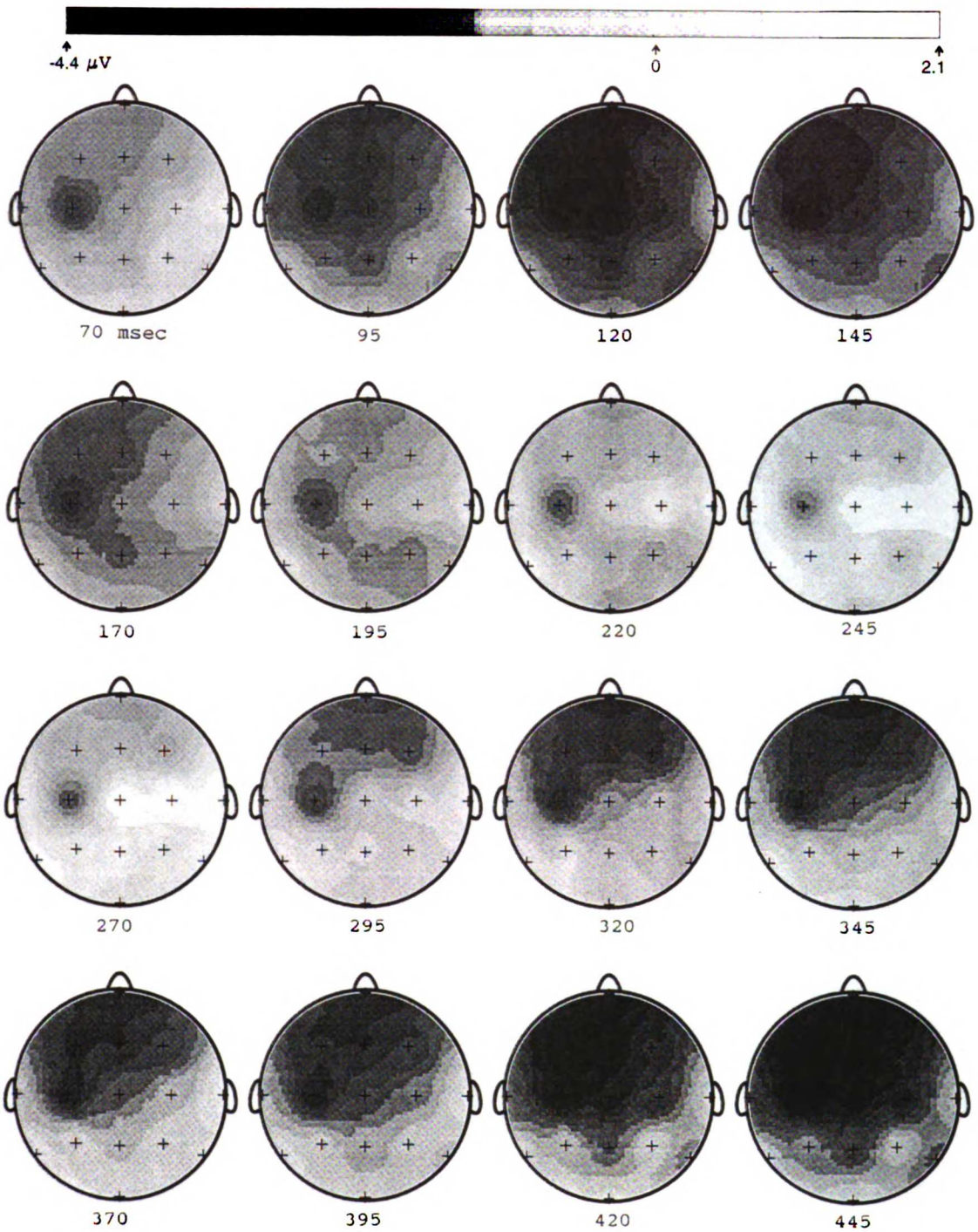
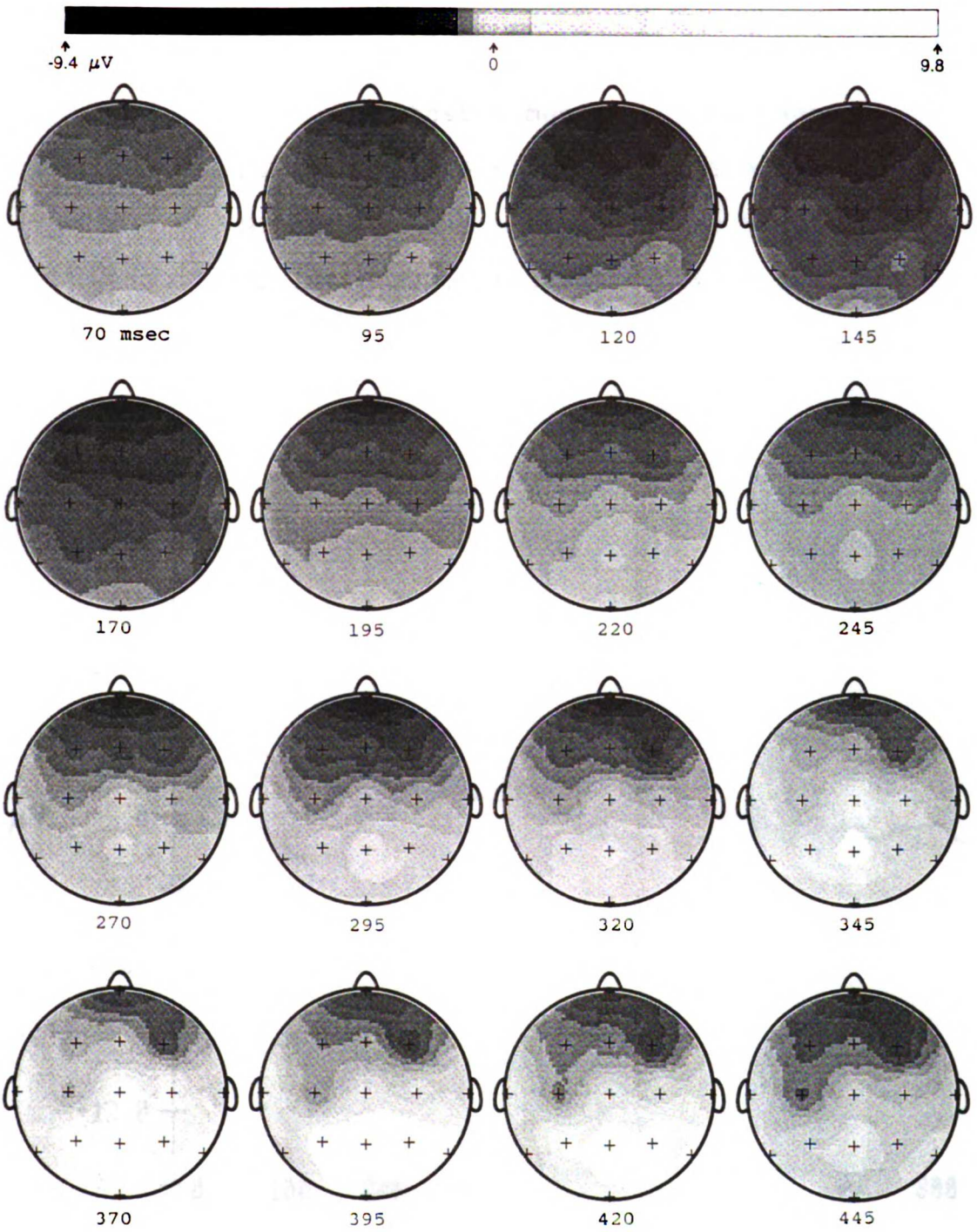


FIGURE 55. Normal Response to Subjects' Own Name (Protocol 6)



	Other Names	Own Name
N1 lat.	139.5 ± 40.3	151.8 ± 41.5
N1 amp.	-1.5 ± 1.1	-2.8 ± 1.9
P2 lat.	222.7 ± 32.9	257.5 ± 71.5
P2 amp.	3.0 ± 1.7	2.7 ± 1.9

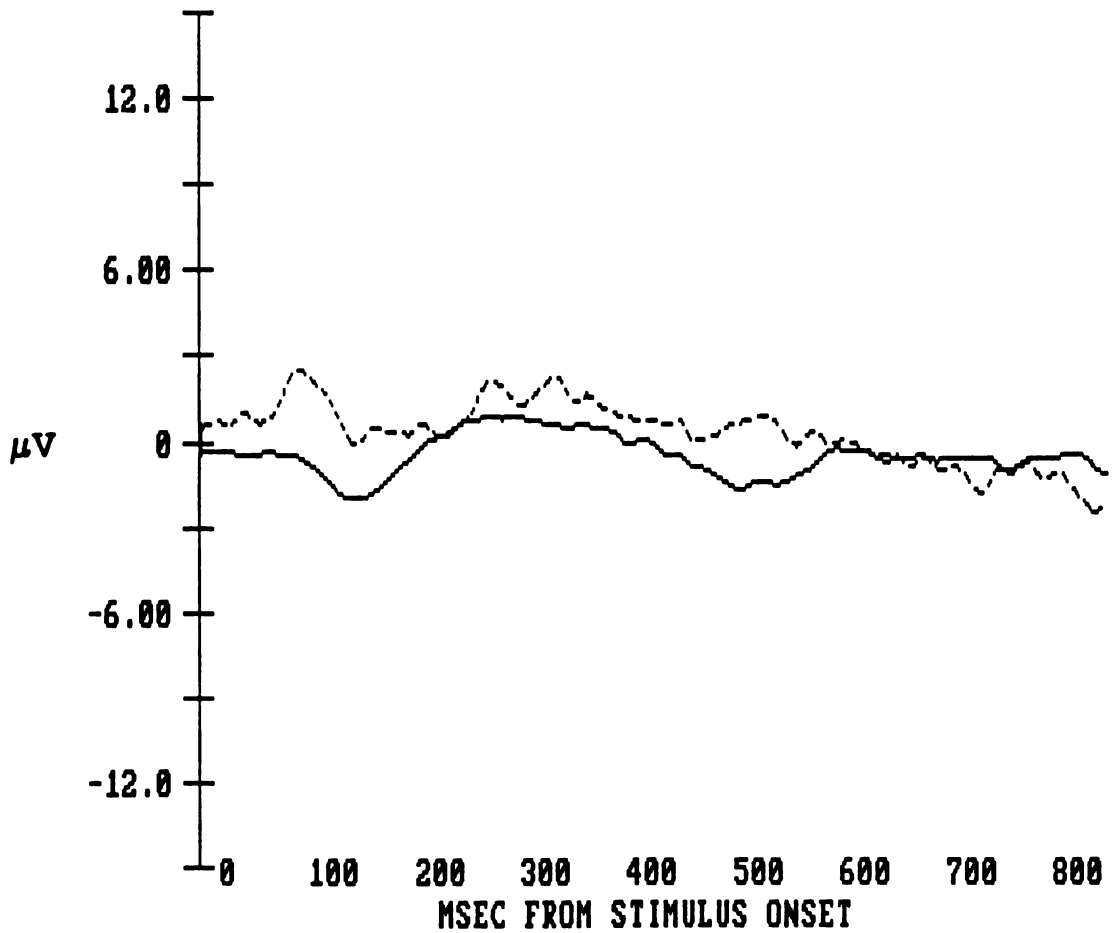


FIGURE 56. Solid = Hearing-impaired Response to Other First Names
Dashed = Hearing-impaired Response to Subjects' Own First Name
(Protocol 6)

