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EEG and Evoked Potential Measured of Age and Sex Differences in Central Nervous System Processing

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EEG AND EVOKED POTENTIAL MEASURES OF AGE AND SEX DIFFERENCES IN CENTRAL NERVOUS SYSTEM PROCESSING

by

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Age and gender differences in CNS information processing were investigated with EEG measures of power spectral analysis and cortical coupling, and evoked potential measures of brainstem auditory evoked potentials (BAEPs), visual evoked potentials (VEPs), pattern reversal evoked potentials (PREPs), and P300 evoked potentials. Eighty normal volunteers comprised four subgroups of 20 subjects: young females and young males (25-35 years); old females and old males (55-70 years).

Trends were generally consistent across evoked potential measures: women and young people produced faster latency responses; females and oldsters produced larger amplitude responses. Old age was associated with reduced variability of electrophysiological responding across recording sites.

Significant age and gender findings may be related to CNS excitatory/inhibitory equilibrium. Females and oldsters reportedly experience reduction of some neurotransmitters believed to be inhibitory in function. Furthermore, old age is accompanied by neuropathological changes which could result in heightened CNS excitability.

(170 pages)

CHAPTER I

INTRODUCTION

As researchers strive to understand cortical information processing, they often observe that individuals differ in electrophysiology. A measure of cortical electrical activity that is frequently employed in human studies is the evoked potential (EP), a non-invasive method of probing the central nervous system. (Note: a Definition of Terms follows the introduction.)

The electroencephalogram (EEG) is a recording of the spontaneous electric potentials of the brain derived from scalp electrodes. Although EEGs are useful for studies of sleep cycles, epilepsy, toxicity, and brain lesions, they are less satisfactory for understanding brain function in specific stimulus-response situations. In 1947, George Dawson developed an oscilloscope-trace method of recording the electrical expression of central neural processing, the evoked potential (EP). This more sensitive approach superimposed samples of EEG recordings elicited by repeated stimulus presentations. While the noise of random background brain activity summed towards zero, the response, a time-locked signal embedded in brain waves, increased in clarity. Digital computers are now used to sum and average large numbers of EEG samples. The resultant

evoked potential reflects stimulus-specific response characteristics.

As a complex pattern for analyzing the electrical activity of the brain, the evoked potential is generally described as a series of alternating positive and negative phase shifts elicited by stimulus presentation. The phase shifts reach peaks at identifiable points in time after the presentation of a stimulus. Peaks are typically compared in terms of latency and amplitude. Latency, or peak delay, refers to the time interval between stimulus presentation and a given peak. Amplitude is a measure of the difference in voltage between a prestimulus baseline and a given peak, or between one peak and the next peak of opposite polarity. A polarity-latency convention is commonly used to refer to peaks. For example, P200 indicates a positive component with a latency of about 200 msec. Evoked potentials of each sensory system have unique characteristics.

The brainstem auditory evoked potential (BAEP) measures electrical reactivity of auditory brainstem structures to sound stimuli, e.g., clicks. A typical BAEP consists of seven waves occurring in the first 10 msec. Using the Jewett (1970) classification, the following waves are believed to be associated with particular neural substrates: wave I - acoustic (VIIIth) nerve; wave II - acoustic nerve and pons; wave III - superior olivary complex; wave IV - lateral lemniscus; wave V - inferior colliculus (midbrain); wave VI -

medial geniculate (thalamus); VII - auditory cortex (Beck, 1979; Hashimoto, Ishiyama, Yoshimoto, & Nemoto, 1982; Kjaer, 1980a; Patterson, Michalewski, Thompson, Bowman, & Litzelman, 1981).

The visual evoked potential (VEP) consists of commonly identifiable wave components occurring within 300 msec following stimulation. Flash stimuli, patterned or unpatterned, may be presented for a brief duration at varying intensities. N80, P100, N130, and P200 are four prominent waves found in the VEP.

The pattern reversal evoked potential (PREP) is a waveform produced in response to a visual presentation of black and white squares in a checkerboard pattern alternating at a fixed rate, i.e., black squares become white, and white squares become black. Pattern reversal potentials are thought to be evoked by changes in stimulus contours. Because of its reliability, the PREP has been widely used for clinical applications.

The P300 is a positive long latency (200-500 msec) component, reflecting a more central, information processing phenomenon. The P300 is believed to represent a process of cognitive evaluation of stimulus significance, or attention (Beck, Swanson, & Dustman, 1980; Callaway & Harris, 1974; Podlesny & Dustman, 1982; Silverman, 1970). Typically, the subject is instructed to attend to one of two similar visual flash presentations which are randomly intermixed, e.g., count

the "Xs" and ignore the "Os." Although the P300 has been reported for other sensory systems, this paper will confine discussion to the visual system.

Electrophysiology

Electroencephalography, the recordings of the electrical activity of the brain, uses modern equipment and techniques to contribute knowledge to the scientific understanding of brain function. The EEG measures spontaneous electrical activity, whereas the evoked potential measures response to sensory stimulation. Physiologically, nerve fibers carry impulses which are then transmitted across synaptic membranes to the cell structure. The normal, healthy brain is estimated to contain approximately 6×10^9 neurones, so there is a considerable volume of cortical electrical activity (Goff, 1974). The evoked potential is considered indicative of the neural activity of the brain involved in the processing of sensory input.

A scalp electrode is a small metallic disc attached to the scalp with an adhesive called collodion. The electrode acts as a conductor between the physiological electrolyte of tissue and the recording circuitry. Electrodes, applied at precise positions, record the activity arising from the electrical

field near that placement. Thus, electrodes positioned at different scalp locations measure the electrical activity from different brain regions. Specific sites on the scalp have been identified which typically correspond with certain cortical functions. These are then abbreviated: e.g., Fz = frontal; Cz = central; C3 = central/left; Pz = parietal; Oz = occipital; "z" is an abbreviation of "zentral," the German word for central. Scalp electrodes used in this study (Figure 1) were positioned according to the International "10-20" System (Jasper, 1958) designed to permit conformity among laboratories.

When waveforms are measured and evaluated, specific points are common to each sensory parameter. Specific latency and amplitude conventions apply to evoked potentials (see Figure 2). For the brainstem auditory evoked potential (BAEP), response peaks are identified on a I-VII basis. Evoked potential measures of the visual system used for this study are the visual evoked potential, the pattern reversal evoked potential, and the P300 evoked potential. Response waveforms for each of these measures are slightly different. In this study, VEP components are referred to as N80, P100, N130, and P200 for latency (e.g., N80 = a negative wave whose peak was identified at approximately 80 msec after stimulus presentation), and N80-P100, P100-N130, N130-P200 for amplitude (e.g., N80-P100 = the peak-to-trough difference between N80 and P100). Similarly, PREP components are referred to as N70,

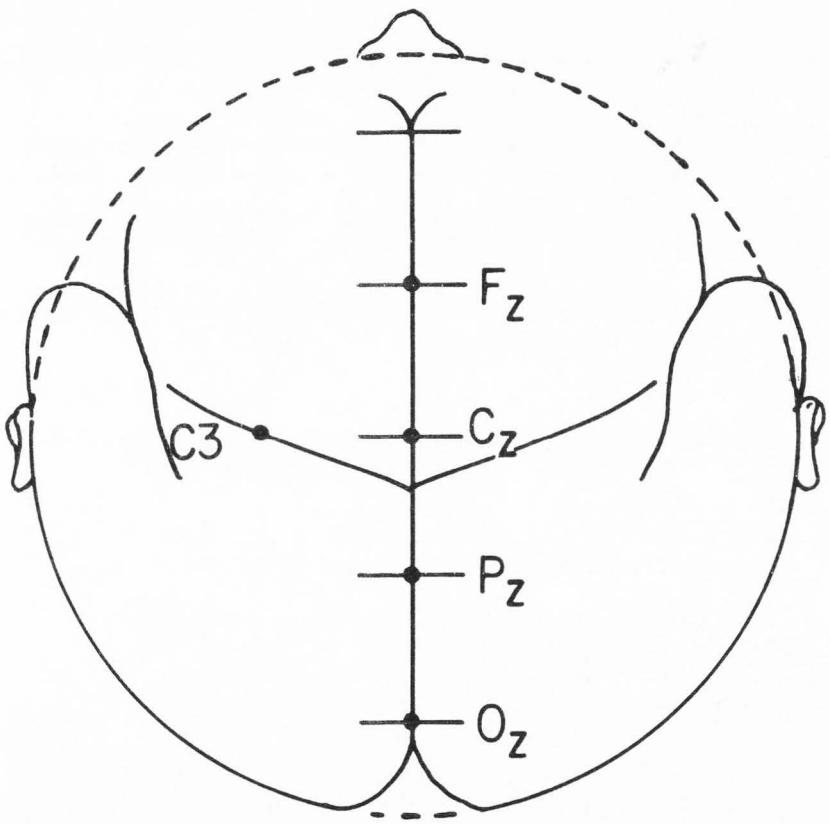

Figure 1. EEG and evoked potential recording sites.

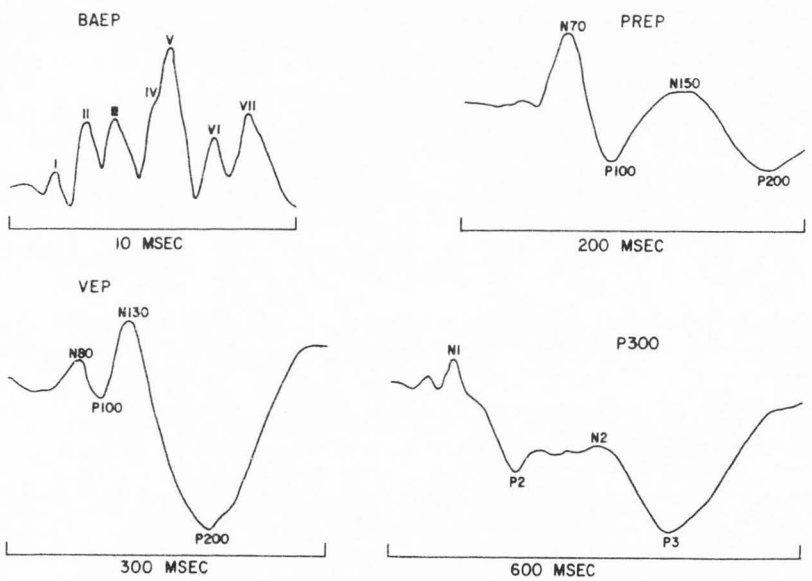

Figure 2. Types of evoked potentials analyzed.

P100, N150, and P200 for latency, and N70-P100, P100-N150, and N150-P200 for amplitude. Response components for P3 latency are labelled P1, N1, P2, N2, and P3 (e.g., P1 = the first identifiable positive peak, usually near 100 msec following stimulus presentation), and P1-N1, N1-P2, P2-N2, and N2-P3 for amplitude (e.g., the peak-to-trough difference between P1 and N1).

Definition of Terms

This glossary has been compiled to clarify terminology. It is not meant to be an authoritative source, but rather an adjunct to and clarification of the present text.

Amplitude. A measure of the difference in voltage between a prestimulus baseline and a given evoked potential peak, or between one EP peak and the next peak of opposite polarity (usually expressed in microvolts).

Amplitude/Intensity Slope. the line of best fit when plotting EP amplitude against stimulus intensity.

Augmenter. an individual who responds to an increasing stimulus intensity with an increase in evoked potential amplitude.

BAEP. brainstem auditory evoked potential; the electrical reactivity of auditory brainstem structures to sound stimuli

measured at the scalp.

CNS. the central nervous system; the brain and spinal cord.

Collodion. adhesive fluid used to attach electrodes to the scalp for EEG recording.

Cortical Coupling. a measure of phase relationships between EEG patterns from two different cortical areas; a computed information-transmission measure which compares actual vs. expected cell frequencies in a matrix of contingencies (4 X 4 contingency table) specifying polarity (+, -) and direction of movement (rising, falling).

EEG. electroencephalogram; record of spontaneous electrical activity of the brain.

EP. evoked potentials; stimulus-specific, time-locked signals embedded in EEG recordings of brain waves.

International "10-20" System. system of standardized scalp electrode placement determined by measuring the head from external landmarks; recommended by the International Federation of Societies for Electroencephalography and Clinical Neurophysiology.

Latency. time interval between stimulus presentation and a given evoked potential peak (expressed in msec).

P300. positive long latency component (200-500 msec); thought to express a psychological component of information processing, e.g., attention.

Power Spectral Analysis. a method of EEG analysis

employing the fast Fourier Transformation to describe the amount and amplitude of activity in specified frequency bands.

PREP. pattern reversal evoked potential; elicited by a visual presentation of a black and white checkerboard pattern which alternates at a fixed rate; i.e., black squares become white, and white squares become black.

Reducer. individual who responds to an increasing stimulus intensity with a decrease, or relatively less increase, in evoked potential amplitude.

VEP. visual evoked potential; the electrical reaction of the brain to visual stimuli, measured at the scalp.

Problem Context

There are two major reasons to study individual differences in electrophysiological responses: (a) a general contribution to scientific knowledge, and (b) a concern for accurate normative data for clinical application. Descriptions of age and sex differences can be helpful in understanding both structure and function of central neural processing. Although pertinent research literature is reviewed later, summary statements regarding age and gender related evoked potential differences can be made.

Age

Results from developmental electrophysiological research highlight life-span changes. A variety of sensory and psychophysiological decrements have been associated with senescence (Botwinick, 1981). In general, age-related evoked potentials changes appear to parallel maturation in youth and decline of function in old age. Because aging rates seem to differ in females and males, a gender X age interaction also seems important to investigate.

Sex

Some electrophysiological studies have noted gender differences both in children and in oldsters (Schenkenberg, 1970). Although there is evidence suggesting that sex differences occur across age on a variety of measures, (McGlone, 1980; Mochizuki, Go, Ohkubo, Tatara, & Motomura, 1982), relatively little evoked potential research has addressed the period of young adulthood. Gender seems to be a significant differentiating EP variable which has received only recent attention.