FIGURE 57. Hearing-impaired Response to Other Names (Protocol 6)

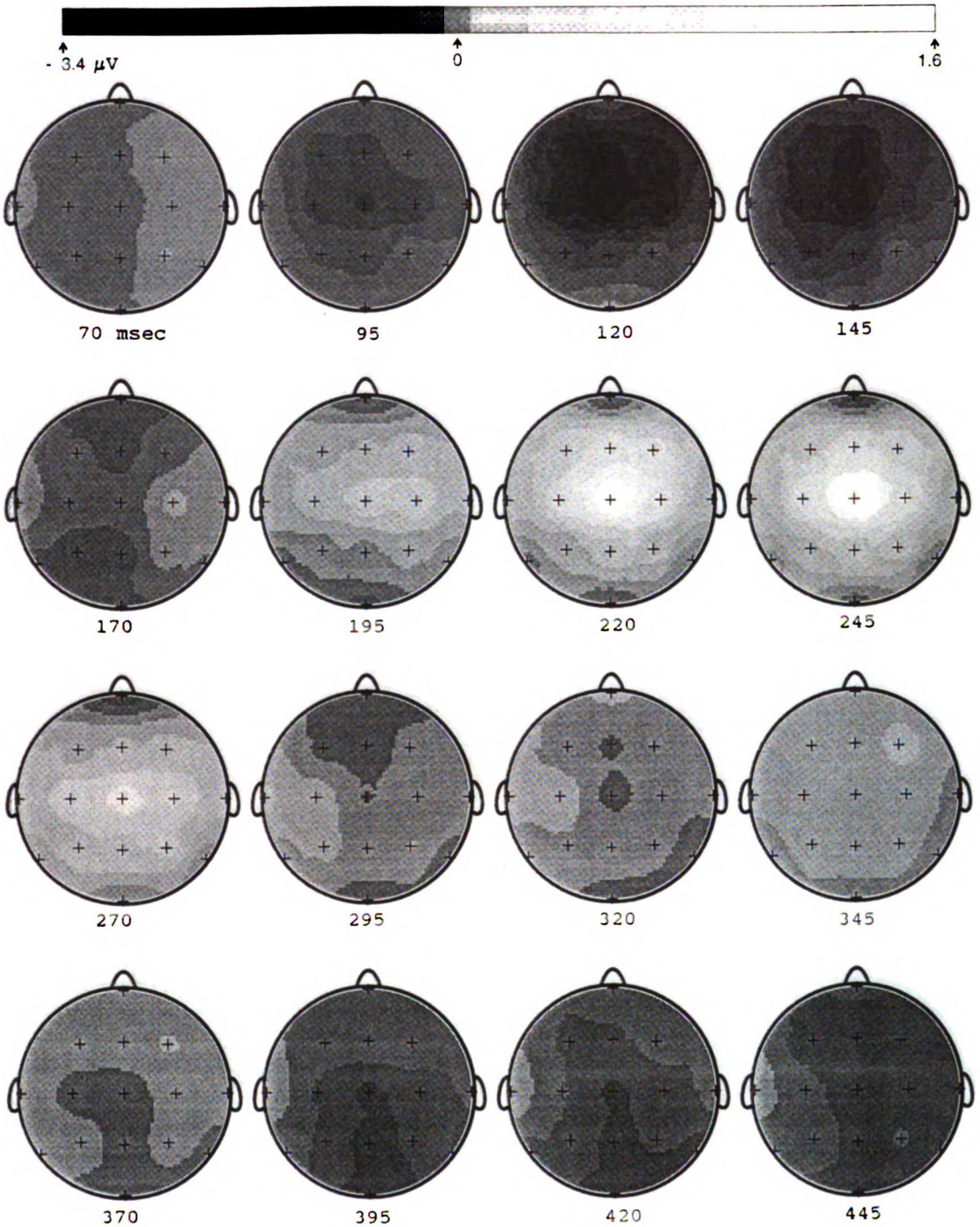
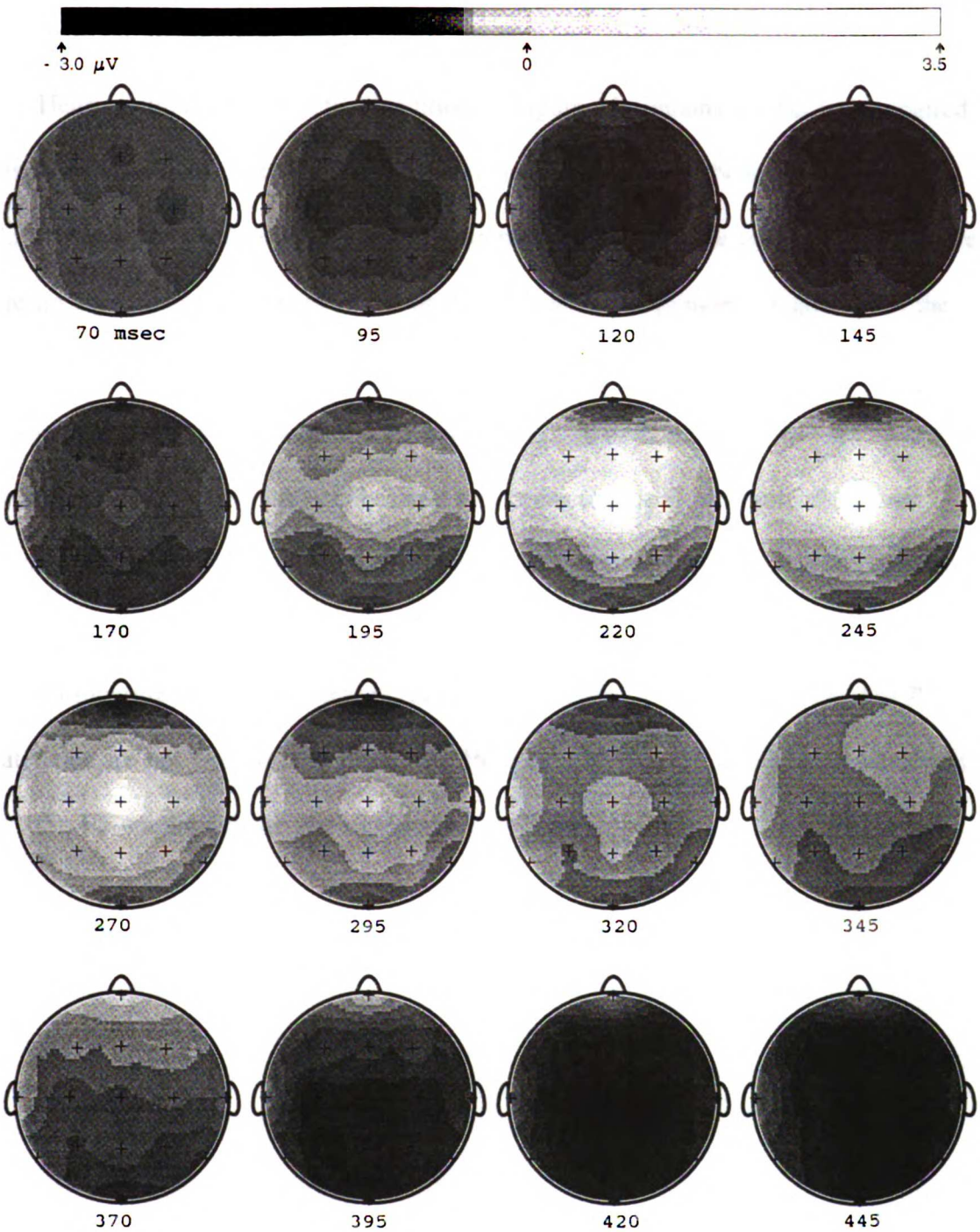


FIGURE 58. Hearing-impaired Response to Subjects' Own Name



symmetrical distributions here.

Hearing-impaired Subjects, Condition 2: Figure 56 contains the hearing-impaired response to at least 30 repetitions of the subject's first name. Note the absence of a P3 component. Comparing N1 and P2 components between the two conditions, there are no significant differences in N1 or P2 latencies or amplitudes. Figure 58 is the topographic map of these data. Note the symmetrical distributions of the N1 and P2 components and the absence of a P3 component. A P3 component was not elicited from hearing-impaired subjects because they were unable to recognize their own names spoken on the tape. In fact, these subjects were not able to distinguish between speech sounds of any kind.

Comparing normal and hearing-impaired responses in Condition 2, N1 and P2 latencies are not significantly different. Hearing-impaired N1 amplitude is smaller ($p < .05$); P2 amplitudes are not significantly different.

PROTOCOL 7 - Normal-hearing Subjects Only

A random presentation
of the following:

Condition 1: 80 first names other than the subject's first name (matched with subject's first name for gender, # of syllables, intensity, and duration)

Condition 2: 30 repetitions of a first name other than the subject's first name (matched with subject's first name for gender, # of syllables, duration and intensity; placed in the same positions within the stimulus sequence as the names in Protocol 6, Condition 2)

Condition 1: Figure 59 contains the normal response to 80 different first names (other than the subject's first name) matched for gender, number of syllables, duration and intensity with the names in Condition 2. Compared to the normal responses in Protocol 6, Condition 1, there are no significant differences in N1 or P2 latencies or amplitudes.

Condition 2: Figure 59 also contains the normal response to at least 30 repetitions of a first name other than the subject's first name (matched for gender, number of syllables, duration and intensity, and placed in the same locations within the stimulus sequence as the subject's name in Protocol 6, Condition 2). Note the absence of a P3 component. Compared to the responses in Condition 1, there are no significant differences in N1 or P2 latencies or amplitudes. Comparing responses in this condition with those in Protocol 6, Condition 2, there are no significant differences in N1 or P2 latencies or amplitudes.

The absence of a P3 here indicates that <u>any</u> first name repeated 30 out of 110

times in a stimulus sequence will not necessarily elicit the component from normal subjects. P3 components were recorded from normal subjects in these protocols only in response to their own first names.

	Other Names	One Name
N1 lat.	118.5 ± 31.0	110.8 ± 26.8
N1 amp.	-5.5 ± 1.3	-5.6 ± 2.8
P2 lat.	214.3 ± 36.2	210.0 ± 23.7
P2 amp.	4.0 ± 2.5	5.0 ± 2.5

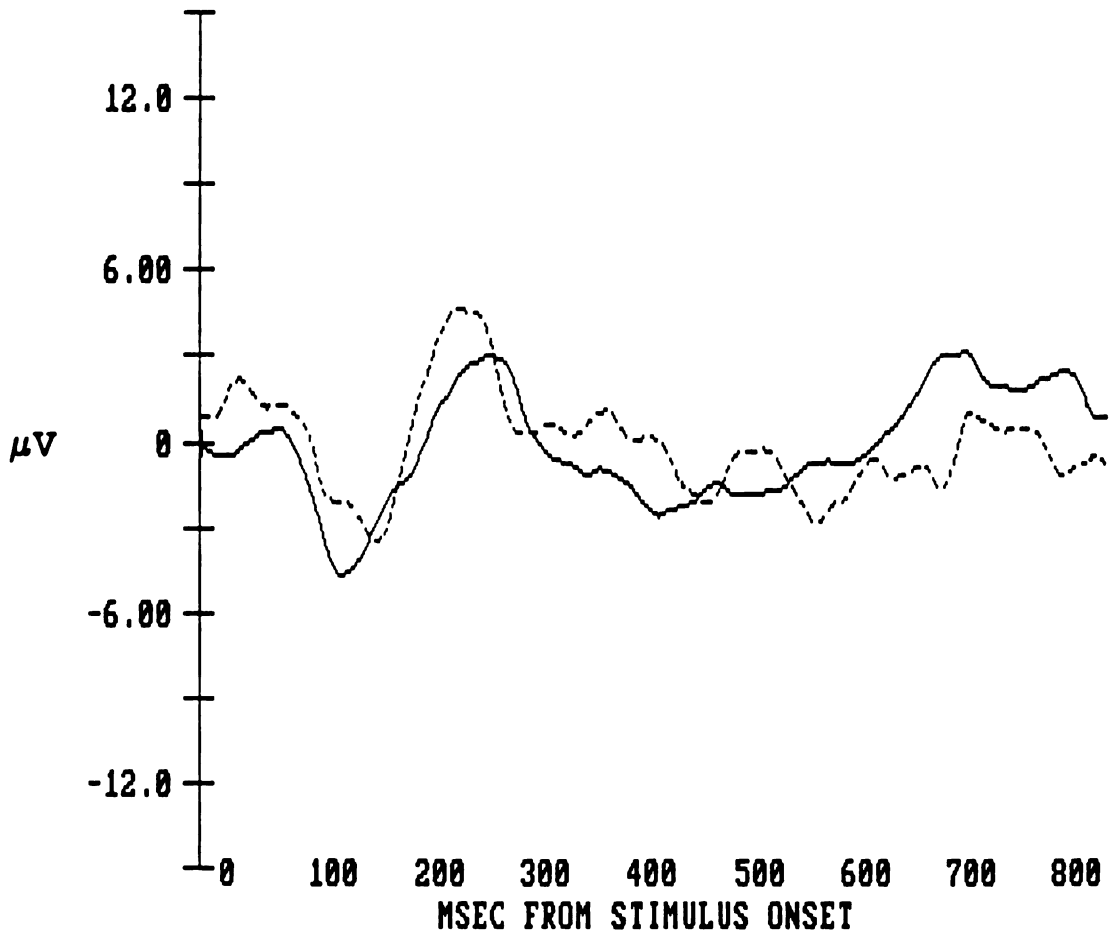


FIGURE 59. Solid = Normal Response to Other First Names
Dashed = Normal Response to A First Name Other Than the Subjects' First Name (Protocol 7)

DISCUSSION

EFFECTS OF STIMULUS DURATION

The effects of stimulus duration on exogenous long latency AEPs were investigated by Onishi & Davis (1968). They reported that latencies and amplitudes of N1 and P2 components recorded from 5 normal-hearing subjects were stable in response to 1000 Hz tones ranging from 0 to 300 msec in plateau duration with rise times of 50 msec or less. N1 latencies did increase with increases in rise times above 50 msec. Polich (1989a) recorded long latency AEPs from 24 normal-hearing subjects in response to 20, 50, and 80 msec duration 1000 and 2000 Hz tones. He reported that N1 and P2 latencies and amplitudes were stable for all three durations. Putnam & Roth (1990) recorded AEPs from 16 normal-hearing students in response to 105 dBA white noise bursts ranging in total duration from 3 to 90 msec. Again, N1 and P2 latencies and amplitudes were not statistically different in response to all durations of stimuli. However, Woods (1995) reported that N1 amplitude increased with duration (from 8 to 24 to 72 msec) of 4000 Hz tones.

According to Lamb & Graham (1967), the latency of the N1 component increased and its amplitude decreased as 1000 Hz tone stimulus rise times increased from 10 to 95 msec. Rose & Malone (1965) and Davis & Zerlin (1966) also reported increases in N1 latency with increases in stimulus rise times. Skinner & Jones (1968) suggested that rise and fall times of auditory evoked response stimuli should be 10 msec or less.

Using magnetoencephalography, Kaukoranta et al. (1989) investigated the long latency responses of 9 normal-hearing subjects to 1000 Hz tones which varied in total duration from 50 to 100 msec. The N100m component (which corresponds to the N1

in electroencephalography) was not affected by changes in stimulus duration. Joutsiniemi et al. (1989) recorded magnetic-evoked responses from 8 normal-hearing subjects to noise bursts and to pauses in continuous noise which varied in duration from 5 to 160 msec. Noise bursts were used rather than tones to eliminate spectral differences between stimuli of different durations. Pauses were used to investigate the perception of silent intervals which contribute to word recognition. N100m amplitude increased slightly with increasing duration of noise bursts and pauses from 5 to 40 msec. N100m latency decreased when stimulus durations increased from 5 to 40 msec. N100m amplitudes and latencies were stable for 40, 80, and 160 msec duration noise bursts and pauses.

Following is a discussion of the effects of stimulus duration on exogenous long latency components observed in the present study. Tonal and noise stimuli used in this study had rise and fall times of 0 msec.

TONAL STIMULI

Low frequency tones ranging from 50 to 500 msec in duration were used in this study. Within the normal-hearing group, as the duration of 250 Hz tones increased from 100 msec to 500 msec in Protocol 4, N1 latencies increased while N1 and P2 amplitudes decreased. This result suggests that habituation occurred during this passive listening protocol. Habituation (as described by Weber, 1970; Picton et al., 1976; and Woods & Elmasian, 1986) is a reduction of N1/P2 amplitudes in response to long duration (300 msec or longer) stimuli repeated during recording sessions.

Within the hearing-impaired group, N1 and P2 latencies did not change significantly in responses to increasing durations of tones. Also, N1 and P2 amplitudes from hearing-impaired subjects did not change significantly in their responses to 500 msec duration tones. This result suggests that habituation did not occur within this group. A possible explanation for this is the hearing-impaired subjects' lack of experience with high intensity sounds such as those used in this study. This usual absence of activation of their auditory pathways is probably responsible for the lack of thalamic or cortical mechanisms that are necessary for habituation to occur.

Comparing normal-hearing and hearing-impaired responses to 250 Hz tones of equal duration in Protocols 1 and 4: N1, P2 and P3 latencies are not significantly different between the subject populations. This suggests that the time required by central auditory pathways to process tones within the range of hearing is not different between the groups. However, hearing-impaired N1 amplitudes were smaller than normals for equal duration tonal stimuli in Protocols 1 and 4. This is a reflection of lower subjective stimulus intensities delivered to the hearing-impaired group during these protocols. N1 appears to be a good indicator of relative stimulus intensity. However, P2 amplitudes were not significantly different between hearing-impaired and normal-hearing subjects which suggests that this component is not so affected by subjective stimulus intensity.

ONSET RESPONSES

Onset responses to continuous white noise after a pause in continuous noise provide examples of differences in temporal processing of complex stimuli between the experimental groups. Regardless of the duration of the pause (50 or 100 msec), onset N1 latencies from hearing-impaired subjects are greater than those from normal subjects. These results indicate differences in the processing of gaps in complex stimuli, simulating gaps in speech. Based on behavioral studies of hearing-impaired subjects, Boothroyd (1990) concludes "The profoundly deaf have poor frequency and time resolution accounting, in part, for their poor speech discrimination." Results from the present study indicate that AEPs are useful for assessing temporal integration. This information could be used in the design of training programs to improve temporal resolution and perhaps speech discrimination in hearing-impaired humans.

Behavioral studies by Florentine & Buus (1983) indicate that gap detection relies more on high-frequency cues than on low-frequency input. AEP studies by Woods et al. (1993) suggest that short duration (10 msec) high frequency (4000 Hz) stimuli are processed faster than equal duration low frequency (250 Hz) stimuli. Lack of ability to detect and process high frequency acoustic stimuli is probably responsible for the delays in onset responses exhibited by hearing-impaired subjects in the present study.

SPEECH STIMULI

Speech stimuli used in this study were components of the word "POT" ranging in duration from 250 to 500 msec. AEPs from normal subjects in this study exhibit particular characteristics in responses to vowels ("AH") when compared to responses to consonant-vowels ("PA" or "POT"). P2 latencies to the vowel "AH" are shorter than P2 latencies to "PA" or "POT." Delays in processing of consonant-vowels might be caused by delays in the onset of maximum acoustic energy of these stimuli (Woods & Elmasian, 1986) compared to the more immediate onset of vowels, tones, or noise. The consonant-vowel (CV) "PA" was chosen as the speech stimulus in this study because it is a plosive with a short duration consonant (approximately 10 msec). In a study of AEP responses to ten different CVs, Lawson & Gaillard (1981) reported that "PA" elicited the shortest latency N1 (117.5 msec) and P2 (195.4 msec).

Comparing the topography of normal AEP components in Protocol 4, the distribution of N1 is more left-frontal in response to the vowel "AH;" more central and symmetrical in response to CVs "PA" or "POT." This raises the possibility that different areas of cortex are activated during the normal processing of vowels vs. CVs used in this study.

Hearing-impaired AEPs do not exhibit similar variability in responses to vowels, consonant-vowels, and words. This is consistent with the observation that profoundly hearing-impaired people are unable to discriminate between different speech sounds. This occurs for two reasons: 1) people with high frequency hearing loss are unable to detect and process the high frequency components of speech; 2) because of this lack

of experience with high frequency stimuli, the organization of their auditory cortex is different from that of people with lower thresholds for these stimuli. It is likely that the auditory cortices of people with profound high frequency hearing loss have greater than normal areas devoted to the representation of lower frequencies (see Schwaber, Garraghty & Kaas, 1993 for an animal model).

Additional evidence for differences in organization of cerebral cortex comes from comparisons of hearing-impaired and normal AEP topography in response to speech sounds. For example, hearing-impaired N1 components have a more frontal distribution than normals in response to all speech stimuli. As stated earlier, N1 is generated at least in part by auditory cortex. There are at least two mechanisms which could account for differences in organization of auditory cortex between normal-hearing and hearing-impaired populations: 1) Because all hearing-impaired subjects in this study used American Sign Language (ASL), it is possible that some areas of cortex which normally would be assigned to auditory processing have instead been organized to process visual stimuli. Evidence of such reassignment of cerebral cortex has been reported in human studies (Neville et al., 1983) and animal studies (Rebillard et al., 1977; Sur et al., 1988).