The relationship of brain to behavior is complex, being influenced by biological, psychological, and sociological factors. EEGs and evoked potentials provide safe, noninvasive

research tools for the study of the neuroelectric responses to sensory stimulation. Recorded electrical activity in response to stimulation reflects neuroanatomical generators, sensory coding, and cognitive processes. Several different techniques and measures have been developed to identify and describe these levels of information processing. As an example, isopotential mapping (Donchin & Lindsay, 1969) illustrates the topographical nature of the brain's response to stimuli. Not surprisingly, correspondence between scalp and cortical recordings has demonstrated that specific areas of the brain react differentially to stimulus presentation. Separate sensory modalities show specific evoked potential characteristics, allowing theoretical interpretation for neural substrates and for cognitive processing.

Nevertheless, many methodological and theoretical questions remain unanswered. The large number of variables which may influence the evoked potentials of human subjects is difficult to quantify and control. Variation in methodology resulting from different laboratory conditions and different experimental paradigms makes interpretation more difficult. Theoretically, in order to make equivalent comparisons, similar subjects (age, sex, health, race, handedness, socio-economic status, etc.) must experience similar experimental procedures, including screening tests, time and duration of testing, sensory system stimulated (auditory, visual, somatosensory). There must also be equivalence among scalp recording sites

(e.g., frontal, central, parietal, occipital) and stimulus parameters (intensity, frequency, interstimulus interval, etc.). A recent review noted that cross-modal comparisons are "essentially impossible" due to an "astounding lack of consistency and inadequate concern" for consistency in electrode recording site (Goff, Matsumiya, Allison, & Goff, 1969, p. 96). Additional studies with careful subject selection and thoughtful design can clearly refine the existing body of knowledge.

Problem Statement

The present research sought to identify that portion of EEG and evoked potential variability attributable to sex and aging differences. Sex differences in young female and male adults (25-35 years of age) were assessed; the results were compared with those of matched older females and males (55-70 years of age).

The primary purpose of this study was to determine the extent to which normal adult (young and old) females and males differ in CNS information processing as measured by EEG and evoked potentials. This experimental variable was considered a sex difference factor.

The secondary purpose of this study was to investigate the

extent to which normal (female and male) young and old adults differ in CNS information processing as measured by EEG & evoked potentials. This experimental variable was considered an aging factor.

Hypotheses

Primary Hypothesis

No significant differences are expected between normal female and male adults for EEG measures of cortical coupling and power spectral analysis, or for evoked potential measures of amplitude and latency of brainstem auditory evoked potentials (BAEPs), visual evoked potentials (VEPs), pattern reversal evoked potentials (PREPs), or P300s.

Secondary Hypothesis

No significant differences are expected between normal young and older adults for EEG measures of cortical coupling and power spectral analysis, or for evoked potential measures of amplitude and latency of brainstem auditory evoked potentials (BAEPs), visual evoked potentials (VEPs), pattern

reversal evoked potentials (PREPs), or P300s.

A brief survey of pertinent electrophysiological research will provide a more detailed context for conceptualizing the present investigation.

CHAPTER II

REVIEW OF LITERATURE

Many researchers share the view that the evoked potential offers a unique opportunity to observe cortical events in the intact brain (Barber, 1980). Seen as a "window to the brain," the evoked potential furthers our understanding of the relationships between electrophysiology and function. As a research technique, the advantage of the evoked potential over the EEG is two-fold: first, it yields a clearer signal with less background noise contamination; and second, discrete stimuli can be used to probe primary receiving areas and separate sensory systems (Coppola, Tabor, & Buchsbaum, 1978; Courjon, Mauguiere, & Revol, 1982; Donchin & Lindsley, 1969; Perry & Childers, 1969).

Evoked potentials have provided stimulus-specific information with clinical applications. However, research results have not been altogether consensual. Differences in laboratory conditions, sensory modalities, and experimental paradigms affect the evoked potential response. Furthermore, individual subject differences such as age and sex produce variations within normal subject populations (Begleiter, 1979; Callaway, Tueting, & Koslow, 1978; Perry & Childers, 1969; Regan, 1972). Defining the range of response considered within

normal limits is obviously important to minimize false negatives and false positives in clinical assessments.

Age

There is overall agreement that humans change developmentally. A slowing of brain function is associated with aging (Everett, 1971; Obrist, 1976). Kenney's (1982) synopsis provides general background information on the physiology of aging. Documented areas of brain deterioration include: memory, reaction time, brain volume index (atrophy), cerebral blood flow, metabolic rates, oxygenation, neuronal circuitry, inhibitory function, and sensory acuity (Birren, 1964; Botwinick, 1981; Cotman & McGaugh, 1980; Hatazawa, Ito, Yamaura, & Matsuzawa, 1982; Woodruff & Birren, 1975). In his review article, Dustman (1984) typified evoked potential responses of the elderly as slower and larger than those of younger adults, thus paralleling other age-related changes.

Sex

Most frequently, females are reported to demonstrate shorter latency and greater amplitude evoked potentials than males, i.e., females respond with bigger, faster potentials

(Beaumont & Mayes, 1977; Buchsbaum & Pfefferbaum, 1971; Buchsbaum, Landau, Murphy, & Goodwin, 1973; Celesia & Daly, 1977; Kjaer, 1979, 1980b; Perry & Childers, 1969; Schenkenberg, 1970; Schenkenberg & Dustman, 1970; Shagass, 1972; Shagass & Schwartz, 1965; Shearer, Cohn, Dustman, & La Marche, 1984). Known gender differences which may be relevant include: smaller head size, brain mass, and skull thickness in women; higher deep body temperature in women; shorter female anatomical pathways; higher basal metabolism and cerebral blood flow in women with differing hormone and CNS maturational rates; larger female splenium and left planum temporale; and, in women, greater amounts of MAO platelets and plasma, associated with catecholamine degredation. (Blatter, 1982; Buffery & Gray, 1972; Denno, 1982; de Lacoste-Utamsing & Holloway, 1982; Gur et al., 1982; Hatazawa et al., 1982; Hutt, 1972; McGeer, Eccles, & McGeer, 1978; McGlone, 1980; Wittig & Peterson, 1979). The relationship of function to structure is still unclear, however. Several investigators found physical variables insignificant: Buchsbaum, Henkin, and Christiansen (1974) for gonadal steroid secretions; Dustman and Beck (1965) for head shape or size; Kjaer (1979) for total body weight; Kooi and Bagchi (1964) for pupil diameter; and Ikuta and Furuta (1981) and Shagass (1972) for length of conduction pathways.

In this chapter, studies which have addressed the variables of age and sex are described. For organizational clarity, these are grouped according to the sensory parameter

measured, i.e., EEG measures of power spectral analysis and cortical coupling, brainstem auditory evoked potentials (BAEPs), visual evoked potentials (VEPs), pattern reversal evoked potentials (PREPs), and P300.

EEG

Developmental research has shown that early periods of childhood are marked by individual age and gender related variability which gradually decreases in adolescence. A diminishing trend of age and sex differences may reflect variable rates of maturation which essentially disappear in adulthood. Such a developmental tendency toward homogeneity, or normalization of the EEG, is described by Eeg-Olofsson (1971, 1980).

A gradual EEG slowing, particularly in alpha frequencies, is characteristic of the aging process (Rodin, Grisell, Gudobba, & Zachary, 1965). Some age-related EEG changes may begin as early as the fourth decade and appear to accelerate by the seventh decade, although many oldsters in good health may show no EEG abnormalities (Friedlander, 1958; Mankovsky & Belonog, 1971).

Individual subject differences such as gender are minimal during young adulthood. In the elderly population, however,

males have exhibited slightly lower mean alpha frequencies than females (Michalewski, Thompson, Patterson, Bowman, & Litzelman, 1980).

Two methods of analyzing EEG tracings which appear to yield new information are the computerized techniques of power spectral analysis and cortical coupling.

Power Spectral Analysis

As developed by Cooley & Tukey (1965) and Gold & Rader (1969), power spectral analysis is computed using fast Fourier transformation techniques. Segments of EEG are analyzed to provide an estimate of the amount and amplitude of specific frequency components, most commonly in the alpha frequency band (Barber, 1980; Marmarelis & Marmarelis, 1978; Yingling, 1977). Beaumont, Mayes, and Rugg, (1978) and Fiore (1978) have used power spectral analysis to assess asymmetry during cognitive tasks.

Cortical Coupling

Cortical coupling has been previously used as a measure of relationship between EEG and brain information processing. A comparison across time of EEG patterns from two cortical sites,

based on polarity and change in direction of polarity, results in a measure of coupling. This time dependent correspondence is considered representative of active functional communication between two areas of the brain. For example, cortical coupling techniques have been used to evaluate areas of cognitive involvement during performance tasks (Callaway & Harris, 1974; Yagi, Bali, & Callaway, 1976; Yingling, 1977).

Brainstem Auditory Evoked Potential (BAEP)

The brainstem auditory evoked potential (BAEP) measures the first 10 msec of auditory brainstem responses to sound stimuli. Clinically, BAEPs have been used to reflect abnormalities in brainstem functioning for neurological patients (Friedreich's ataxia, Charcot-Marie-Tooth, and olivo-ponto cerebellar atrophy), to localize suspected lesions (neuromas, leukodystrophy), to assess function in comatose patients (drug overdose, brain death), and to manage uremia and dialysis regimen (Chiappa & Ropper, 1982; Creel, Spekreijse, & Reits, 1981; Kandel & Schwartz, 1981; Kelly, 1981; Kjaer, 1979; Kooi, 1979; Lewis, Dustman & Beck, 1978).

In general, the majority of BAEP clinical and research reports have historically neglected individual subject differences. Jerger and Hall (1980) reviewed the literature to

compile a total N of 617 subjects evaluated in published studies. Of these, only 19% (120) were even identified by sex or age. Only recently have age and gender provided further insight into BAEP variability. Those studies which find significant differences receive current attention.

Published reports for age and gender effects are relatively consistent. Characteristically, females demonstrated larger amplitude, shorter latency responses than males (Beagley & Sheldrake, 1978; Campbell et al., 1981; Jerger & Hall, 1980, Kjaer, 1980a; Michalewski et al., 1980; Mochizuki et al., 1982). Older subjects most often had responses which were smaller in amplitude and longer in latency than those of young adults (Jerger & Hall; Kjaer).

Although these trends appear somewhat consensual, note that many studies only partially replicated results of previous research. Some found a latency difference but no significant difference in amplitude, or vice versa. Also, there was diversity in waveform measurement. The most often reported BAEP component was Wave V, although this varied from study to study. It may be important to note which of these waves was studied when reading reports of BAEP findings.

For latency comparisons, several studies used the interpeak latency (IPL) which is commonly accepted as a measure of brainstem neural transmission time (Fabiani, Sohmer, Tait, Gafni, & Kinarti, 1979). Interpeak latency is a particularly useful comparator because it is not affected by conductive

hearing loss (Otto & McCandless, 1982; Rowe, 1978), body length (Kjaer, 1979) or head size (Edwards, Squires, Buchwald, & Tanguay, 1983). IPLs are obtained by subtracting latency of Wave I from III, I from V, or III from V. As an impressive indication of the reliability of this measure, Rowe compared conduction times from four laboratories: a mean peak IPL (I-V) of 4.0 msec was reported from each. Kjaer (1980a) reviewed published norms from four other laboratories that reported mean peak IPLs averaging 4.1 msec (I-V).

Age

The smaller, slower BAEP response pattern associated with aging has been reported by Kjaer (1980a). Similar findings have been reported by Wedel (1979) for Waves I and IV; by Patterson et al. (1981) for Wave III, by Jerger and Hall (1980) for Wave V, and by Allison, Wood, and Goff (1983), Beagley and Sheldrake (1978), and Rowe, (1978) for interpeak latencies (IPLs). Harkins (1981) studied young versus elderly females and found delayed peak latencies for the older subjects for all components, but no IPL differences. Otto and McCandless (1982) found no significant age-related IPL differences.

Sex

Gender was found to affect BAEP latency by Allison et al. (1983), and Kjaer (1980a). Wave-specific gender differences have been reported for waves IV and V (Patterson et al., 1981), for Wave V (Jerger & Hall, 1980; McClelland & McCrae, 1979; Michalewski et al., 1980) and for IPLs (Allison et al., 1983; Beagley & Sheldrake, 1978; Campbell et al., 1981; Edwards et al., 1983; Mochizuki et al., 1982; Stockard, Stockard, & Sharbrough, 1978; Stockard, Stockard, Westmoreland, & Corfits, 1979). All results indicated shorter latencies for females. Wedel (1979) found no significant sex differences for BAEP latency, but did find amplitudes for females greater than those for males. Others reporting similar amplitude findings include Kjaer, Jerger and Hall, and Mochizuki et al. for Wave V; Michalewski et al. for waves IV-VII; and Beagley and Sheldrake, Campbell et al., and Edwards et al. for IPLs.

Visual Evoked Potential (VEP)

The visually evoked potential, probably the most systematically studied of the EPs, has proven to be a reliable measure of central nervous system response (Begleiter, 1979; Dustman & Beck, 1965). Clinically, VEPs have been used to

diagnose suspected problems such as infant visual acuity, visual field defects, optic neuritis, and compression of anterior visual pathways (Regan, 1972). Although the pattern reversal and the P300 evoked potentials are also responses to visual stimuli, these measures are considered separately.

Although some generalized conclusions regarding the effect of age and gender on VEPs are understood, there are complicating factors. For instance, significant findings are often only partially replicated, e.g., for latency but not for amplitude. Another factor is the physical parameters which may differ from laboratory to laboratory. For example, some methods have used the same absolute flash intensity for all subjects while others have varied intensity according to individual subject's visual threshold to insure the same receptive intensity, or retinal illumination (Dustman, Snyder, & Schlehuber, 1981).

Recording site must also be considered in comparing VEP studies. In particular, VEPs recorded from anterior scalp have been shown to differ significantly from those recorded from posterior areas (Dustman, Shearer, & Snyder, 1982; Dustman et al., 1981).

Again, the EP response component measured and reported is important to note. The VEP P100, believed to reflect activity of visual cortex, has been widely used diagnostically (Halliday, Barrett, Carroll, & Kriss, 1982).

Age

In general, life-span changes in evoked potentials are thought to be related to maturational CNS development in youth and to the decline of CNS function in senescence. An important factor when reviewing aging literature is the actual age range studied. For example, Buchsbaum et al. (1974) reported that amplitude for P100-N140 and N140-P200 decreased with increasing age, but careful reading indicates that the subjects were aged 6-40 plus years. When some reviewers cited a decrease in amplitude associated with increasing age, they may have been referring to maturational studies of infants or adolescents (Perry & Childers, 1969).