2) Because the hearing-impaired subjects in this study had profound, bilateral, high-frequency hearing loss, it is possible that the organization of their auditory cortex is determined by the limited range of auditory stimuli they can perceive and process. It is likely that the organization of their auditory cortex is markedly different from that of subjects with normal hearing. The EEG study by Wolff & Thatcher (1990)

provided evidence for differences in organization of cerebral cortex between normal and hearing-impaired children.

Several animal studies have demonstrated that the organization of auditory cortex can change in response to an individual's experience with sound. Robertson & Irvine (1989) reported reorganization of primary auditory cortex (AI) in guinea pigs after unilateral cochlear lesions. Harrison et al. (1991) described a similar reorganization of AI in kittens deafened with the ototoxic aminoglycoside amikacin. Plasticity of auditory cortex has also been demonstrated in mice (Willott et al., 1993), macaque monkeys (Schwaber, Garraghty & Kaas, 1993), and owl monkeys (Recanzone, Schreiner & Merzenich, 1993). In light of these observations in animals, it seems likely that differences in organization of auditory cortex exist between the normal-hearing and hearing-impaired humans in this study. These differences in cortical organization are reflected by differences in topography of the N1 component between the normal and hearing-impaired groups.

There are other differences in AEP responses to speech stimuli between the two experimental groups in this study. The most common difference is that N1 latencies from hearing-impaired subjects are greater than those from normal subjects. P2 latencies to speech stimuli are not usually different. Also, N1 amplitudes from hearing-impaired subjects were usually smaller than normals while P2 amplitudes were usually not different. These results indicate that the N1 component is an effective indicator of differences in processing of complex auditory stimuli. Because several areas of auditory cortex contribute to the generation of N1 (Woods, 1995), differences

in N1 latency or amplitude might reflect differences in organization of auditory cortex between the two experimental groups.

How did these cortical differences result in temporal delays of N1 components in AEPs recorded from the hearing-impaired group? Behavioral studies of normal-hearing humans have shown that auditory discrimination thresholds decrease with increasing stimulus duration (Hughes, 1946; Zwicker & Wright, 1963; Watson & Gengel, 1969). The decrease in thresholds is thought to result from temporal integration in the central auditory system (Plomp & Bouman, 1959; Zwislocki, 1965). Temporal integration -- or temporal summation -- occurs when presynaptic neurons fire repeatedly, increasing their effect on the membrane potential of the postsynaptic neuron (Kandel & Schwartz, 1985). In studies of psychoacoustics, the maximum integration time has been estimated between 100 and 300 msec. That is, stimuli longer than 300 msec will not significantly decrease behavioral auditory thresholds of normal-hearing humans.

Humans with cochlear damage typically show less temporal integration than normal-hearing subjects (Miskolczy-Fodor, 1953; Elliott, 1963; Pedersen & Elberling, 1972; Carlyon & Sloan, 1987; Florentine et al., 1988). Wright (1968) suggested that excessive adaptation of impaired cochleas in response to the later portions of long-duration signals would produce fewer neural events to summate at later stages of auditory processing than would the earlier portions of the same signals. This would cause a smaller-than-normal reduction of threshold with increasing stimulus duration.

A related hypothesis called "perseverance" or "persistence" (Carlyon et al., 1990)

states that the internal representations of signals in impaired auditory systems persist for a fixed duration after signal offset. This would reduce the ratio between the effective durations of long and brief tones, and thereby also reduce temporal integration.

A third model of temporal integration in the auditory system was proposed by Penner (1978). Studies of cat auditory nerves (Kiang, 1968; Sachs & Abbas, 1974) and guinea pig cochleas (Russell & Sellick, 1978) provided evidence that sound stimuli undergo peripheral compressive nonlinearity in normal ears. That is, a specific area of the cochlea gives a sharply tuned response to lower intensity sounds of a particular frequency. As sound intensity increases, the sharp tuning disappears (see Figure 60 below). This occurs because as stimulus intensity increases, the cochlear response grows relatively slowly in the region of the peak, and faster for lower frequencies.

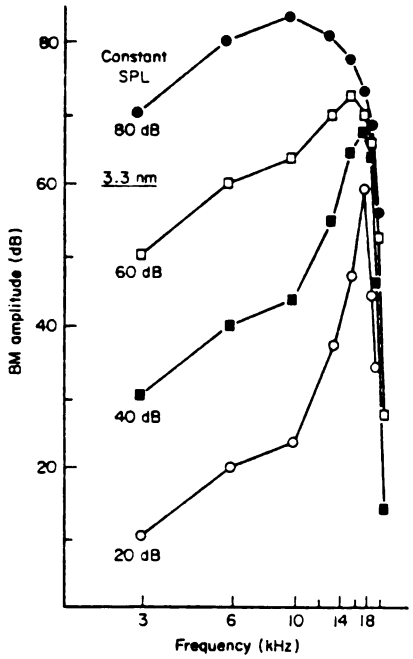


FIGURE 60. Amplitude of basilar membrane vibration plotted as a function of stimulus frequency. Responses were measured at the 18kHz point on the guinea pig basilar membrane. From Johnstone et al., 1986.

Penner hypothesized that if cochlear impairment reduced the compressive nonlinearity, this would decrease the slope of the temporal integration function. Subsequent studies of guinea pig cochleas by Sellick et al. (1982) and Cody & Russell (1988) demonstrated that nonlinearity is reduced in impaired ears.

EEG signals represent the fluctuating sum of excitatory and inhibitory postsynaptic potentials occurring at the apical dendrites of pyramidal cells (Epstein & Andriola, 1983). Evoked potentials represent averaged summations of neural activity which are time-locked to specific stimuli. Consequently, auditory evoked potentials provide a measure of temporal integration of auditory stimuli. Therefore, differences in AEP component amplitudes and especially latencies reflect differences in temporal integration between subject populations.

ENDOGENOUS COMPONENTS

P3 components were recorded from normal and hearing-impaired subjects in response to tones and words in Protocol 1. Why would the P3 component be generated during Protocol 1? Within the normal-hearing group, P3s were more common and had greater amplitudes in response to words rather than tones. It is possible that normal subjects were more attentive to the word "POT" than to the 250 Hz tone. Larger P3s in response to words could be a reflection of this enhanced attention. The presence of P3 components might indicate activation of brain areas which contribute to states of attention (including the reticulothalamus and frontal cortex) and have been proposed as generators of the P3. Parietal P3s were evident in normal responses to CVs ("PA" or "POT"), but not to the vowel "AH." This might

be indicative of differential neural processing of these speech sounds.

Within the hearing-impaired group, P3 components of equal amplitude were generated in response to both tones and words. In fact, the amplitude of the hearing-impaired P3 to tones is larger than the normal P3 to tones. Why would hearing-impaired subjects generate P3 components to both types of stimuli in this protocol? Perhaps because it was somewhat novel for them to hear sounds at relatively high intensities. P3 components are also present in AEPs of hearing-impaired subjects in responses to "PA" and "POT," but not to "AH." The topography of this hearing-impaired P3 is more frontal than that from normal subjects. This indicates that the P3 is not merely an effect of enhanced attention, but might be the result of specific neural processing of these stimuli. Once again, when compared to normals, the more frontal distribution of this component in hearing-impaired AEP maps might reflect differences in organization of cerebral cortex between the groups.

TONAL STIMULI - The "Oddball" Paradigm

Protocol 5 compared P3 generation between the experimental groups in responses to low frequency tones (125 Hz "common" / 250 Hz "rare"). Compared to normal responses, hearing-impaired N1 and P2 latencies are greater; N1 and P2 amplitudes are smaller. These results are a reflection of lower subjective stimulus intensities presented to the hearing-impaired group. However, even though relative stimulus intensities were not equal, P3 latencies and amplitudes recorded from each group are not significantly different. This result suggests that profound hearing impairment does not necessarily result in delays in processing of "rare" auditory stimuli.

The distribution of the normal P3 component in response to rare tones in this protocol is more central than it was in response to consonant-vowels. Again, this might indicate a difference in neural processing of tonal vs. speech stimuli. Hearing-impaired P3 components are centrally distributed in response to both tones and words. This indicates 1) differences in organization of cerebral cortex between the normal and hearing-impaired groups and/or 2) reduced differential processing of tonal vs. speech stimuli by hearing-impaired subjects.

NAME STIMULI

During Protocol 6, AEPs were recorded from each subject in response to 30 repetitions of his or her own first name randomly interspersed among an assortment of 80 other first names. A large P3 component is present in the AEPs of every normal subject in response to his or her own name, but there is no P3 in response to other names. This result indicates an increase in activation of higher-order processing centers in response to one's own name. The distribution of this P3 component is centered around the Pz electrode, similar to the distribution of normal P3s in response to consonant-vowel stimuli.

No P3 component is present in responses from hearing-impaired subjects. This indicates that hearing-impaired subjects did not recognize their own names during this protocol (confirmed by asking each subject after data collection).

Protocol 7 was designed as a control experiment for the normal-hearing group only. The absence of a P3 component in Condition 2 indicates that subjects do in fact respond preferentially to their own names, not just to any name that is repeated more

often or one that appears in particular positions within the stimulus sequence. During interviews after data collection, all normal subjects stated that they noticed one name was repeated more often than others in this protocol. However, their notice of this fact did not result in the generation of a P3 component in response to the repeated name.

EFFECTS OF ATTENTION TO STIMULI

According to studies by Hillyard et al. (1973, 1978), amplitudes of N1 and P2 components increase when subjects are instructed to pay attention to (e.g., by counting) stimuli. However, Polich (1989b) reported that latencies of N1 and P2 components did not change significantly with attentional states of normal, awake subjects. Similarly, Kaukoranta et al. (1989) reported no significant differences in latencies of N100m magnetic components with changes in normal subject attention.

Subjects were not required to respond in any way to any of the stimuli during data collection in the present study. All of the protocols can thus be considered "passive" (Ritter et al., 1968; Picton et al., 1976; Polich, 1987). However, there were differences in subjects' attention to stimuli. During Protocols 1 through 3 and 5 through 7, subjects sat quietly in the booth with their eyes open and fixed on a spot. During Protocol 4 subjects read newspapers, magazines, or books during data collection. Responses to identical stimuli presented during these different states of attention were compared.

Within the normal-hearing group, N1 latencies to 500 msec tones while subjects were reading (Protocol 4) are greater than N1 latencies to the same tones while subjects passively attended to stimuli (Protocol 1). P2 amplitudes in response to these

tones in Protocol 1 are greater than P2 amplitudes in Protocol 4. The habituation phenomenon -- if it occurred -- in response to these tones during Protocol 4, was not apparent during Protocol 1. This difference in responses to the same stimulus could have various explanations: 1) differences in subject attention caused by reading vs. passive listening; 2) differences in the number of conditions in each protocol (two conditions in Protocol 1 vs. twelve conditions in Protocol 4); 3) differences in subject attention caused by the time at which each protocol was presented (Protocol 1 near the beginning of the session; Protocol 4 near the end).

However, normal responses to the word "POT" in Protocol 1 are not significantly different from normal responses to "POT" in Protocol 4. Again, this result might reflect the fact that normal-hearing subjects are more attentive to speech stimuli than to repetitious tonal stimuli.

Within the hearing-impaired group, there is only one significant difference in responses to tones and words between Protocols 1 and 4: P2 amplitudes to tones in Protocol 1 are greater than those in Protocol 4. Responses to the word "POT" are not significantly different between the protocols. It is possible that complex auditory stimuli attract the attention of all humans (even those with profound hearing impairment) more than pure tone stimuli.

SIMULATED HEARING LOSS IN NORMAL SUBJECTS

Protocols 2 and 3 were designed to test the hypothesis that AEP responses recorded from hearing-impaired subjects can be reproduced in normal-hearing subjects under conditions of simulated hearing loss. Hearing loss was simulated in normal

subjects two ways: 1) by reducing intensity of stimuli 40 dB; 2) by using masking noise to raise audiometric thresholds 40 dB. Because normal stimulus intensities were 40 dB lower than hearing-impaired intensities during all other protocols, the effective difference in stimulus intensity during Protocols 2 and 3 was 80 dB.

The primary effect of increased stimulus intensity on auditory EPs is increased amplitudes of components. A secondary effect of increased stimulus intensity is decreased latencies of components. In a review of long latency studies, Picton et al. (1977) concluded that latencies of N1 and P2 components recorded from normal-hearing subjects decrease with click intensity up to 50 dB SL, above which latencies stabilize. N1 and P2 amplitudes continue to increase with increasing click intensity up to 90 dB HL. Adler & Adler (1989) used 1000 Hz tones to elicit N1 and P2 component responses from 10 normal-hearing subjects. As stimulus intensity increased from 30 to 70 dB SL, N1 and P2 latencies decreased and amplitude increased. However, at stimulus intensities from 70 to 90 dB SL, N1 and P2 latencies increased, N1 amplitude decreased, and P2 amplitude increased at a slower rate than it did between 30-70 dB. This could be another example of habituation.

In the present study, the same stimulus tape (containing 500 msec duration tones and the word "POT") used in Protocol 1 was also used in Protocols 2 and 3. Hearing-impaired responses to tones were replicated by normal subjects during both protocols. That is, normal N1 latencies increased and N1 amplitudes decreased in response to tones during Protocols 2 and 3. However, normal responses to the word "POT" were not the same in each of these protocols. In Protocol 2, normal N1

amplitudes were reduced to hearing-impaired levels, but normal N1 latencies did not increase significantly. In Protocol 3, normal N1 latencies increased and N1 amplitudes decreased in response to the word "POT" + masking noise. Under these conditions, normal AEP waveforms in response to "POT" were not significantly different from hearing-impaired AEP waveforms. This matching of AEP waveforms probably occurred because the noise masked the high frequency elements of the word, simulating high frequency hearing loss in normal subjects.

Numerous psychoacoustic studies have used masking noise to simulate sensorineural hearing impairment in normal subjects (Florentine et al., 1988; Moore & Glasberg, 1993; Sommers & Humes, 1993; Buus et al., 1995). Buus et al. suggested using the term "equivalent-threshold masking (ETM)" as a substitute for "simulated hearing impairment." This is preferred terminology, they contend, because "ETM produces only an approximation of some of the effects of a cochlear hearing impairment." Buus et al. state that "frequency selectivity, loudness summation across frequency, temporal summation, and temporal resolution all appear relatively unaffected by masking a normal ear." Even when the addition of masking noise causes audiograms of normal subjects to match those of hearing-impaired subjects, these populations continue to exhibit differences in auditory perception and processing. What is the cause of these differences? It is likely that differences exist in the organization of auditory cortex between humans with normal hearing and those with sensorineural impairment.

Schoonhoven (1992) recorded auditory brainstem responses (ABRs) to clicks from

twelve normal subjects with simulated high frequency sensorineural hearing impairment produced by the addition of masking noise. ABR components recorded during the masked condition had increased latencies and decreased amplitudes compared to those recorded during the unmasked condition. This is a predictable result caused by increased auditory thresholds of normal subjects with simulated hearing impairment. However, ABR studies such as this give little insight into the organization of central auditory pathways. Ferman et al. (1993) recorded ABRs to clicks from 20 subjects with normal audiograms but with elevated speech reception thresholds (SRTs) in noise. Normal ABRs were recorded from 19 of these subjects. This result led Ferman et al. to conclude that "an elevated SRT in noise does not lead to an abnormal auditory brainstem-evoked potential. This indicates that the cause of an elevated SRT in noise probably lies at a more central level."

In the present study, the addition of masking noise caused normal AEP waveforms to match those of hearing-impaired subjects in responses to the same stimuli. However, the topography of normal AEPs in response to "POT" remained consistently distinct from hearing-impaired topography *even during the addition of masking noise.* This result indicates that differences exist in the organization of central auditory pathways between normal and hearing-impaired humans. These organizational differences probably came about as a consequence of differences in abilities to perceive high frequency sounds (including the high frequency components of speech). This might explain why simulating hearing impairment in normal subjects by using masking noise does not, in the words of Buus et al. (1995), "reproduce other

abnormalities that may result from cochlear impairments."

CLINICAL UTILITY OF THESE PROCEDURES

As stated in the INTRODUCTION section of this dissertation, long latency AEPs can be useful in assessing auditory and cognitive functions in various patient populations. In particular, the P3 component could be used to assess name recognition in non-responsive patients.

According to Dr. Larry Squire, Professor of Psychiatry and Neurosciences at the University of California, San Diego, "To forget your own name is to lose one of the most strongly encoded pieces of information you have in your brain. Even people with advanced Alzheimer's or head trauma answer to their name" (Boubion & Schmitz, 1995). However, some patients do not have the ability to give an appropriate response. For example, it is often difficult to determine if a comatose patient can understand his or her own name or anything else that is said (Goshen-Gottstein, 1990). It is similarly difficult to assess the comprehension of neonates or infants. A behavioral study by Mandel et al. (1997) suggests that infants respond to the sound of their own name by the age of 4½ months.

Several studies have proposed the auditory P3 component as a predictor of patients' recovery from a comatose state (Yingling et al., 1990; Gott et al., 1991; De Giorgio et al., 1993). O'Mahony et al. (1990) suggested that passive P3 paradigms used to evaluate comatose or demented patients are less effective than active procedures. In order to enhance patients' responses to tones during a passive P3 paradigm, Signorino et al. (1995) used a set of conditioning trials during which tones

were coupled to verbal stimuli with an emotional content (including the patient's name). The verbal stimuli followed the tones with a delay of 900 msec. Signorino et al. reported that "emotional conditioning" increased the probability of obtaining P3 components from the 16 comatose patients they studied.

The next logical step in this progression is to record AEPs in response to the patient's first name. A procedure for assessing name recognition in non-responsive patient populations would be a valuable tool for clinicians, researchers, and family members of patients.

During the 68 year history of electroencephalography, very little has been published regarding brain activity recorded from humans in response to their own name. A study by Shimizu et al. (1977) correlated alpha activity during REM sleep with waking recall of names spoken on tape. Shimizu's results indicated that longer duration alpha activity during REM sleep was positively correlated with better waking recall of names.

Flinn et al. (1993) recorded long latency AEPs from malnourished and normal infants (aged 3 to 12 months) in response to their names spoken on tape. Flinn et al. reported that the malnourished infants had fewer peaks in their recorded responses to their own name. Flinn et al. did not report latencies of AEP components nor did they attempt to identify components. It is important to note that the infants were asleep during these procedures. Responses recorded during sleep can vary greatly from cortical potentials recorded from awake individuals.

The procedure described in the present study was used to assess name recognition

in one patient (male, age 87 years) with global aphasia which occurred as a result of hemorrhage of the left middle cerebral artery two years earlier. The patient also had right side hemiparesis, but was conscious and alert to his surroundings during this procedure. The patient did react to sounds (e.g. environmental sounds or voices), but his auditory thresholds and level of comprehension were unknown. Observations indicated that the patient could not comprehend spoken or written language.

Figure 61a contains long latency AEPs recorded from this patient in response to 80 msec duration tones in a P3 "oddball" paradigm. AEPs were recorded by the QSI System 9000 from Cz electrode referenced to earlobe electrodes. AEPs were amplified 40,000 times. AEPs accompanied by eye movements greater than 60 μV in amplitude were not included in the averages. Stimuli were presented through two Sony Walkman speakers, one placed 70 cm lateral to each of the patient's ears. Rate of stimulus presentation was 1/sec. Stimulus intensity was 85 dB SPL. Recordings took place in the patient's room in a convalescent hospital in San Bruno, California. Note the presence of N1 and P2 components in the patient's response to 250 each of the "common" (1000 Hz) tones. N1 and P2 latencies and amplitudes are within the normal range. N1 and P2 components are also present in his response to 46 each of the "rare" (2500 Hz) tones. A P3 component is not evident in response to 2500 Hz tones. It is possible that brain damage suffered by this patient interfered with generation of the P3 component. Could he, in spite of the absence of a P3

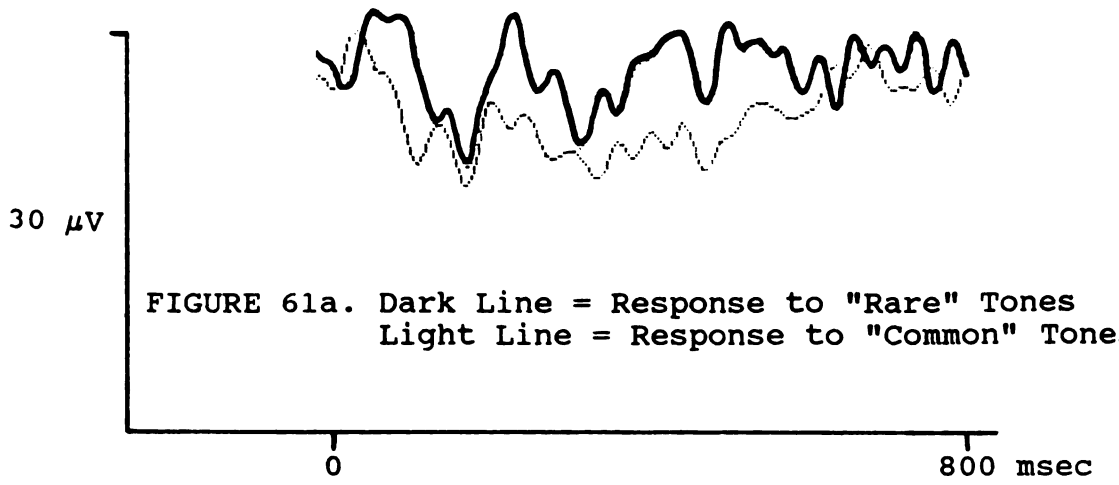


FIGURE 61a. Dark Line = Response to "Rare" Tones
Light Line = Response to "Common" Tones