Reviewing results from a 20-55 year old adult population, Allison et al. (1983) related that most laboratories have found no VEP latency changes attributable to aging. However, latency does appear to correlate positively with age when wider age spans are studied. For example, when subjects past the sixth decade were compared with young adults, younger subjects responded with shorter latencies (Beck & Dustman, 1975; Dustman, Schenkenberg, Lewis, & Beck, 1977; Kooi & Bagchi, 1964; Mintz, Tomer, Radwan, & Myslobodsky, 1981; Ordy & Brizzee, 1979; Schenkenberg & Dustman, 1970; Shagass & Schwartz, 1965; Straumanis, Shagass, & Schwartz, 1965).

Nevertheless, some investigations of the relationship between age and VEP amplitude have produced apparently

contradictory reports. For example, Buchsbaum et al. (1974) reported that amplitude decreased with age; Kooi and Bagchi (1964) stated that amplitude increased with age. Dustman et al. (1977) offered some resolution to the relationship of amplitude and aging. Their findings revealed that the amplitudes for oldsters were higher for early VEP waves, while the reverse occurred for later VEP waves. Contrasting reports regarding the VEP amplitude response for oldsters may be accounted for by amplitude increase in early components but attenuation in later components. Therefore, the response component reported may influence published research conclusions.

Investigation into the topography of the visual response showed an age difference which was more pronounced at the brightest intensity and was localized to recordings from visual cortex (Dustman et al., 1981). Furthermore, VEP amplitude responses elicited by patterned stimulation were greater than those elicited by unpatterned stimulation (see below) when analysis was calculated for occipital electrode site, while the opposite effect occurred for frontal and central electrode sites. The additional factors of stimulus intensity and electrode recording site must also be considered in evaluating and comparing visual evoked potential results (Dustman et al., 1982).

Sex

Latency for females has been shown to be shorter than that for males by Shagass and Schwartz (1965), Schenkenberg (1970), and others. Studies of VEP amplitudes indicated that females produced larger amplitude responses (Barber, 1980; Beaumont & Mayes, 1977; Buchsbaum et al., 1974; Dustman et al., 1977; Perry & Childers, 1969; Rodin et al., 1965; Schenkenberg & Dustman, 1970; Shagass & Schwartz, 1965). Thus, the typical female VEP response might be described as larger and faster than that for the typical male.

There are two further topics of interest associated with VEP responses: amplitude/intensity slope, and differences in VEPs elicited by patterned versus unpatterned flashes.

Amplitude/Intensity (A/I) Slope

The VEP amplitude/intensity slope is the line of best fit when plotting amplitude across stimulus intensity. A/I slope has been computed for the several sensory modalities, although discussion here relates to the visual system. Individuals who responded to increasing stimulus intensity with an increase in EP amplitude have been termed "augmenters;" those who responded with a decrease or a comparative lack of increase in EP amplitude have been called "reducers" (Silverman, Buchsbaum, &

Henkin, 1969). Amplitude reduction has been interpreted as reflective of a central inhibitory feedback mechanism, which theoretically serves to protect sensory cortex from overstimulation (Zuckerman, Murtaugh, & Siegel, 1974).

Increased catecholamine levels correlate with advanced age (McGeer, 1981; Vaccari, 1980). Robinson (1975) found a significant positive correlation between monoamine oxidase plasma and blood platelets: both increased with advanced age. As would be expected, the extremes of the age span (youngsters and oldsters) have relatively lower levels of catecholamines (McGeer & McGeer, 1980; Robinson et al., 1977), demonstrate reduced inhibitory functioning (Scheibel & Scheibel, 1975) and also show augmentation for VEP amplitude (Buchsbaum, Haier, & Murphy, 1977; Cohn, 1983; Dustman et al., 1981, 1982; Schafer & McKean, 1975). Interestingly, in the two studies conducted by Dustman and his colleagues, response augmentation occurred specifically for anterior rather than visual areas, contributing additional evidence for an inhibitory deficit hypothesis (children with immature frontal cortex and oldsters with deteriorating frontal cortex).

Although some authors have extended the augmenter/reducer dichotomy to include psychological descriptions, the present report confines concern to neurophysiology.

Shagass (1972) reported that females had higher amplitude/recovery curves than males, which is believed to reflect a sex difference in cortical inhibition. Gender has

yet to be thoroughly researched as a variable in this measure. Although some studies included males and females, most were not numerically and/or age matched for sex comparisons (Buchsbaum & Pfefferbaum, 1971; Connolly & Gruzelier, 1982; Iacono, Gabbay & Lykken, 1982; Knorring & Perris, 1981; Raine, Mitchell, & Venables, 1981; Soskis & Shagass, 1974).

The recording site used for response measurement is a matter of debate. Buchsbaum and Pfefferbaum (1971) reported that amplitude reduction to increasing intensity of visual stimuli does not occur at occipital sites, and so they used a vertex lead (Cz). However, Dustman et al. (1981) measured for occipital scalp (Oz), and found that 4-12 year old boys tended to be reducers, while male adolescents and male adults aged 14-90 years tended to be augmenters.

VEPS Elicited by Patterned versus Unpatterned Stimuli

From Hubel and Wiesel's (1962) initial nobel-prize winning studies of single cells in animal striate cortex, theories have been developed which view the visual cortex as a "kind of spatial Fourier analyser" which is sensitive to lines, edges, and contours, and processes information such as position, orientation, contrast, length and width of stimuli (Marr & Hildreth, 1980, p. 187). As might be expected, VEPs elicited by patterned flashes do differ from those elicited by

unpatterned flashes (Beck, 1975; Regan, 1972).

The responsiveness of the visual system to patterned stimuli can be measured by comparing VEPs elicited by patterned (checkered) flashes with VEPs elicited by unpatterned (diffused) flashes. A relative measure of similarity of VEP waveforms can be determined by correlating the digital values of the patterned VEP with the corresponding digital values of the unpatterned VEP. A relatively large correlation reflects greater similarity, or less differentiation, between responses elicited by patterned and unpatterned stimuli. Dustman et al., (1981) examined 211 healthy males aged 4-90 using this method. They found that life-span effects followed a U-shaped curve wherein the young adults had lower correlations, or greater differentiation, than the very young or the very old. They hypothesized that VEP responses were most alike during childhood and old age due to reduced inhibitory function, and that the greater differentiation observed in young adulthood was attributable to enhanced inhibitory surround effects in the visual system, apparently mediated by monoamine levels (Schafer & McKean, 1975).

Another finding of the Dustman et al. (1981) study was a topographical analysis. Unlike the mean correlations for occipital scalp recordings, reported above, the mean correlations for frontal and central scalp recordings showed no significant adult age effects. Comparing VEP amplitude, they further noted that unpatterned flash stimulation produced

greater response amplitude for central electrode sites, and that patterned flash stimulation produced greater response amplitude for occipital electrode sites. They theorized that these topographical differences related to differences in function (visuo-spatial functioning for central scalp vs. line, edge, and contour detection from occipital scalp).

Gender has yet to be investigated by this measure.

Pattern Reversal Evoked Potential (PREP)

The pattern reversal evoked potential is elicited by a visual presentation of black and white squares in a checkerboard pattern which alternate at a fixed rate; i.e., black squares become white, and white squares become black. Halliday, McDonald, and Mushin (1973), Kjaer (1980b), and others have employed the PREP as a tool for diagnosing multiple sclerosis; Sokol (1978) has used the PREP in evaluating infant visual acuity; Bodis-Wollner and Yahr (1978) for assessing Parkinson's disease; and Shearer, Snyder, and Dustman (1984) for investigating uremic dysfunction and renal hemodialysis. Further clinical applications can be found in Chiappa and Ropper's (1982) review article.

Research reports include both age and sex effects. As with other evoked potential literature, while some generalized

results are consistently reported, there remains considerable discussion within more specific parameter comparisons. Furthermore, age and sex effects reported by each study may vary according to the waveform component examined. The P100 has most commonly been used for clinical applications.

Age

In general, oldsters can be characterized as responding to pattern reversal stimulation with slower, smaller waveforms than young adults (Celesia & Daly, 1977; Kjaer, 1980b; Kriss et al., 1982; Shearer & Dustman, 1980; Sokol, Moskowitz, & Towle, 1981). However, closer inspection of results indicates a more complex picture. For example, Celesia and Daly found longer latencies for oldsters only for waves corresponding to N70 and P100; no age related amplitude differences were found. Shearer and Dustman discussed their results in terms of two separate patterns: (a) a P50, N65, P100 early wave pattern where latency increased and amplitude decreased with increased age, and (b) an N150, P200 late wave pattern where latency and amplitude both decreased with increased age.

Sex

Females can be typified as demonstrating faster, larger PREP responses than males (Halliday et al., 1982; Kjaer, 1980b; Kriss et al., 1982; Shearer & Dustman, 1980; Stockard, Hughes, & Sharbrough, 1979). Again, individual studies specified some discrepancies. For example, Stockard et al. reported latency as shorter for females, but just for P100. Halliday et al. found similar latency results, but reported significance for the P100 component only. In the Shearer and Dustman study, females displayed shorter latency for P50 only; amplitude was greater for N65-P100 and P100-N150. This amplitude result was similar to that reported by Halliday's report of amplitudes greater for females than males for P100.

A further complication is the possibility of gender differences associated with aging. Analysis by Halliday et al. (1982) showed that, for an early PREP wave, latency for females increased with advanced age, but males showed no comparable significant age effects.

P300

The P300 (positive component, 200-500 msec in latency) commands interest due to its uniqueness as a psychological

component of information processing. Because of its relative independence from the actual stimulus presentation, the P300 has been referred to as an endogenous rather than exogenous component (Beck et al., 1980). Halgren et al. (1980) discussed the hippocampal formation as the neurogenerator for the P300, while Wood, Allison, Goff, Williamson & Spencer (1980) related P300 activity to hypothalamic functioning; Yingling & Hosobuchi (1984) suggested a more medial, possibly thalamic source. There has been some evidence that the P300 may represent a late positive complex, or LPC, suggesting a more complex, possibly interactive, family of component sources (Friedman, Vaughan, & Erlenmeyer-Kimling, 1981). A two-process theory was postulated by Beck et al. : a tonic arousal state wherein the mesencephalic reticular formation is activated by mediothalamic-frontalocortical connections, and a phasic discriminative state which additionally implicates an inhibitory gating mechanism such as might be found in the nucleus reticularis. Their P300 latency data showed greater peak delay for the elderly which they related to age-associated decrements in neural circuitry and cognitive behavior, particularly for frontal cortex.

Because stimulus conditions are exactly the same for background and target flash presentations (e.g., "O"s vs. "X"s), any difference in P300 waveform response may be attributed to the cognitive effect of instructing the subject to attend to the target presentation (e.g., to count the "X"s).

Thus, the P300 has been considered reflective of attention or information processing (Beck et al., 1980; Desmedt & Debecker, 1979; Donchin, 1979; Podlesny & Dustman, 1982; Skrandies, 1983). Although the late wave response has been reported for other sensory systems, this review will confine discussion to the visual system.

Several authors have documented increased P300 peak delay associated with increased age (Beck et al., 1980; Goodin, Squires, Henderson, & Starr, 1978; Pfefferbaum, Ford, Wenegrat, Roth, & Kopell, 1984). Podlesny and Dustman (1982) noted a pattern of decreased amplitude with aging. An increase in latency as well as a decrease in amplitude typified the aging pattern found by Picton, Stuss, Champagne, and Nelson (1984).

P300 results appear to have been complicated by experimental differences in task instruction. For example, Ford, Pfefferbaum, Tinklenberg, and Kopell (1982) evaluated process time needed for a decision task using a visual memory retrieval task and requiring a single finger-pressing response for target recognition. Citing this and previous studies, they concluded that P300 latency was delayed with age. Similarly, Beck et al. (1980) reported prolonged latency associated with aging and no amplitude-age effects. In contrast, Podlesny and Dustman (1982) found no latency-age effects, but reduced amplitude was associated with aging. It seems noteworthy that in the Podlesny and Dustman study a "ready" signal was given before presentation and the subject responded to the stimulus

by selectively pressing one of two switches to identify target versus background flashes. Further investigation into P300 age effects seems necessary.

P300 gender effects, amplitudes for females greater than those for males, have only recently been reported (Picton et al., 1984). Studies which specify age and sex effects for P300 have thus far been neglected. For example, the Ford et al. (1982) study used all female subjects, whereas Beck et al. (1980) and Podlesny and Dustman (1982) studied all male subjects.

CHAPTER III

METHOD

Subjects

Eighty subjects participated in this study. There were four subgroups (young females, old females, young males, and old males) of 20 subjects each. "Young" adults were aged 25-35 years ($\bar{X}$ = 28); "old" adults were aged 55-70 years ($\bar{X}$ = 60). Males and females were carefully matched for selection descriptors; all subjects were right-handed Caucasian paid volunteers with at least a high school education. Applicants were excluded if they reported a history of sensory impairment, neurological disease, or psychological disorder. Subjects chosen for participation were thus considered normal, healthy individuals.

Apparatus

Instrumentation can be categorized as calibrating, stimulating, recording, and data processing equipment.

Routine calibration insured consistent frequency and amplitude characteristics of recorded signals. Calibration was

accomplished by passing a 20 Hz, 100 uv sine wave through the recording and data processing systems. A computer monitored the end stage of these signals and was programmed to detect variations in amplitude among the recording channels and to correct for variations. The following equipment was used for calibration: a Hewlett Packard (HP) Oscillator (Model 204C), an HP Attenuator (Model 350D), an HP Microvoltmeter (Model 3410A), a Sony Tektronix Oscilloscope (Model 323), and a Grass Impedance Meter (Model EZM).

Recording and stimulating equipment consisted of: a Grass (Model 78B) 8-channel EEG/polygraph, an HP (Model 3968A) 8-channel magnetic instrumentation tape recorder, a Grass Photic Stimulator (Model PS22), a Grass Multi-stimulator (Model S10SCM), a Grass Auditory Stimulator (Model S10ASCM), and a 23" Ball TV Monitor. All electrophysiological recordings were made with the 8-channel Grass EEG/polygraph. Lower and upper band pass settings were as follows: EEG, VEPs, and PREPs at 1-100 Hz; BAEPs at 30-3000 Hz; and P300 at .1-100 Hz.