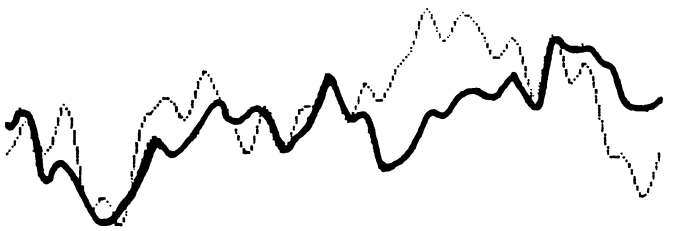


FIGURE 61b. Dark Line = Response to Words
Light Line = Response to 250 Hz Tones

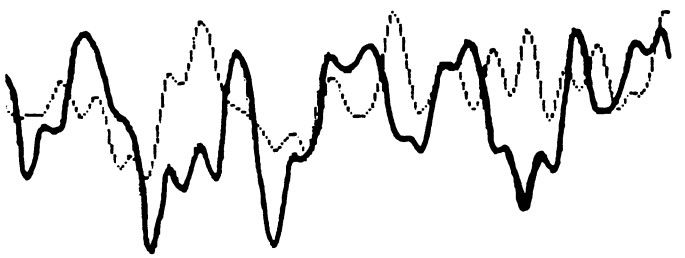


FIGURE 61c. Dark Line = Response to Patient's Own Name
Light Line = Response to Other Names

component, distinguish between 1000 and 2500 Hz tones? This could not be determined.

Figure 61b contains AEPs recorded from this patient in response to 90 each 500 msec duration tones (250 Hz) and 30 each 500 msec duration one-syllable words (pot, pit, pet, top, tip, tap). N1 and P2 components are present in responses to both tonal and word stimuli. Perhaps the longer durations of these stimuli are responsible for these more distinct AEP waveforms. Again, N1 and P2 latencies and amplitudes are within the normal range. However, a P3 component is not present in response to words. It is likely that the patient was unable to distinguish words from other auditory stimuli.

Figure 61c contains AEPs recorded from this patient in response to 30 repetitions of his own first name ("George"). Figure 61c also contains AEPs recorded from the patient in response to an assortment of 90 one-syllable male first names other than George. N1 and P2 components appear in responses to both conditions. This indicates processing of stimuli by auditory cortex and probably means that the patient could hear the sounds. However, the absence of a P3 component in response to his own name indicates that the patient did not recognize his name. This is consistent with observations of the patient's inability to comprehend spoken or written language.

The procedure used in the present study could be a valuable tool for assessing states of consciousness and cognitive functioning in the following populations: 1) comatose patients; 2) stroke patients; 3) patients with Alzheimer's Disease or other forms of dementia; 4) psychiatric patients; 5) head trauma patients; 6) patients who

claim to have amnesia; 7) autistics; 8) mentally retarded patients; 9) infants and children.

The procedure described in the present study could provide valuable insight into the cognitive processing of non-responsive patients. Further investigations should be undertaken to determine 1) if all normal adults generate a P3 component in response to their own first name, but not to other names; 2) how a male vs. female or familiar vs. unfamiliar voice affects the results; 3) at what age normal infants or children generate characteristic AEP responses to their own name; 4) how various pathological conditions affect these responses.

In addition to name recognition, AEPs can be used to assess auditory functions and cortical organization. For example, AEPs can be used to monitor cortical reorganization in patients who receive cochlear prosthesis implants. Several investigators recorded auditory brainstem or middle latency responses from cochlear implant recipients post-operatively (e.g. Gardi, 1985; Kileny & Kemink, 1987; Shallop et al., 1990). The stimuli in all of these studies were delivered electrically via the electrode array within the cochlea.

Pelizzone et al. (1986) recorded long latency AEPs from one recipient of the 4-channel Ineraid cochlear prosthesis manufactured by Symbion Corporation. Stimuli were 20 msec duration 1000 Hz tones delivered through a loudspeaker to the microphone of the speech processor. Responses recorded from vertex included a negative component 65 msec after stimulus onset (N1) and a positive wave 180 msec post-stimulus onset (P2). Component amplitudes were within the normal range.

Oviatt & Kileny (1991) compared the P3 response components from ten recipients of Nucleus multichannel prostheses with P3s recorded from ten normal-hearing subjects. Frequent stimuli were 100 msec duration 500 Hz tones delivered through loudspeakers. 1000, 2000, or 3000 Hz tones were presented at random 20% of the time to elicit the P3 component. Oviatt & Kileny reported that P3 latency was significantly longer for implant recipients in response to 500 Hz/1000 Hz common/rare tone combinations than it was in response to other combinations.

Kaga et al. (1991) recorded the P3 component from one recipient of the House-3M single channel cochlear implant. Stimuli were 100 msec duration 250 and 500 Hz tones; and 400 msec duration words: "aka" (red) or "kuro" (black). Pairs of tones or words were presented in ratios of 80% common to 20% rare in order to elicit the P3 component. Small amplitude P3 components were elicited from the subject three months after prosthesis implantation. P3 amplitude increased significantly when tests were repeated six months post-implantation.

Micco et al. (1995) recorded long latency AEPs in response to the syllables "da" (the "rare" stimulus presented 20% of the time) and "di" (the "common" stimulus presented 80% of the time) from nine recipients of the Nucleus cochlear implant and from nine age-matched subjects with normal hearing. Latencies and amplitudes of P3s from each group were not significantly different. N1 and P2 latencies recorded from each group were also similar. However, N1 amplitude was significantly smaller in responses from cochlear implant recipients. As was the case in the present study, differences in audiometric thresholds and subjective stimulus intensities (65 to 70 dB

SPL used for all subjects) were probably responsible for differences in N1 amplitude.

All of the investigators mentioned here recorded AEPs from subjects after cochlear prosthesis implantation. Pre-operative AEPs have been recorded from cochlear prosthesis candidates in response to electrical stimulation of the round window, promontory, or external auditory meatus (Kileny & Kemink, 1987; Brix & Gedlicka, 1991; Shallop, 1993). This type of electrical stimulation is an invasive procedure which usually requires local anesthesia and causes varying degrees of discomfort for patients.

A simpler, less invasive procedure for recording pre-operative AEPs is to deliver stimuli through external speakers. This method was used in the present study to record long latency AEPs from a cochlear implant patient pre- and post-operatively. This procedure has the following advantages:

1) Delivery of stimuli through external speakers abolishes the need for anesthesia and greatly increases patient comfort during recording sessions.

2) Because long latency AEPs from each patient have individual characteristics, recordings made after implantation will be more meaningful when compared to AEPs recorded from the same patient before implantation.

3) Studying long latency AEPs recorded from cochlear prosthesis patients might provide insights into the organization of central auditory pathways. Comparing long latency AEPs recorded from the same patients before and after implantation might provide information about how the organization of central auditory pathways changes in response to increased acoustic stimulation.

Patient JC, a 58-year-old male, first noticed hearing deficits at age 36. His hearing loss progressed slowly for twelve years, then the progression became rapid, reaching profound levels within two years. It is likely that JC's progressive hearing loss was caused by loud industrial machinery (including a flame cutter) he operated in a naval shipyard for more than 30 years. JC was also exposed to ototoxic chemicals (including mercury, lead, and cobalt) at his workplace. There was no familial history of deafness and no report of head trauma. JC was selected to receive a cochlear prosthesis implant at the University of California, San Francisco Medical Center. Figure 62 contains the audiogram of JC eight days before he was implanted with the Clarion cochlear prosthesis. Left and right ear thresholds were obtained by presenting 500 msec duration tones through standard headphones which covered the pinnae. JC could not detect tones with frequencies above 2000 Hz presented at 110 dB HL and he could not understand speech without lipreading. His ABR recorded from Cz showed no response to 90 dB HL clicks presented monaurally to each ear. Figure 62 also contains auditory thresholds of JC 20 weeks after implantation (16 weeks after he began using a speech processor). These thresholds were obtained by presenting 500 msec duration tones through speakers to the prosthesis microphone mounted above his left ear. At the time of this testing JC still could not understand speech without lipreading, but he reported that he could hear differences between speakers more clearly than he had for many years. Auditory evoked potentials were recorded from JC one day before and again five months after implantation with the Clarion 8-channel cochlear prosthesis (manufactured by MiniMed Technologies; Sylmar, CA). The 16-

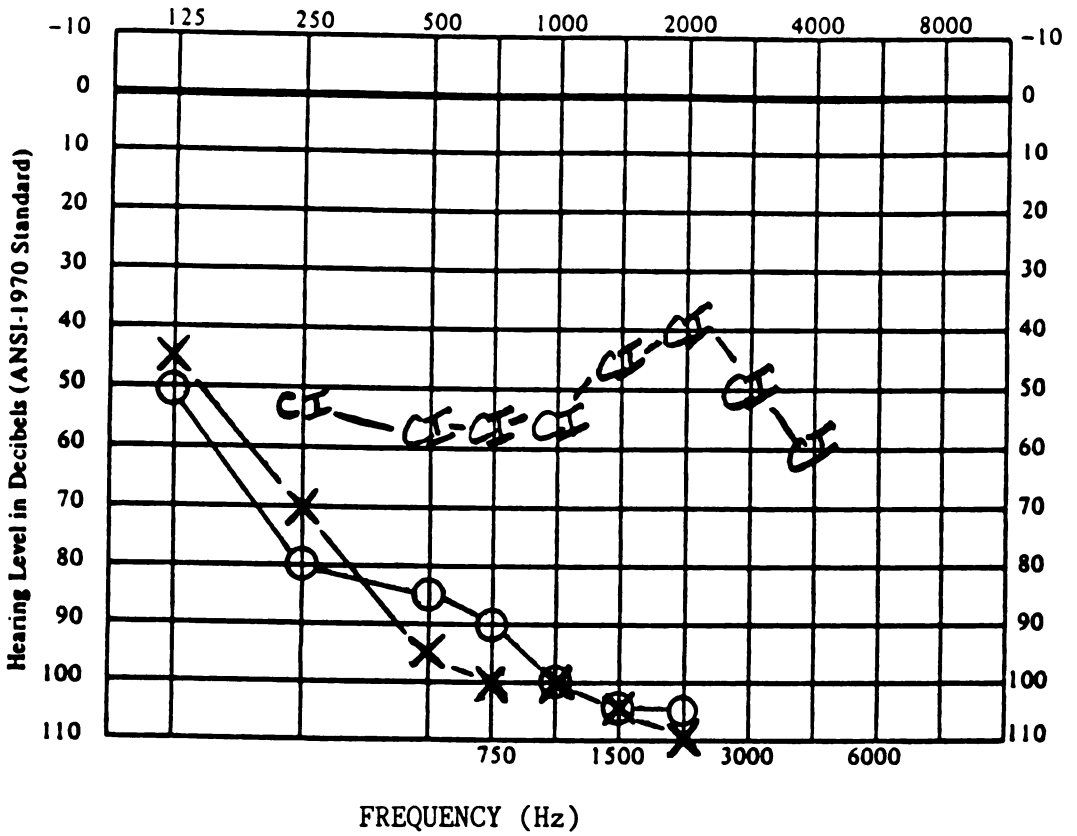


FIGURE 62. Audiometric evaluation of patient JC
O = right ear thresholds before implantation
X = left ear thresholds before implantation
CI = thresholds after prosthesis implantation

electrode array was implanted into his left cochlea. At the time of the second recording session, JC had been using a speech processor for four months. Responses were recorded from JC according to the following experimental conditions.

Experiment 1

Stimuli: 30 each 80 msec duration sinusoidal tones presented in a random sequence of frequencies: 125, 250, 1000, 1500, 2000, 2500, and 3000 Hz. The interstimulus interval varied between 1.5 to 2.0 sec. Tones were synthesized by a Wave-Tek Model 184 Sweep Generator then amplified by one channel of a Realistic SA-1001 stereo amplifier. Tones were presented through speakers at 30 dB SL to a maximum of 110 dB SPL.

Experiment 2

Stimuli: 30 each 500 msec duration, 250 Hz tones interspersed at random with 30 each 500 msec duration one-syllable words (such as pot, pit, pet, top, tip, tap) pronounced by a male speaker. The interstimulus interval again varied between 1.5 to 2.0 sec. Tones and words were recorded and digitized according to the procedures described earlier in the present study. Tones and words were presented through speakers at 30 dB SL to a maximum of 110 dB SPL.

Procedure/EP Recording

JC sat in a comfortable reclining chair within a sound-attenuated room. Speakers were positioned 30 cm from and lateral to each of his ears. Auditory thresholds were verified before AEP recording began. JC was instructed to keep his eyes open and fixated on a spot of light projected on the wall in front of him. AEPs were recorded,

averaged, and filtered by Grass amplifiers and Erpsystem according to parameters described in the present study.

RESULTS

Experiment 1

Figure 63 contains the auditory evoked responses of patient JC to 80 msec duration tones one day before and 21 weeks after prosthesis implantation. Each trace is the averaged response to 30 similar tones recorded from Cz. Responses recorded from a normal-hearing subject to the same stimuli are included in Figure 63 for comparison. Most striking is the reduction of JC's N1 latencies after implantation. Also notice the N1 and P2 components in JC's responses to 2500 and 3000 Hz tones after implantation which were not evident before implantation.

Experiment 2

Figure 64 contains the averaged responses recorded from patient JC to 30 each 500 msec duration, 250 Hz tones and to 30 each 500 msec duration one-syllable words. Responses from a normal-hearing subject to the same stimuli are also shown in Figure 64. Table 3 contains N1 and P2 latency and amplitude values for the responses from patient JC before and after implantation. Again note the reduction of N1 and P2 latency in JC's responses to both tonal and word stimuli after implantation.

One of the aims of this study was to compare long latency AEPs recorded pre-operatively from a profoundly hearing-impaired patient with his auditory evoked responses recorded after cochlear prosthesis implantation. The latencies of patient JC's N1 components in response to 80 msec duration tones of various frequencies

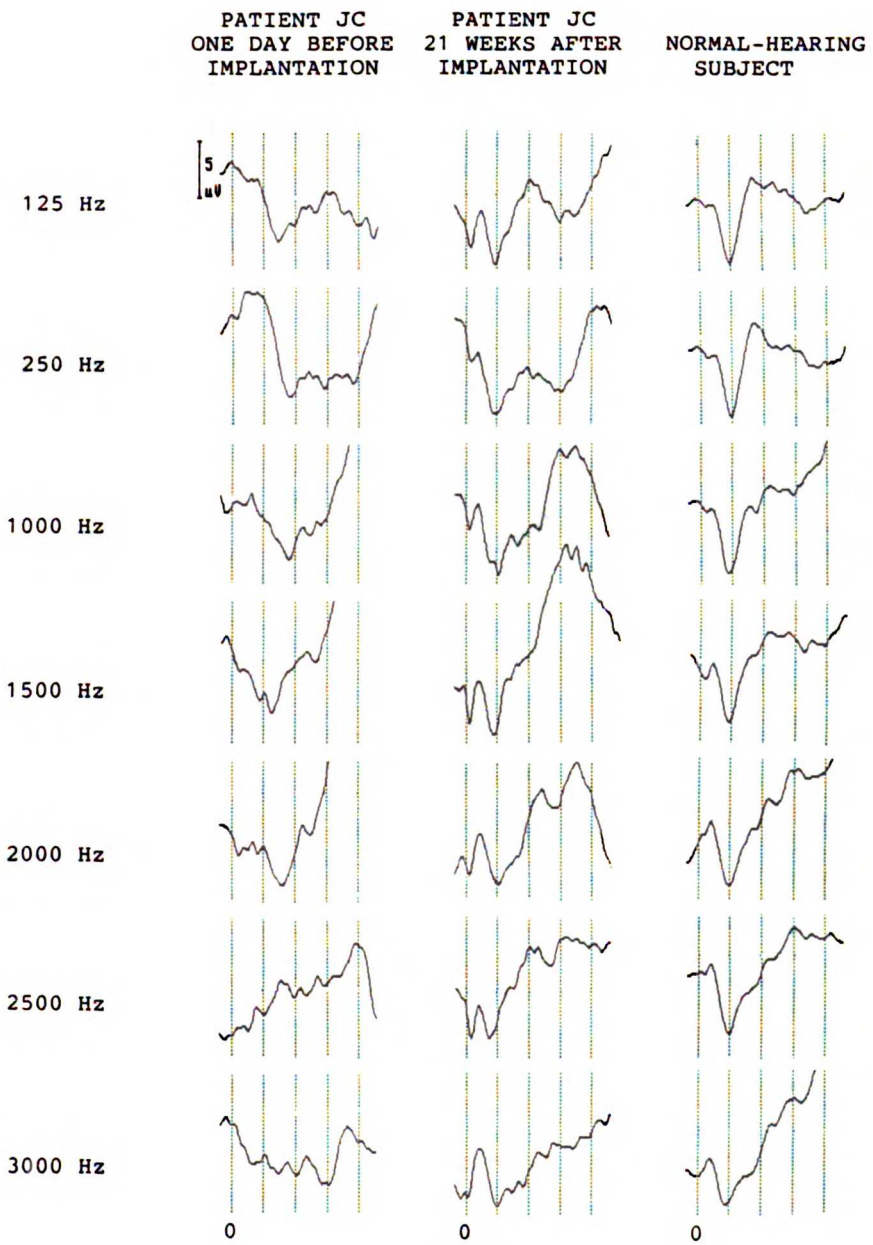


FIGURE 63. Responses to 80 msec Tones recorded from Cz.
Vertical lines every 100 msec.
0 = stimulus onset

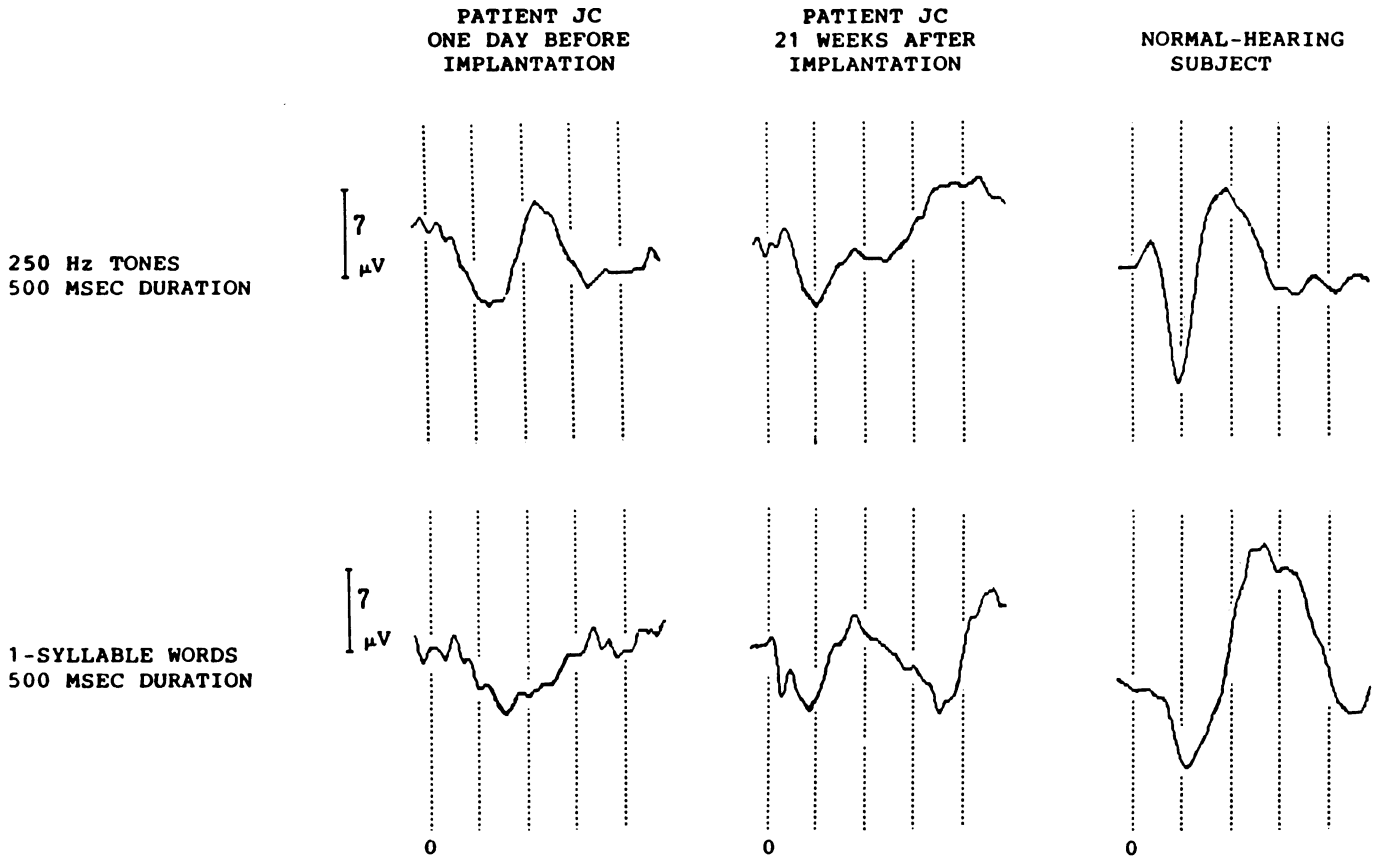


FIGURE 64. Responses recorded from Cz.
Vertical lines every 100 msec.
0 = stimulus onset

TABLE 3. Latencies and amplitudes of N1 and P2 components recorded from patient JC before and after cochlear prosthesis implantation

	BEFORE	AFTER
N1 lat. to Tones	130	102
N1 amp. to Tones	6.6	5.4
P2 lat. to Tones	227	180
P2 amp. to Tones	2.6	0.0
N1 lat. to Words	154	90
N1 amp. to Words	5.3	5.6
P2 lat. to Words	-	182
P2 amp. to Words	-	2.9

were reduced 21 weeks after implantation. In fact, after implantation, JC's responses were similar to AEPs recorded from normal-hearing subjects. A similar reduction of N1 latency in JC's responses to 500 msec duration tones and words occurred after implantation. These reductions of N1 latency are probably produced by increased auditory sensitivity accomplished through JC's speech processor and cochlear prosthesis (see Audiogram - Figure 62). It is possible that these dramatic reductions of N1 component latencies represent more than a simple increase in subjective stimulus intensity. When possible, stimuli were delivered at a constant sensation level (30 dB SL) throughout these experiments in order to reduce the influence of increased stimulus intensity on AEP component latency. The reduction of N1 latency in response to tones and speech sounds represents an increase in the speed at which the central auditory system processes stimuli. Such a change within patient JC could be indicative of reorganization of auditory cortex in response to increased sensory input via the cochlear prosthesis.