Data processing equipment included a Terak (LSI-11) computer with 64K of 16 bit words. The computer was interfaced with flexible and hard disk storage, with video terminals for both accessing the computer and presenting visual stimuli to subjects, with analog-digital and digital-analog converters, and with other devices which permitted accurate and rapid handling of electrophysiological data.

Procedure

Classification of Subjects

Subjects were recruited from voluntary response to local advertisement. Data from 20 females and 20 males aged 55-70 years which had been previously recorded in the Neuropsychology Research Laboratory at the Salt Lake City Veterans Administration Medical Center was also included in the present study. These "oldsters" were matched with the young adults on all selection descriptors. Initial screening occurred during telephone responses to advertisement; more comprehensive data was acquired by questionnaire responses which provided physical data, including height, weight, head size, and physical, neurological, and psychological histories (see Appendix A).

Experimental Treatment

All subjects followed the same procedures (see Appendix B for procedural protocol). They were advised that there were no known risks associated with the procedures used, and they were required to read and sign the consent forms. Each subject spent approximately 3 hours total participation time, including initial screening and introduction to the laboratory,

electrophysiological measurements, and debriefing. Subjects were familiarized with the laboratory and then given a near visual acuity test (Bausch & Lomb Vision Tester, Model 14019). Minimum level for acceptance was 20/50.

Subjects were seated in a comfortable, padded chair in a light, sound, and temperature controlled, electrically shielded room. Disc electrodes were attached to the scalp with collodian at midline (Fz, Cz, Pz, and Oz) and left central (C3) sites, and referred to linked earlobes (A1, A2) according to the International "10-20" System (Jasper, 1958). Electrode impedances were below 5K ohms; eye movement artifact was monitored by electrodes attached to inner and outer canthi of the left eye. Prior to stimulus presentation, subjects were instructed to relax, remain still yet alert to the stimuli. The experimenter monitored EEG recordings for subject artifact, i.e., eye blinks, muscle tension, movement, etc., and provided verbal reminders to maintain the relaxed, attentive state.

EEG. The first procedure was to record three minutes of EEG while subjects relaxed with eyes closed. They were not stimulated during this time. EEG data was later analyzed by two methods: power spectral analysis and cortical coupling (see Data Analysis).

BAEP. The subject's auditory threshold, i.e., the lowest sound intensity at which the presence of stimulation was

reported, was obtained by asking for a verbal response of "on" or "off" to click presentations at decreasing intensities. Clicks were generated by the Grass auditory stimulus control module at a 70db sensation level (SL). With the lights off to enhance relaxation, 2,000 clicks were administered monaurally (to the ear with the best threshold) for about three minutes at an 11.3/sec rate. The first 20 responses were analyzed by computer to establish an average response; subsequent responses which were 2 standard deviations beyond this average were automatically rejected as artifact.

VEP. A viewing box, attached to and backlighted by the Grass photostimulator lamp, was positioned 40 cm from the subject's eyes to provide visual stimulation. Flashes of about 25 usec duration were visible through a 10 X 10 cm opening in the viewing box. Visual threshold, i.e., the lowest light intensity at which the subject could correctly report the orientation (left or right) of a narrow diagonal black line, was determined for each subject. The experimenter changed neutral density filters (which fit into the viewing box) to produce three luminance conditions, i.e., 1, 2, and 3 $\log_{10}$ steps above threshold. Two stimulus slides were employed: (a) a checkerboard patterned slide for patterned flash stimulation (each check edge was approximately 20' visual arc) and (b) an opaque slide was used for unpatterned flash stimulation. The two slides were approximately equivalent for percentage of

light transmitted. The checkerboard stimulus slide was used with 1, 2, and 3 $\log_{10}$ intensity filters; the diffuse stimulus slide was used with the 2 $\log_{10}$ intensity filter. Subjects received approximately 65 trials for each of four stimulus conditions (patterned flashes at three intensities, and unpatterned flashes at one intensity). The order of conditions was randomized across subjects. To enhance attention, subjects were instructed to depress a hand-held switch after every tenth flash. This manual response was automatically marked on the EEG record. The EEG was visually inspected for artifact, i.e., blinks, swallows, or other muscle activity. Fifty artifact-free trials from each stimulus condition provided the raw data for visual evoked potentials.

PREP. Binocular pattern reversal evoked potentials were elicited by presentation on the 23" Ball TV monitor of a checkerboard pattern which alternated at a fixed rate of 2/sec, that is, two times per second, black squares became white, and white squares became black. Maximum contrast checks measured 30' of visual arc. Subjects were instructed to focus on the center of the TV monitor while viewing the pattern reversal stimulation for 2.5 minutes. The first 20 responses served to establish an average; subsequent responses which exceeded 2 standard deviations beyond this average were automatically rejected as artifact. PREPs were averaged from 200 artifact-free single trial responses.

P300. For the P300, stimuli consisted of 200 letter presentations, 32 "X"s (target) and 168 "O"s (background). Each stimulus was generated by the Terak computer and displayed on the TV monitor for 40 msec with a 1-1.5 sec interstimulus interval (ISI). The "X"s and "O"s appeared in random sequence. To enhance attention, subjects were instructed to count the total number of target presentations, i.e., the number of times the "X" appeared on the screen, and to ignore the "O"s. Responses contaminated by artifact, apparent on visual inspection, were eliminated. The first 25 artifact-free target responses were averaged to provide P300 evoked potential data.

Debriefing. Any questions and/or concerns were discussed. Subjects were allowed to take a sample of their brain wave recording with them, as provided in the consent forms. As also agreed, a nominal payment of $25.00 was mailed to them.

Data Analysis

BAEPs were directly recorded and stored on hard disk; all other data was recorded and stored on the HP 8-channel magnetic instrumentation tape recorder for later off-line analysis. Recordings were played into the analog-digital converter interfaced to the computer which digitized, summed, and

averaged evoked potentials. VEPs were digitized at a 500/sec rate, PREPs and EEG for cortical coupling at a 250/sec rate, while EEG for power spectral analysis was digitized at a 128/sec rate. Averaged evoked potential and digitized EEG data were permanently stored on floppy disks. Averaged evoked potentials were digitally smoothed by computer to reduce the amplitude of fast activity without significantly altering the relatively slower EP latency and amplitude values. A digital plotter was used to reproduce averaged responses on graph paper. For all evoked potential data (BAEPs, VEPs, PREPs, and P300s), the experimenter measured component latencies and amplitudes (Shucard, Horn, & Metcalf, 1971). Component identification and measurement was reviewed by a person with years of experience in EEG and evoked potential recording. When necessary, adjustments were made to a criterion of consensus.

Statistical treatment is explained for each measure. Generally, analysis of variance was the statistic used. The Duncans Multiple Range Test was employed to test for differences among means when a significant main effect was found that involved three or more means (power spectral analysis, cortical coupling, visual evoked potential, and P300).

EEG

Power Spectral Analysis. Power spectral analysis was computed to provide a description of the amount and amplitude of activity within specified frequency bands. To simplify analyses, the 5-13 Hz range was examined in four bands of 5.25-7.00, 7.25-9.00, 9.25-11.00, 11.25-13.00 Hz with a resolution of .25 Hz. For ease of reading, frequency bands are referred to as 5-7, 7-9, 9-11, and 11-13 Hz. Consecutive four second segments of EEG recordings, which had been digitized and stored on disk, were retrieved for visual inspection on a computer terminal screen. Segments with artifact were eliminated. PSA was computed from the first 40 artifact-free segments using fast Fourier Transformation techniques. Mean PSA values were computed from those segments to provide comparisons of power in the various frequency bands. To better meet the assumption of normal distributiuons, $\log_{10}$ transforms of the PSA data for each frequency band were used for age X sex X area (Fz, Cz, Pz, Oz, and C3) ANOVAs.

Cortical Coupling. Cortical coupling was computed to measure phase relationships between EEG patterns from two different cortical areas. This time dependent relationship may be considered representative of active functional communication between two areas of the brain, i.e., an information-transmission measure.

Based on procedures used by Callaway and Harris (1974) and Yagi, Bali, and Callaway (1976) the Shannon-Weaver information transmission statistic was calculated for each of 100 successive blocks of 400 digital values of EEG from each pair of scalp electrodes. The two EEG patterns were compared on polarity (+, -) and change in direction of polarity (rising, falling). These comparisons were entered as tallies into a 4 X 4 contingency table (see Figure 3). Cortical coupling was computed from probabilities associated with the distribution of the 400 tallies across rows, columns, and cells. For a completely random distribution of tallies, cortical coupling = .00. For a perfect correspondence between two EEG signals, e.g., tallies are equally distributed across four diagonal cells and do not occur in other cells, cortical coupling = 2.00. A mean of the 100 cortical coupling values was used as the estimate of EEG correspondence for the present study.

Brainstem Auditory Evoked Potential (BAEP)

The electrical reactivity of auditory brainstem structures to sound stimuli was measured from the Cz. BAEPs were visually displayed on a computer terminal screen and major component latencies and amplitudes were measured. Interpeak latencies (IPLs) were computed from the latency measures. Age X sex ANOVAs were calculated for each of these three response

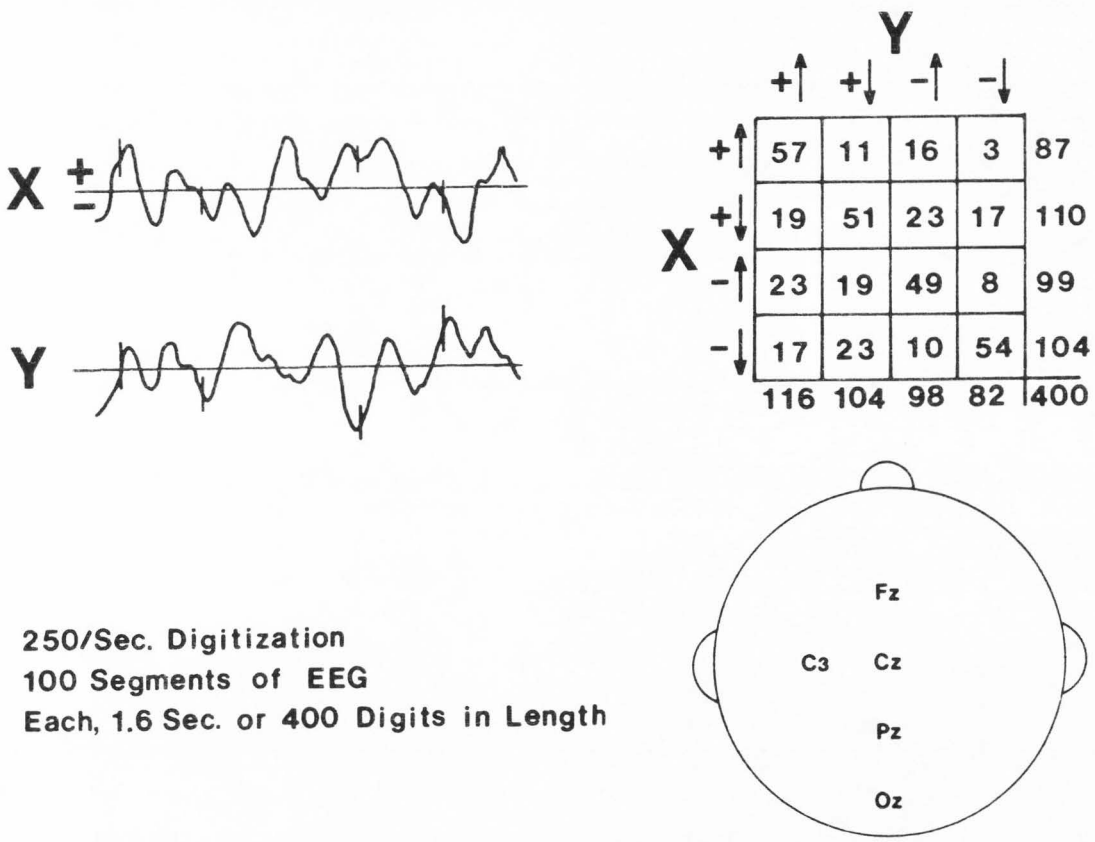

Figure 3. Method of determining EEG cortical coupling; scalp areas from which EEG was recorded.

characteristics.

Visual Evoked Potential (VEP)

EEG tracings for the first 300 msec following visual stimulus presentation were visually inspected to eliminate trials contaminated by artifact. VEPs were then averaged from 50 artifact-free trials and digitally plotted on graph paper so that waveform latencies and amplitudes could be measured. Four stimulus conditions were employed: 1, 2, and 3 $\log_{10}$ intensities for patterned flash; 2 $\log_{10}$ intensity for unpatterned flash. Age X sex X intensity ANOVAs with repeated measures on intensity were calculated for latencies of components N80, P100, N130, and P200 and amplitudes of components N80-P100, P100-N130, and N130-P200 for each of three electrode sites (Fz, Cz, and Oz).

Amplitude/Intensity Slope. Amplitude/intensity slope was calculated using a least squares solution with VEP amplitudes for the 1, 2, and 3 $\log_{10}$ intensities being Y-coordinates and the nearest integer roots of intensity ratios (1:10:100), i.e., 1, 3, and 10 being the X-coordinates (Dustman et al., 1982; Knorring, 1978). Slope values provided a measure of VEP amplitude change across intensity.

VEPs Elicited by Patterned versus Unpatterned Stimuli. For the 2 $\log_{10}$ intensity, VEP responses elicited by patterned stimulation were compared with those elicited by unpatterned flashes to describe the relative degree of similarity between VEP waveforms. This was done by correlating the digital values comprising the 0-300 msec segment of a VEP elicited by patterned stimulation with similarly derived values for a VEP to unpatterned stimulation. Coefficients of correlation were transformed to Fisher z-coefficients to better meet requirements for a normal sampling distribution (Cohen & Cohen, 1975). Degree of visual differentiation, i.e., the ability of the visual system to detect patterns as measured by difference between VEPs to patterned vs. unpatterned stimuli, is inversely related to the z-coefficient. Lower z-coefficient suggests greater differentiation (Dustman et al., 1981). Age X sex ANOVAs were calculated for z-coefficients for frontal and occipital electrode sites.

Pattern Reversal Evoked Potential (PREP)

Two hundred artifact-free single trial responses to binocular pattern reversal stimulation provided the PREP data for analysis. Age X sex ANOVAs were calculated for latencies of components N70, P100, N150, P200 and amplitudes of components N70-P100, P100-N150, N150-P200 recorded from

occipital scalp.

P300

For each subject, the first 25 artifact-free (by visual inspection of EEG tracings) responses to target stimulation were analyzed. P300 amplitudes and latencies from frontal, central, and parietal electrode sites were analyzed by age X sex X area (Fz, Cz, and Pz) ANOVAs for latency (P1, N1, P2, N2, and P3) and for amplitude (P1-N1, N1-P2, P2-N2, and N2-P3).

CHAPTER IV

RESULTS

The results of this study, gender and age effects of EEG and evoked potentials, may be more easily understood when reported in subsections, i.e., EEG measures of power spectral analysis and cortical coupling, brainstem auditory evoked potentials (BAEPs), visual evoked potentials (VEPs), pattern reversal evoked potentials (PREPs), and P300s. Age and sex results are described for latency, amplitude, and other examined variables such as intensity, scalp electrode site, etc.

EEG

Power Spectral Analysis

Age X sex X area (Fz, Cz, Pz, Oz, and C3) ANOVAs were computed on $\log_{10}$ transforms of the power spectral analysis (PSA) data for each of four frequency bands: 5-7, 7-9, 9-11, and 11-13 Hz.

There were no age or sex effects for any of the frequency

bands.

Area. The effect of area on power spectral analysis was highly significant. Significant differences among area means for each frequency band are provided in Table 1).

Interactions. Age X area interactions occurred for the 5-7, 7-9, and 11-13 Hz frequency bands. Respective levels of significance: $F = 13.40$, $p < .001$; $F = 3.97$, $p < .01$; and $F = 4.48$, $p < .01$, each 3, 228 df.

As can be observed from Figure 4, EEG power of young adults appeared to vary more across electrode sites than that of older adults. This observation was investigated by computing a homogeneity score, i.e., the standard deviation of PSA loadings across the four electrode sites for each subject. T-tests were then calculated to determine if PSA homogeneity significantly differentiated the young from the older group. Significant differences were obtained for all frequency bands: PSA for the older adults was more homogeneous than that for the younger adults (see Figure 4).

Cortical Coupling

An age X sex X electrode pair ANOVA was computed on cortical coupling values (Table 2 lists the electrode pairs).

Table 1

Summary of EEG Power Spectral Analyses

Electrode Site	5-7 Hz $\bar{X}$ (SD)	7-9 Hz $\bar{X}$ (SD)	9-11 Hz $\bar{X}$ (SD)	11-13 Hz $\bar{X}$ (SD)
Fz	.89 (.28)	1.01 (.41)	.98 (.45)	.55 (.35)
Cz	.92 (.31)	1.06 (.43)	1.07 (.47)	.68 (.41)
Pz	.84 (.33)	1.04 (.47)	1.18 (.56)	.84 (.52)
Oz	.71 (.33)	.93 (.46)	1.16 (.57)	.81 (.50)
F	69.4	18.4	32.3	62.9
p	< .001	< .001	< .001	< .001
	Fz,Cz>Pz,Oz** Pz>Oz**	Fz,Cz,Pz>Oz** Cz>Fz*	Cz,Oz,Oz>Fz** Pz,Oz>Cz**	Cz,Oz,Pz>Fz** Pz,Oz>Cz**

* *p* > .05; ** *p* > .01; Duncan's Multiple Range Test for electrode site.

Note. Degrees of freedom for area F-ratios were (3, 228).

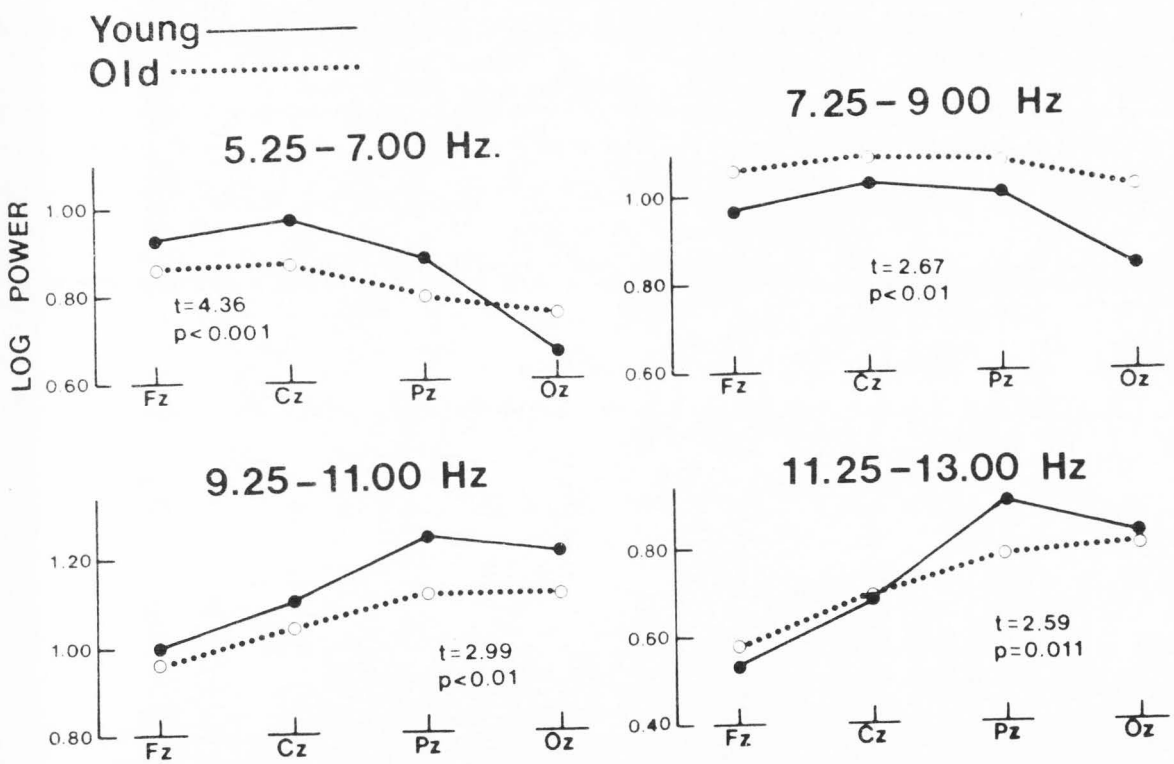

Figure 4. EEG power spectral analysis values for young and old adults, and t-tests which reflect age differences in variability of power loadings across electrode sites.

Table 2

Summary of Cortical Coupling Analyses

Electrode Pair	Young $\bar{X}$	Young (SD)	Old $\bar{X}$	Old (SD)	F	p
Fz-Cz	.63	(.12)	.74	(.15)	12.61	< .001
Fz-Pz	.24	(.08)	.34	(.12)	19.50	< .001
Fz-Oz	.09	(.04)	.16	(.08)	26.28	< .001
Fz-C3	.39	(.11)	.42	(.13)	.91	NS
Cz-Pz	.51	(.10)	.65	(.13)	31.84	< .001
Cz-Oz	.16	(.07)	.28	(.10)	41.88	< .001
Cz-C3	.54	(.13)	.58	(.15)	1.73	NS
Pz-Oz	.42	(.12)	.63	(.14)	53.69	< .001
Pz-C3	.40	(.09)	.52	(.11)	29.57	< .001
Oz-C3	.15	(.05)	.29	(.12)	46.65	< .001

Note. Degrees of freedom for age and sex F-ratios were (1, 76).

Significant effects were found for age and for electrode pair.

Age. For all electrodes combined, cortical coupling values were significantly lower (less similarity between pairings) for young adults ($\bar{X}$ = .35) than for older adults ($\bar{X}$ = .46), F (1, 76) = 44.20, $p < .001$. Thus, EEG from the different electrode sites was more similar, or more homogeneous, for older than for younger subjects.

Sex. There were no gender differences revealed by cortical coupling analysis.

Area. Mean cortical coupling values were generally higher for electrode sites which were closer together (e.g., Fz-Cz, $\bar{X}$ = .68,) and lower for sites which were further apart (e.g., Fz-Oz, $\bar{X}$ = .12). Post hoc mean comparisons revealed that all electrode pairings were significantly different from one another ($p < .01$), with two exceptions: Cz-C3 did not differ from Cz-Pz, and Cz-Oz did not differ from Oz-C3.

Interactions. An age X electrode pair interaction occurred, F (9, 684) = 7.41, $p < .001$. To further investigate this effect, each pairing was separately analyzed by an age X sex ANOVA. For all pairings except Fz-C3 and Cz-C3, cortical coupling values of the older adults were significantly greater than those for the younger group (see Table 2). There were no

significant sex differences revealed by any of these analyses. Only one significant age X sex interaction occurred; for electrode pairing Cz-Pz, age differences were larger for women than for men, F (1, 76) = 3.99, $p < .05$).

Brainstem Auditory Evoked Potential (BAEP)

The BAEP response was studied for latency, interpeak latency (IPL), and amplitude. Age X sex ANOVAs were calculated for each of these three response characteristics.

BAEP Latency

Age. No significant age-related BAEP latency effects were found.

Sex. BAEP latency was significantly affected by gender. Latency for females was shorter than for males for waves II, III, IV, V, and VII, as reported in Table 3.

Interactions. Age and sex interacted significantly for wave II latency, F (1, 76) = 6.35, $p < .05$. The young females ($\bar{X}$ = 2.54 msec) responded wtih significantly shorter latency

Table 3

Summary of BAEP Latency Analyses (msec)

Age

Wave	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
I	1.56	(.16)	1.50	(.14)	3.07	NS
II	2.66	(.25)	2.58	(.24)	2.23	NS
III	3.66	(.24)	3.60	(.22)	1.69	NS
IV	4.97	(.22)	4.90	(.26)	1.84	NS
V	5.61	(.22)	5.58	(.26)	.27	NS
VI	7.35	(.32)	7.38	(.35)	.15	NS
VII	9.07	(.31)	8.98	(.36)	1.31	NS

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
I	1.52	(.16)	1.54	(.14)	.54	NS
II	2.57	(.20)	2.67	(.28)	3.97	= .05
III	3.56	(.22)	3.70	(.23)	7.61	< .01
IV	4.86	(.21)	5.02	(.25)	9.67	< .01
V	5.52	(.26)	5.67	(.21)	7.63	< .01
VI	7.30	(.31)	7.44	(.35)	3.64	NS
VII	8.92	(.34)	9.13	(.30)	8.51	< .01

Note. Degrees of freedom for age and sex F-ratios were (1, 76).

than did the young males ($\bar{X}$ = 2.77 msec), t (38) = 3.32, $p \leq .01$.

BAEP IPLs

Age. There were no significant age-related IPL effects.

Sex. Interpeak latency was significantly affected by gender. IPLs I-III and I-V were significantly shorter for females than for males (see Table 4).

Interactions. No significant IPL interactions occurred.

BAEP Amplitude

Age. For BAEP amplitude, the age factor produced significant wave-specific differences (see Table 5). BAEP wave I was larger for young adults than for older adults. However, for waves III, V, and VII, the young adults had smaller BAEP amplitudes than did the older adults.

Sex. Analysis by gender revealed that female response amplitudes were greater than those for males for waves IV, V, and VII (see Table 5).

Table 4

Summary of BAEP Interpeak Latency Analyses (msec)

Age

Waves	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
I-III	2.10	(.28)	2.10	(.19)	.01	NS
I-V	4.05	(.23)	4.08	(.26)	.34	NS
III-V	1.94	(.24)	1.98	(.24)	.45	NS

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
I-III	2.04	(.24)	2.16	(.23)	4.36	< .05
I-V	4.00	(.25)	4.12	(.23)	5.00	< .05
III-V	1.96	(.23)	1.97	(.25)	.04	NS

Note. Degrees of freedom for age and sex F-ratios were (1, 76).

Table 5

Summary of BAEP Amplitude Analyses (uv)

Age

Wave	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
I	.32	(.16)	.20	(.15)	12.94	< .001
II	.15	(.10)	.16	(.11)	.11	NS
III	.21	(.10)	.30	(.19)	5.78	< .05
IV	.10	(.07)	.12	(.11)	1.92	NS
V	.44	(.16)	.59	(.23)	11.93	< .001
VI	.21	(.13)	.20	(.13)	.41	NS
VII	.13	(.07)	.18	(.13)	7.02	< .01

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
I	.30	(.19)	.23	(.13)	3.89	< .001
II	.18	(.11)	.14	(.09)	3.17	NS
III	.28	(.18)	.22	(.13)	3.10	NS
IV	.14	(.11)	.08	(.06)	8.60	< .01
V	.58	(.23)	.45	(.16)	10.40	< .01
VI	.19	(.15)	.22	(.11)	.64	NS
VII	.18	(.12)	.13	(.08)	6.04	< .05

Note. Degrees of freedom for age and sex F-ratios were (1, 76).

Interactions. Age and sex interacted significantly for wave VII amplitude, F (1, 76) = 12.61, $p < .001$. Group comparison by t-test showed that young females were not significantly different from young males, nor were young males significantly different from older males in wave VII amplitude. However, mean amplitude for older females, $\bar{X}$ = .25 uv, was twice as large as that for both young females and older males ($\bar{X}$ = .12 uv, each). Levels of significance were t (38) = 3.94, $p < .01$, and t (38) = 3.46, $p < .01$, respectively.

Visual Evoked Potentials (VEP)

Figure 5 compares young with old (left column) and female with male subjects (right column) for all visual measures, i.e., VEPs, PREPs, and P300s. Each is discussed separately.

An age X sex X intensity (1, 2, and 3 logs above threshold) ANOVA with repeated measures on intensity was calculated for latencies of the N80, P100, N130, and P200 components and for N80-P100, P100-N130, and N130-P200 amplitudes. Results from frontal and occipital recording sites for each of the four subject subgroups and for each of three stimulus intensities are represented in Figure 6.