Additional studies of cochlear prosthesis recipients are recommended in order to correlate changes in AEPs with changes in behavioral thresholds and speech discrimination. This information could be useful in the design of speech processors and training programs for these patients.

TEMPORAL PROCESSING DISORDERS

In addition to the applications described previously, long latency AEPs could also be useful in assessing individuals with language disorders or learning disabilities. For example, the present study demonstrated differences in neural temporal integration

of auditory stimuli between hearing-impaired and normal-hearing humans. Behavioral studies by Tallal & Piercy (1973, 1974) have shown that children with developmental dysphasia are impaired in their ability to perceive the temporal sequence of nonverbal and verbal auditory stimuli. Their subjects with language impairment, but not their control subjects, were adversely affected by decreases in the duration of stimuli, decreases in the interstimulus interval (ISI), and increases in the number of stimuli presented. Tallal & Piercy found that children with language disorders showed no impairment on any of the perceptual or serial memory tasks when steady state vowels were used. In contrast, however, the children showed considerable impairment on *all* processing tasks when syllables differentiated by stop consonant contrasts were employed.

In a later study, Tallal & Piercy (1975) extended the duration of the initial formant transitions of the consonant-vowel pairs from 40 msec to 80 msec. With these extended-duration speech sounds, the children with language impairment were able to perform as accurately as the controls on both discrimination and sequencing tasks. Stark, Tallal, & McCauley (1988) conclude that "deficits in basic neuropsychological processing, particularly auditory temporal processing, may contribute to deficits in the development of productive as well as receptive aspects of speech communication." (p. 7)

Merzenich & Jenkins (1995) state that "integration time, whatever its neurological origins, can be shortened by behavioral training." Their belief that behavioral training can improve temporal integration within the brains of people with language-based

learning impairments (LLIs) is based on decades of research on cortical plasticity (see Merzenich et al., 1993 for a review).

Merzenich et al. (1996) developed computer "game" training exercises which were designed to improve temporal processing skills in children with LLIs. After a total of 8 to 16 hours of training during a 20-day period, most of the 29 children in the study "improved markedly in their abilities to recognize brief and fast sequences of nonspeech and speech stimuli." In February 1996 Michael Merzenich, Paula Tallal, and their colleagues founded Scientific Learning Corporation in San Francisco. A version of their language training program, Fast ForWord, is now available to speech and language professionals.

Long latency AEPs can be used to assess LLI individuals before, during, and after training. Procedures described in this dissertation could be used to monitor the improvement in temporal processing (and associated changes in cortical responses) exhibited by people using Fast ForWord or other training programs. Additional AEP studies should be undertaken to investigate the differences in temporal processing between normal and learning disabled people. It is possible that training programs similar to those developed by Merzenich, Jenkins, Tallal and their colleagues could also improve speech discrimination in hearing-impaired people. For this reason, AEP studies should be continued to more completely describe differences in temporal integration between normal-hearing and hearing-impaired populations. Reaction times should be measured simultaneously with AEP recordings in order to correlate behavioral and neurophysiological responses.

SUMMARY

In summary, the findings of this study are:

1) Compared to N1 components recorded from subjects with normal hearing, latencies of N1 components are delayed in hearing-impaired responses to speech stimuli. These delays in AEP component latency indicate differences in neural temporal processing of complex auditory stimuli. Delays in processing of speech stimuli were not duplicated in normal-hearing subjects by reducing stimulus intensity. However, they were duplicated by using masking noise to simulate hearing loss. Additional investigations of temporal processing of speech stimuli are recommended. It is possible that training programs can be developed to improve speech discrimination of hearing-impaired people.

2) As the duration of tonal stimuli increases, N1 and P2 amplitudes decrease and N1 and P2 latencies increase within the normal-hearing group. These changes were not observed in the hearing-impaired group. This could indicate habituation of responses to 500 msec duration tonal stimuli present in people with normal hearing, but not in the profoundly hearing-impaired. This is another example of differences in organization of central auditory pathways between the two groups caused by differences in their experience with auditory stimuli.

3) There are differences in onset responses to noise bursts between the groups. Compared to the normal-hearing group, N1 latencies of onset responses were delayed in hearing-impaired subjects, regardless of the duration of the pause between stimuli. Again, differences in AEP latencies reflect differences in temporal integration of

complex auditory stimuli.

4) In the tonal "oddball" paradigm, even though N1 and P2 amplitudes were smaller; and N1 and P2 latencies were greater from hearing-impaired subjects, P3 amplitudes and latencies were not significantly different from those recorded from normal subjects. These results indicate that profound hearing loss does not in itself cause a delay in the generation of this cognitive component.

5) Normal-hearing subjects produced large P3 components in response to their own first name, but not to other names. P3 components were not recorded from hearing-impaired subjects in response to their own name. This protocol could be useful for assessing name recognition in non-responsive patients.

6) Topographies of AEPs recorded from subjects with normal hearing were different in response to tonal vs. vowel vs. consonant-vowel stimuli. These differences in distributions of AEP components probably represent differential neural processing of these stimuli.

7) Topographies of AEP components were different between the normal and hearing-impaired groups in responses to complex stimuli. Distributions of components recorded from normal subjects in response to the word "POT" remained distinct from hearing-impaired topographies even during conditions of simulated hearing loss. These findings support the hypothesis that AEP topographies are reflections of neural organization.

8) AEPs can be used to assess auditory functioning, cortical organization, temporal integration, and cognitive processing in both normal-hearing and hearing-

impaired populations. Investigations should be undertaken to provide additional data which could be used to develop diagnostic procedures and habilitation programs.

Results from the present study support the central hypotheses of this dissertation:

Differences in experience with sound between normal-hearing and hearing-impaired humans produce differences in the organization of central auditory pathways. This causes delays in temporal processing of complex auditory stimuli within the brains of people with hearing impairment. These delays are reflected in increased AEP component latencies. Differences in AEP topography to similar stimuli reflect differences in the organization of central auditory pathways between normal and hearing-impaired populations.

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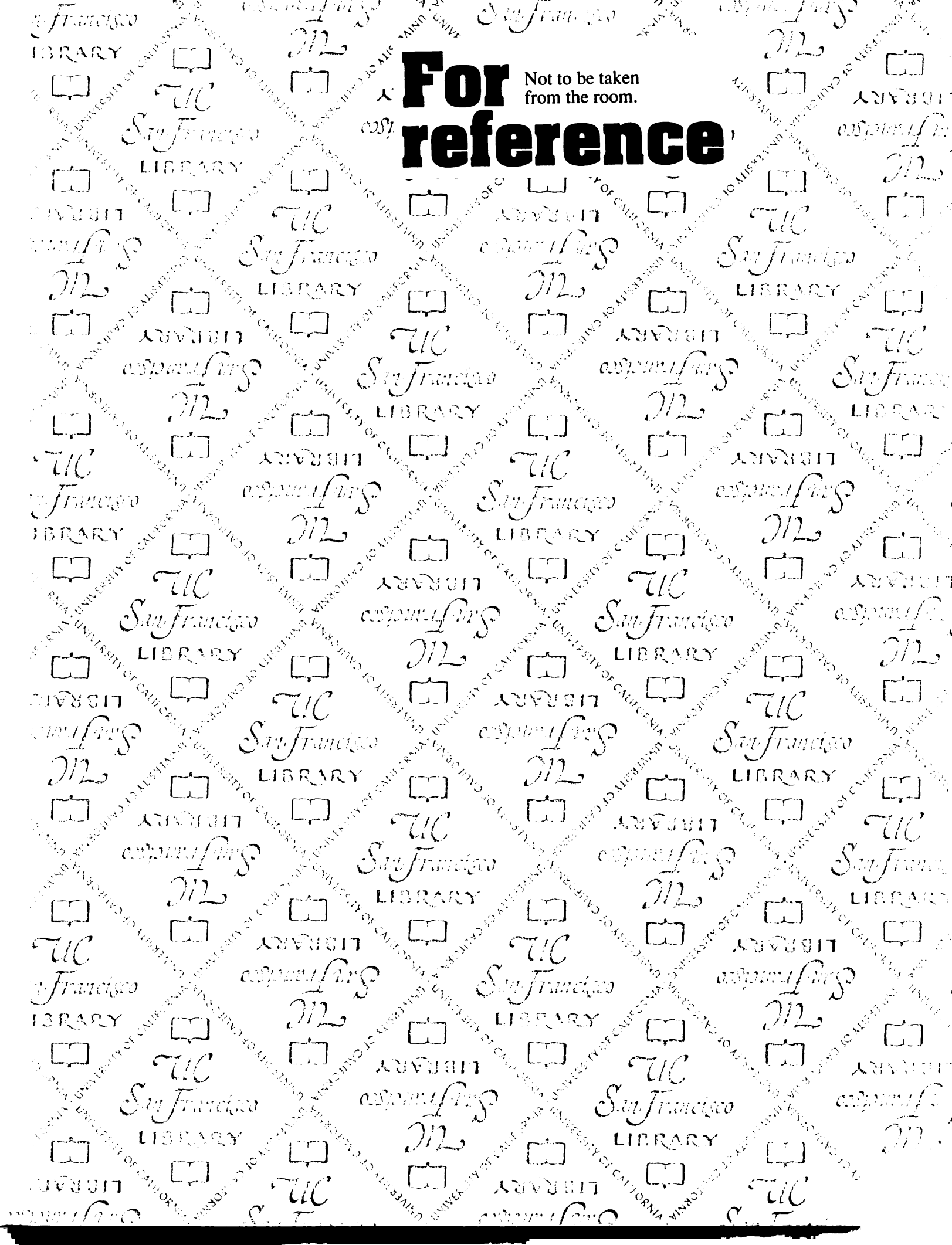
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