The relationship between VEP amplitude and intensity was further analyzed by amplitude/intensity slope, a measure of

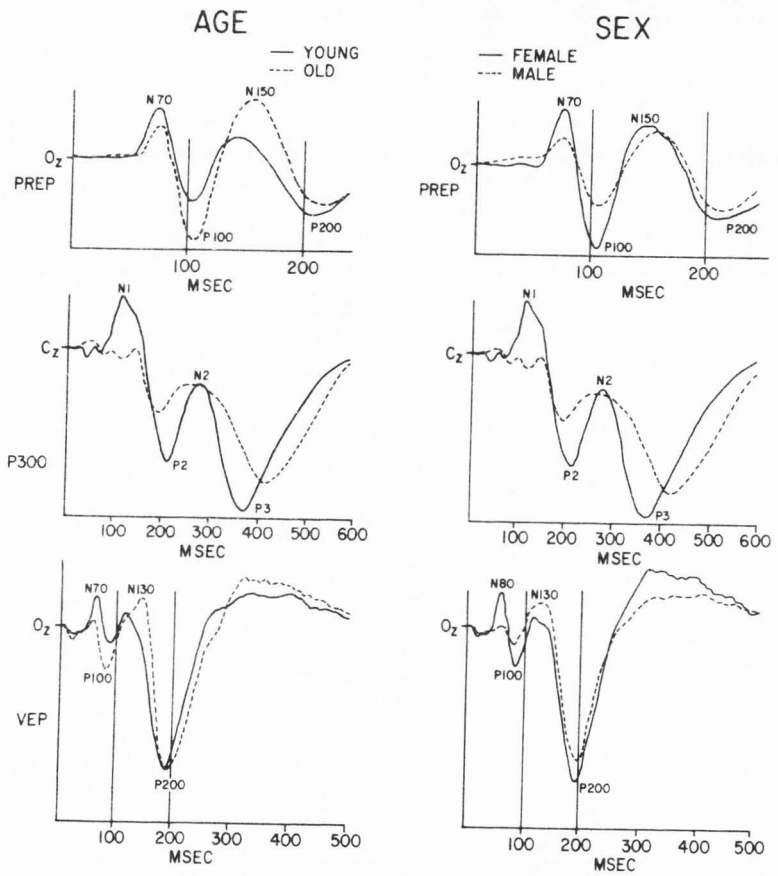

Figure 5. A comparison of young and old adults (left column) and female and male subjects (right column) for VEPs, PREPs, and P300s.

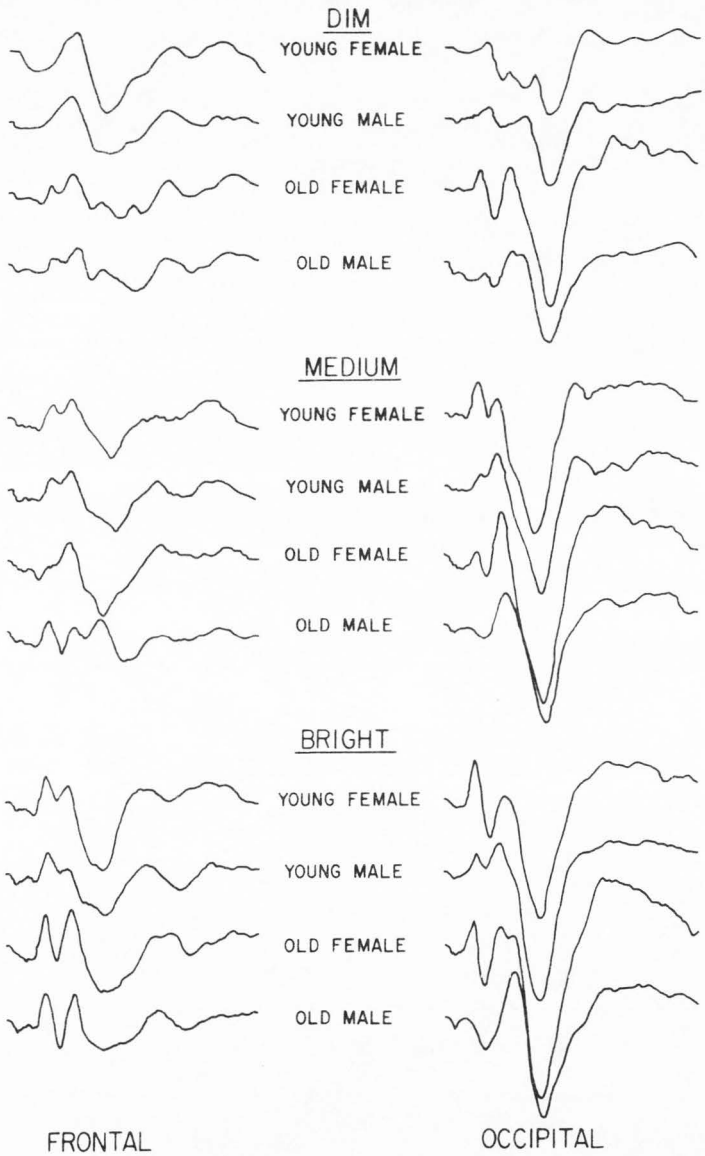

Figure 6. Group VEPs for young females, young males, old females, and old males elicited by dim, medium, and bright flashes for frontal and occipital recording sites.

amplitude change across intensity. Amplitude/intensity slope is reported in the subsection which follows VEP amplitude.

Electrode site (Fz, Cz, Oz) was also investigated as a variable affecting VEP response. Because results from frontal and central scalp recordings were essentially the same, data from Fz are reported in the text, and Cz data can be found in Appendix C.

Frontal Electrode Site (Fz)

Fz Latency Age. There was a consistent trend for P100, N130, and P200 components: latency was significantly shorter for young adults than for older adults (see Table 6).

Sex. For each of VEP component, latency for females was shorter than latnecy for males. This gender effect was consistently significant for all components of the response, as noted in Table 6.

Intensity. Significant intensity effects were found for N80, N130, and P200. In general, latency and intensity were inversely related, i.e., the dimmest flash produced the longest latency response and the brightest flash produced the shortest latency response. Specific results are contained in Table 6.

Table 6

Summary of Age X Sex X Intensity ANOVAs for VEP Latencies (msec) from Frontal (Fz) Scalp

Age

Component	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
N80	84.5	(12.1)	83.8	(10.5)	.23	NS
P100	103.0	(12.0)	107.0	(12.3)	4.31	< .05
N130	128.1	(13.8)	136.4	(17.3)	9.07	< .01
P200	197.7	(21.4)	208.6	(26.2)	9.58	< .01

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
N80	81.7	(11.3)	86.7	(10.7)	11.33	< .01
P100	102.1	(12.3)	108.0	(11.6)	9.09	< .01
N130	128.4	(13.2)	136.1	(17.8)	7.83	< .01
P200	197.6	(23.0)	208.7	(24.8)	9.87	< .01

Intensity

	1 $\bar{X}$	(SD)	2 $\bar{X}$	(SD)	3 $\bar{X}$	(SD)	F	p
N80	87.8	(13.4)	84.3	(11.1)	80.4	(7.6)	10.0 1>3** 1,2>3*	< .001
P100	106.4	(14.5)	105.6	(12.4)	103.3	(9.4)	2.12	NS
N130	134.7	(18.5)	131.4	(15.3)	139.6	(14.1)	3.24 1>3*	< .05
P200	208.4	(25.4)	207.8	(22.0)	193.2	(23.2)	16.49 1,2>3**	< .001

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for stimulus intensity conditions.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for intensity F-ratios.

Interactions. There were no significant interactions resulting from the analysis of frontal VEP latency.

Fz Amplitude Age. There were significant age effects for the amplitudes of N80-P100 and P100-N130 in VEPs from frontal scalp (Fz). Amplitudes for young adults were smaller than those for older adults for these components (see Table 7).

Sex. Amplitude for females was significantly greater than that for males for N130-P200.

Intensity. For all components, intensity significantly affected VEP amplitudes. The typical relationship was that an increase in intensity was accompanied by an increase in amplitude. Specific findings from the ANOVA and post hoc comparisons are contained in Table 7.

Interactions. No significant interactions occurred in analyses of frontal VEP amplitudes.

Occipital Electrode Site (Oz)

Oz Latency Age. The effect of age on occipital latency was apparent for P200. Latency was significantly shorter for the young adults than for the older adults (see Table 8).

Table 7

Summary of Age X Sex X Intensity ANOVAs
for VEP Amplitudes (uv) from Frontal (Fz) Scalp

Age

Component	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
N80-P100	2.8	(2.4)	5.2	(4.9)	13.28	< .001
P100-N130	3.8	(2.9)	6.5	(4.9)	14.95	< .001
N130-P200	9.9	(4.9)	8.9	(5.1)	1.46	NS

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
N80-P100	4.2	(4.7)	3.8	(3.3)	.36	NS
P100-N130	5.8	(4.7)	4.5	(3.6)	3.41	NS
N130-P200	10.8	(5.4)	8.0	(4.1)	11.32	< .01

Intensity

	Dim $\bar{X}$	(SD)	Medium $\bar{X}$	(SD)	Bright $\bar{X}$	(SD)	F	p
N80-P100	2.3	(2.4)	4.2	(4.8)	5.5	(3.8)	28.82 2,3>1** 3>2**	< .001
P100-N130	4.3	(3.4)	5.3	(4.7)	5.8	(4.4)	4.58 3>1*	< .05
N130-P200	9.0	(4.7)	8.7	(4.7)	10.6	(5.5)	6.65 3>1,2**	< .01

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for stimulus intensity conditions.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for intensity F-ratios.

Table 8

Summary of Age X Sex X Intensity ANOVAs
for VEP Latencies (msec) from Occipital (Oz) Scalp

Age

Component	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
N80	73.6	(11.3)	72.8	(14.9)	.14	NS
P100	99.4	(16.1)	99.2	(16.8)	.00	NS
N130	129.2	(21.3)	132.1	(17.9)	.92	NS
P200	195.8	(13.9)	200.4	(15.3)	5.91	< .05

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
N80	70.4	(11.5)	76.1	(14.3)	6.99	< .01
P100	97.8	(15.1)	100.8	(17.6)	1.19	NS
N130	128.3	(20.1)	133.1	(19.1)	2.48	NS
P200	196.0	(15.0)	200.2	(14.3)	5.04	< .05

Intensity

	1 $\bar{X}$	(SD)	2 $\bar{X}$	(SD)	3 $\bar{X}$	(SD)	F	p
N80	78.9	(11.1)	73.2	(12.7)	67.6	(13.4)	28.74	< .001
							1,2>3**	
							1>2**	
P100	107.8	(13.1)	96.9	(17.1)	93.2	(15.3)	40.63	< .001
							1>2,3**	
							2>3*	
N130	141.2	(18.4)	124.3	(18.2)	126.4	(18.2)	36.22	< .001
							1>2,3**	
P200	206.0	(12.5)	195.4	(15.3)	192.9	(13.3)	23.33	< .001
							1>2,3**	

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for stimulus intensity conditions.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for intensity F-ratios.

Sex. N80 and P200 were affected by gender: latency for females was again significantly shorter than that for males, as noted in Table 8.

Intensity. Intensity was a highly significant source of occipital latency variance for all response components, $p < .001$. As with frontal scalp recordings, post hoc comparison revealed an inverse relationship between latency and stimulus intensity (see Table 8). Typically, brighter flashes were associated with shorter latencies.

Interactions. An age X sex interaction occurred for N130, F (2, 152) = 6.20, $p < .05$. As shown in Figure 7, latency means for the older males were larger than those of the other subjects. T-test analysis of this interaction for the brightest flash intensity confirmed that latency for older males was significantly longer than that for younger males, young females, and for older females, t (38) = 3.58, $p < .001$; 2.45, $p < .05$; and 2.37, $p < .05$, respectively.

Age and intensity interacted significantly for P100 and N130, F (2, 152) = 4.87, $p < .01$ and 7.17, $p < .01$, respectively. Compared to the older subjects, young adults responded with longer latencies for dim flashes, but shorter latencies for bright flashes (see Figure 8). For example, for dim flashes, the N130 latency means of young subjects was 6.45 msec longer than that for the older subjects, but 8.6 msec

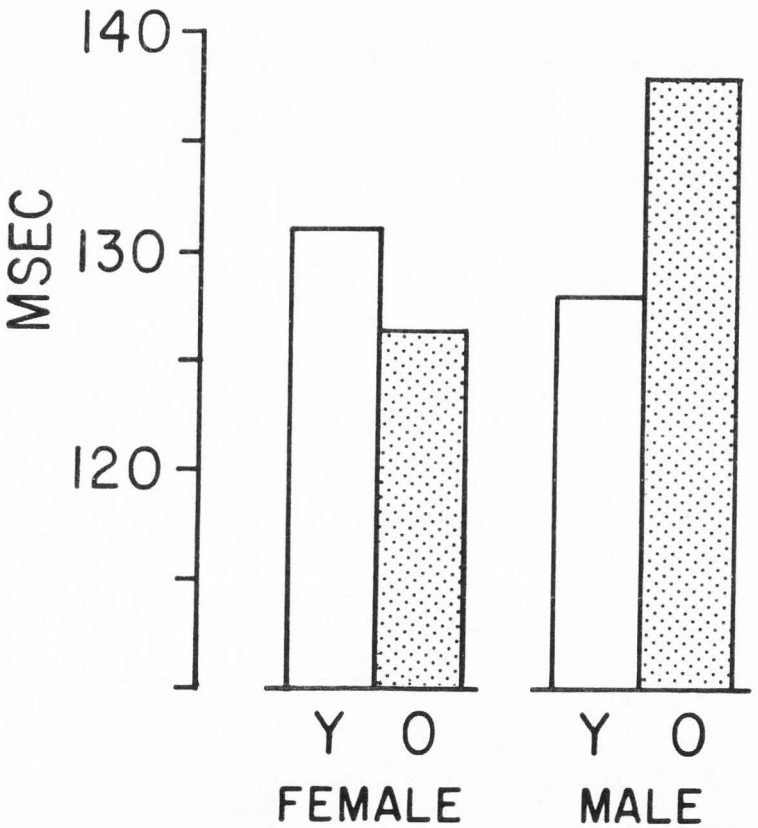

Figure 7. Latencies of VEP component N130 recorded from occipital scalp.

shorter for the bright flash condition.

Another way of looking at these interactions is the comparative degree of latency change. Young adults appeared to respond with greater magnitude of latency change across intensity (see Figure 8). This observation was tested (a) by computing a score for response intensity difference, i.e., the difference between the bright flash and dim flash latencies, and (b) by comparing t-test mean differences for the young and old subjects. Mean differences were 19.1 for the young group vs. 10.3 for the oldsters. The significant difference between means, t (78) = 2.92, $p < .01$, supported the observation that young adults responded with greater magnitude of latency change across intensity.

Oz Amplitude Age. There were two occipital VEP amplitudes which were significantly affected by age: P100-N130 and N130-P200. For both components, amplitude was smaller for the younger than for the older adults (see Table 9).

Sex. A significant sex effect occurred for N80-P100: compared to that for males, VEP amplitude for females was greater (see Table 9).

Intensity. The effect of intensity on amplitude was highly significant for all components; results are summarized in Table 9. Again, a direct relationship of increasing

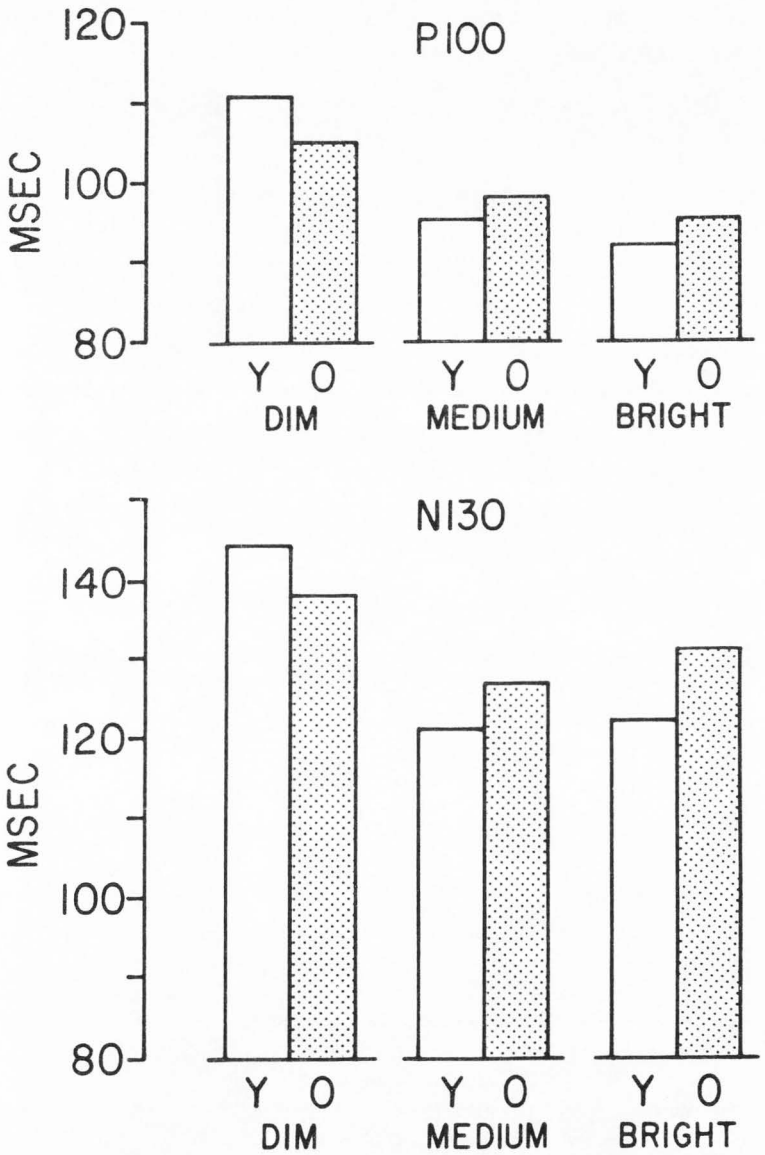

Figure 8. Latencies of VEP components P100 and N130 recorded from occipital scalp.

Table 9

Summary of Age X Sex X Instensity ANOVAs
for VEP Amplitudes (uv) from Occipital (Oz) Scalp

Age

Component	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
N80-P100	6.3	(4.8)	7.9	(5.4)	3.61	NS
P100-N130	6.7	(5.6)	11.0	(8.2)	11.29	< .01
N130-P200	14.9	(9.4)	19.2	(11.7)	4.76	< .05

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
N80-P100	8.9	(5.3)	5.3	(4.3)	19.07	< .001
P100-N130	10.0	(7.8)	7.7	(6.6)	3.29	NS
N130-P200	18.8	(11.2)	15.3	(10.2)	3.04	NS

Intensity

	1 $\bar{X}$	(SD)	2 $\bar{X}$	(SD)	3 $\bar{X}$	(SD)	F	p
N80-P100	6.4	(3.8)	6.0	(4.8)	8.9	(6.2)	17.38	< .001 3>1,2**
P100-N130	7.4	(5.4)	7.4	(5.9)	11.7	(9.2)	24.38	< .001 3>1,2**
N130-P200	12.7	(8.6)	18.3	(10.9)	20.0	(11.5)	34.28	< .001 3>1,2**

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for stimulus intensity conditions.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for intensity F-ratios.

amplitude with increasing stimulus intensity was observed.

Interactions. An age X intensity occurred for N80-P100, F (2, 152) = 3.06, p = .05. Figure 9 shows that as stimulus intensity increased, the amplitude for the younger subjects did not increase as much as that for the older subjects. The difference between younger and older adults appeared to be greatest at brightest flash.

For N80-P100, sex and intensity also interacted significantly, F (2, 152) = 4.48, $p < .05$. Females differed most from males at the dimmest and brightest flash intensities. VEP amplitude for females increased more from the dim to the bright intensity than it did for males (Figure 10).

The relationship of amplitude change across intensity is further described in the amplitude/intensity slope section (below).

Summary. In general, visual evoked potentials were sensitive to the effects of age, sex, and intensity. The characteristic differences were consistent: young adults responded with greater amplitudes and shorter latencies than older adults; females responded with greater amplitudes and shorter latencies than males; brighter flashes were associated with larger, faster responses.

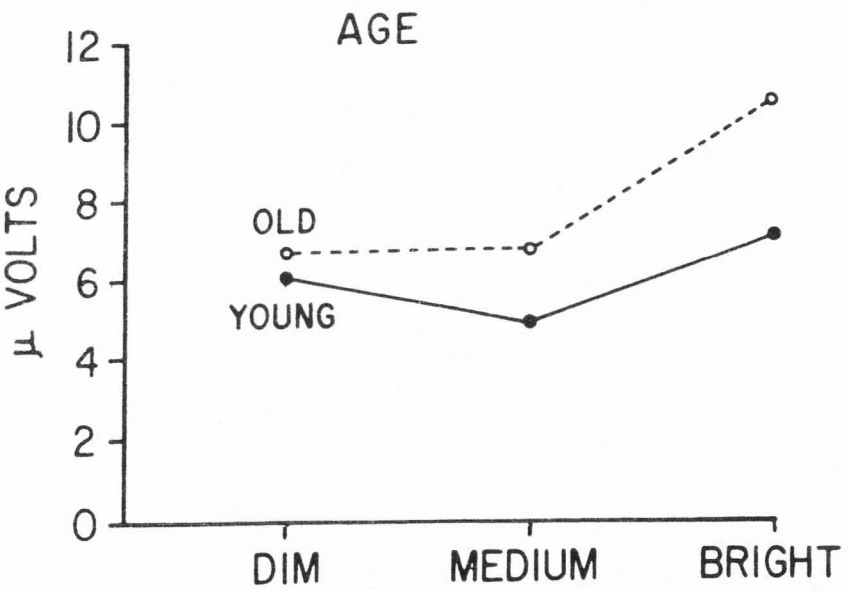

Figure 9. Amplitudes of VEP component N80-P100 recorded from occipital scalp.

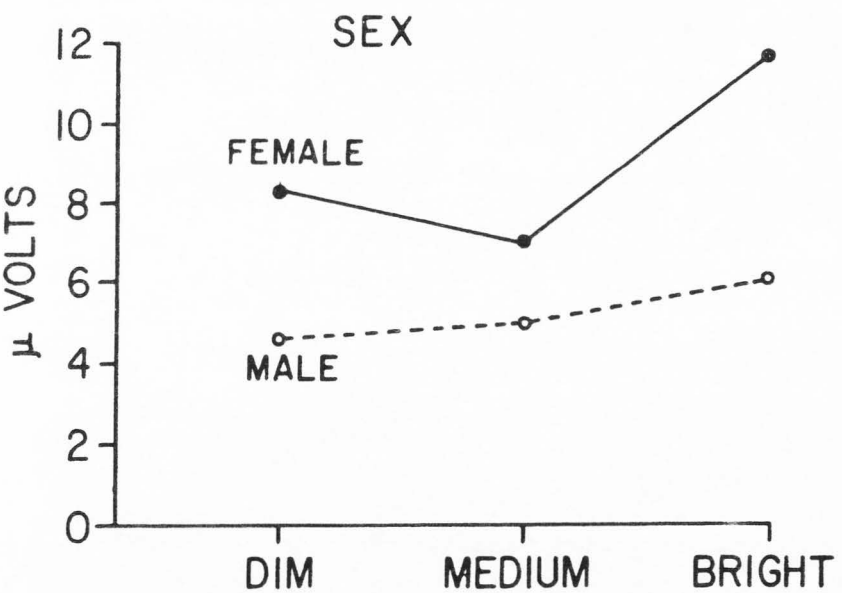

Figure 10. Amplitudes of VEP component N80-P100 recorded from occipital scalp.

Amplitude/Intensity Slope

An age X sex X area (Fz, Cz, Oz) ANOVA was calculated on amplitude/intensity slope values. Analysis results are reported in Table 10.

Age. A significant age effect was found for P100-N130: younger adults produced smaller mean slope values than older adults (1.4 versus 3.6). That is, a significantly greater amplitude increase across intensity occurred for oldsters (see Table 10).

Sex. Gender had no significant effect on amplitude/intensity slope.

Area. Area was a highly significant factor for both P100-N130 and N130-P200 (see Table 10). Post hoc comparison of electrode sites showed that, for both components, mean slope values for the posterior site (Oz) was significantly larger than that for the anterior sites (Fz, Cz). This area difference is illustrated in Figure 11.

Interactions. As shown in Figure 12, sex and area interacted signficantly for N80-P100, F (2, 152) = 4.25, $p < .05$. Particularly for occipital recordings, females displayed a relatively greater increase in VEP amplitude compared to

Table 10

Summary of Amplitude/Intensity Slope Analyses

Component	Age		Sex		Area		
	Young	Old	Female	Male	Fz	Cz	Oz
N80-P100							
MN	2.3	3.3	3.0	2.6	3.2	2.1	3.1
SD	4.0	4.9	5.0	3.8	4.2	3.0	5.8
F	2.51		.39		2.07		
P	NS		NS		NS		
P100-N130							
MN	1.4	3.6	2.4	2.6	1.4	1.0	5.1
SD	5.2	7.7	7.2	6.0	5.0	5.6	8.1
F	4.10		.02		15.20		
P	< .05		NS		< .001		
					Oz>Fz,Pz**		
N130-P200							
MN	3.6	4.0	3.7	3.9	2.0	2.8	6.6
SD	7.4	7.4	7.3	7.5	5.8	6.0	9.2
F	.21		.03		10.79		
P	NS		NS		< .001		
					Oz>Fz,Cz**		

** $p < .01$; Duncan's Multiple Range Test for electrode site.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for area F-ratios.

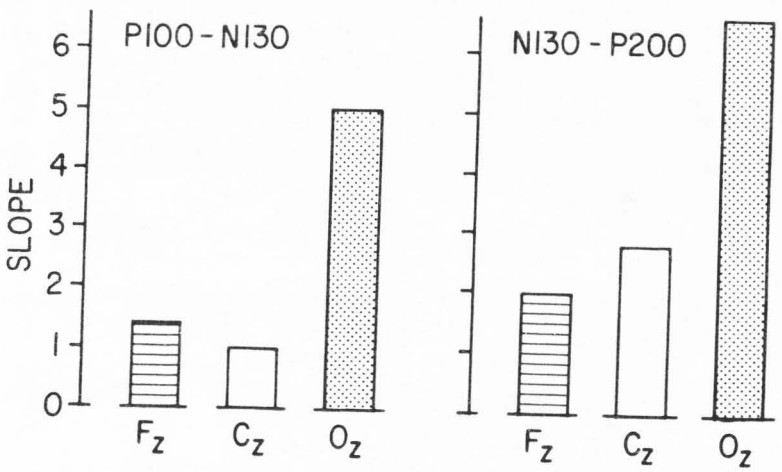

Figure 11. Amplitude/intensity slope: area effects for P100-N130 and for N130-P200.

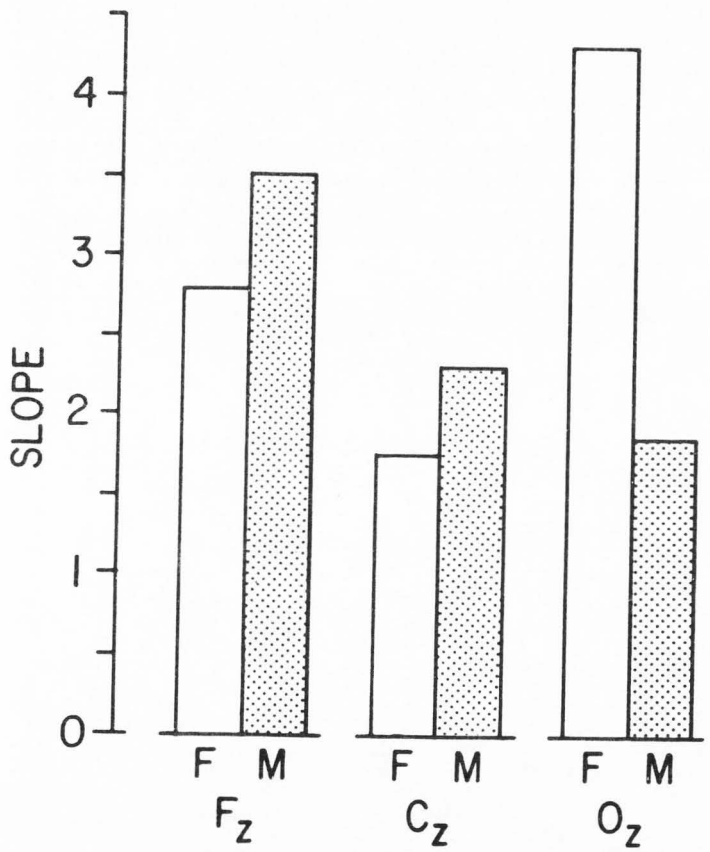

Figure 12. Amplitude/intensity slope for N80-P100 sex X area interaction.

males. In order to clarify this finding, as well as the age X intensity and sex X intensity interactions found for N80-P100 VEP amplitude for occipital electrode site (see Figures 9 and 10), a two factor ANOVA (age X sex) on A/I slope for Oz was calculated. This additional analysis revealed significant main effects for age and for sex. Older subjects responded with greater mean slope values than younger subjects (4.5 versus 1.8, F [1, 76] =4.72, $p < .05$). Compared to males ($\bar{X} = 1.9$), females ($\bar{X} = 4.4$) showed greater mean slope values, F (1, 76) = 4.08, $p = <.05$. Figure 13 indicates that as intensity increased, VEP amplitudes increased more for oldsters than for young people, and more for women than for men.

Analysis of N130-P200 amplitude/intensity slope revealed an age X area interaction, F (2, 152) = 3.35, $p < .05$. For the frontal electrode site, A/I values were lower for young adults than for older adults. However, for the occipital electrode site, A/I values were higher for young adults than for older adults (see Figure 14). Younger adults appeared to respond with relatively less increase in VEP amplitude than did oldsters.

VEPs Elicited by Patterned versus Unpatterned Stimuli

Age. Figure 15 illustrates the main effects of age on z-coefficients resulting from correlations of VEPs elicited by

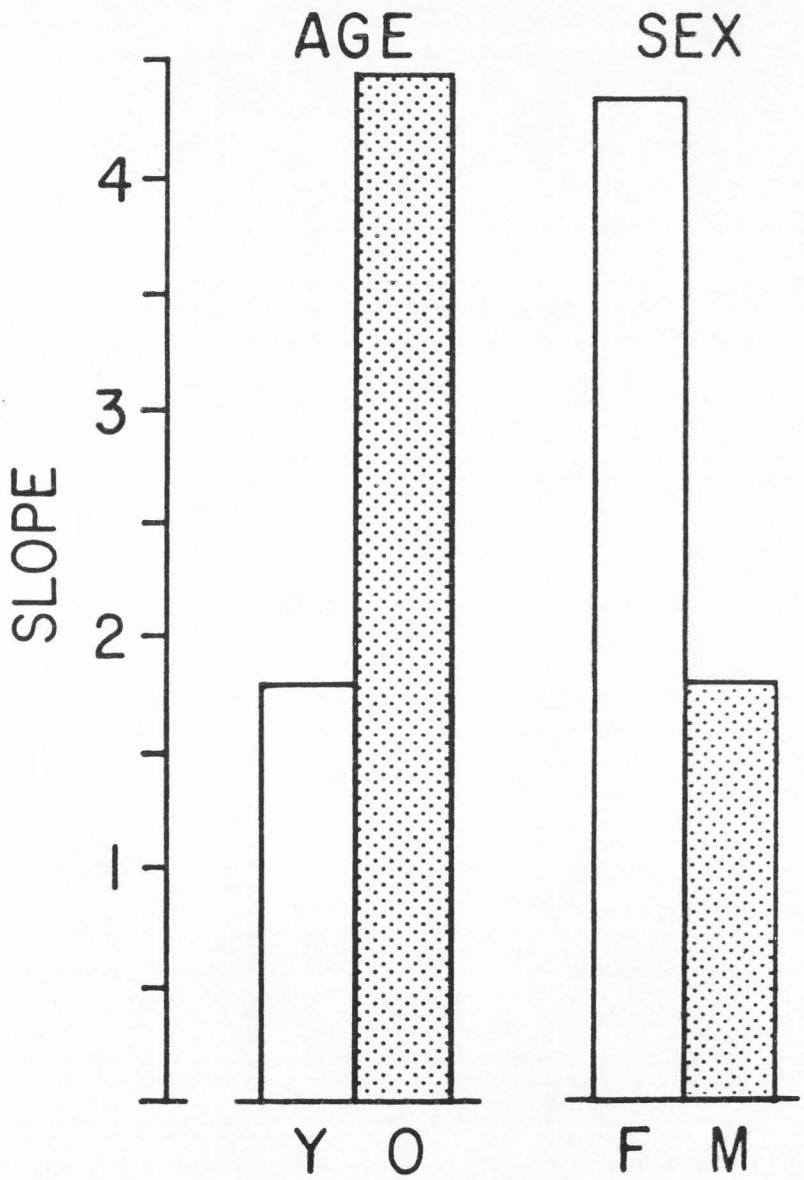

Figure 13. Age and sex effects for amplitude/intensity slope of N80-P100 recorded from occipital scalp.

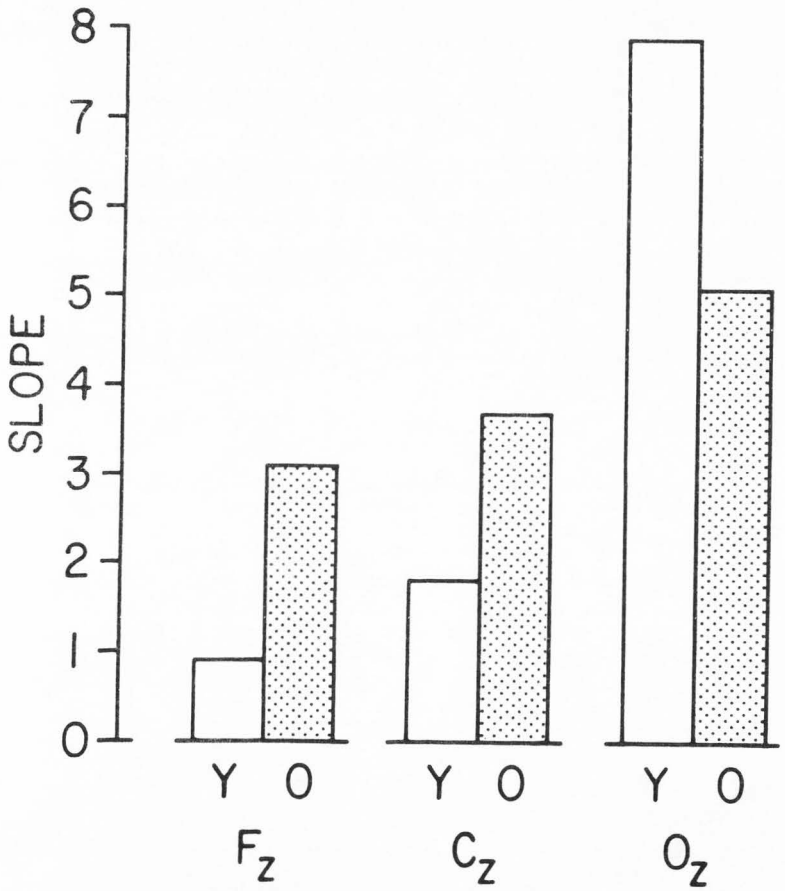

Figure 14. Amplitude/intensity slope for N130-P200 recorded from occipital scalp: age X area interaction.

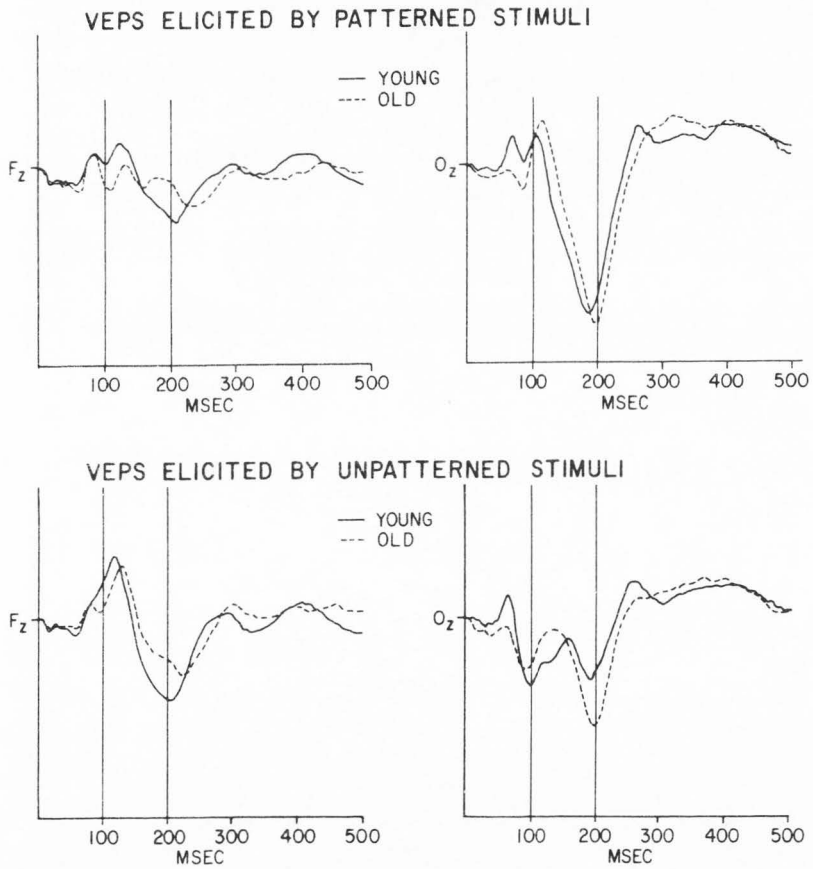

Figure 15. Age and sex effects for VEPs elicited by patterned vs. unpatterned stimuli.

patterned flashes with those elicited by unpatterned flashes. For frontal (Fz) recordings, a significant age difference was found: patterned vs. unpatterned VEP waveforms were more similar for young than for old people. The mean z-coefficient for the young subjects was 1.04 versus .77 for the older subjects, F [1, 76] = 6.12, $p < .05$).

For occipital (Oz) recordings, the age factor approached significance: $\bar{X}$ = .75 for young adults; $\bar{X}$ = .97 for older adults; F (1, 76) = 3.71, p = .058. Younger subjects showed less similarity, or greater differentiation for occipital responses. An interesting pattern can thus be observed: compared to older subjects, the younger subjects had less differentiation (higher z-coefficients for VEPs measured from Fz, but greater differentiation (lower z-coefficients) for VEPs measured from Oz (see Figure 16).

Sex. No significant sex differences were found for this measure.

No interactions occurred as a result of correlating digital values of VEP responses elicited by patterned flash stimuli with values for unpatterned flash stimuli.

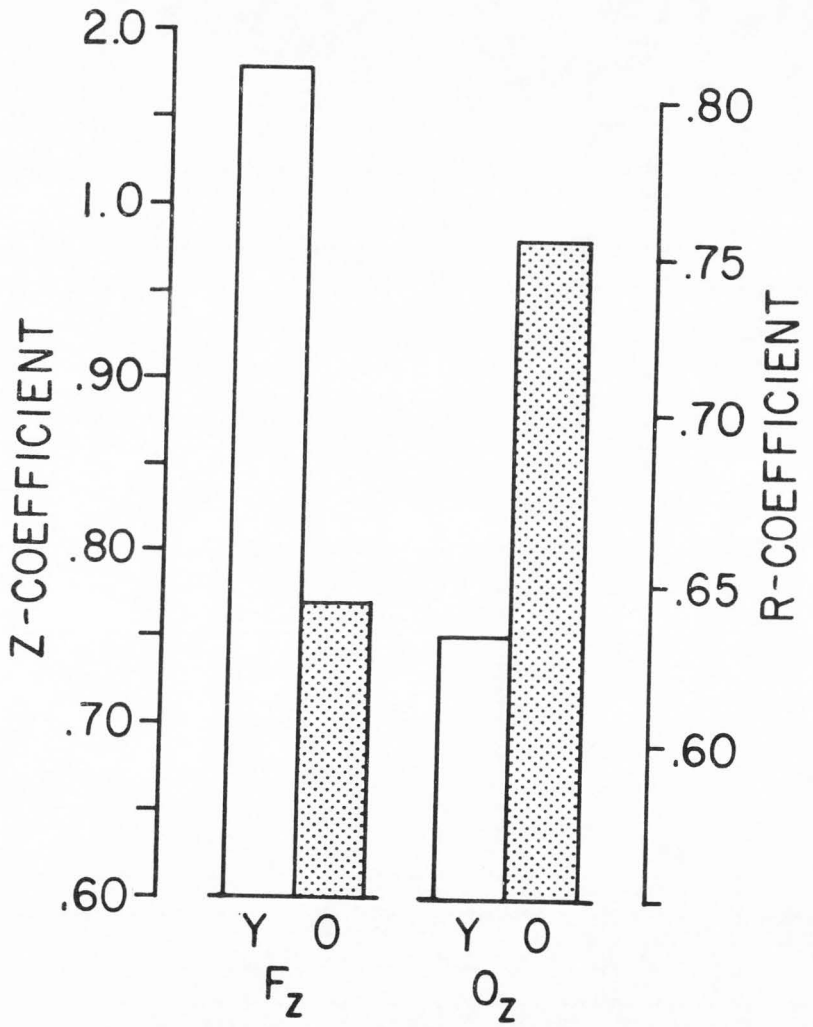

Figure 16. Age comparisons for frontal and occipital z-coefficients resulting from correlations of VEPs elicited by patterned stimuli with those elicited by unpatterned stimuli.

Pattern Reversal Evoked Potential (PREP)

Averaged pattern reversal evoked potentials for young females, young males, old females, and old males are reproduced in Figure 17.

The pattern reversal evoked potential was recorded from Oz and analyzed for age and gender effects by a 2 X 2 ANOVA. Table 11 summarizes analyses for N70, P100, N150, and P200 latency; Table 12 contains N70-P100, P100-N150, N150-P200 amplitude results.

PREP Latency

Age. PREP latency was significantly shorter for young adults than for older adults for both N70 and N150. The latencies of P100 and P200 were also earlier for the young than for the older adults although these age differences were not significant (see Table 11).

PREP latencies were not significantly different for females and males, and there were no significant interactions.

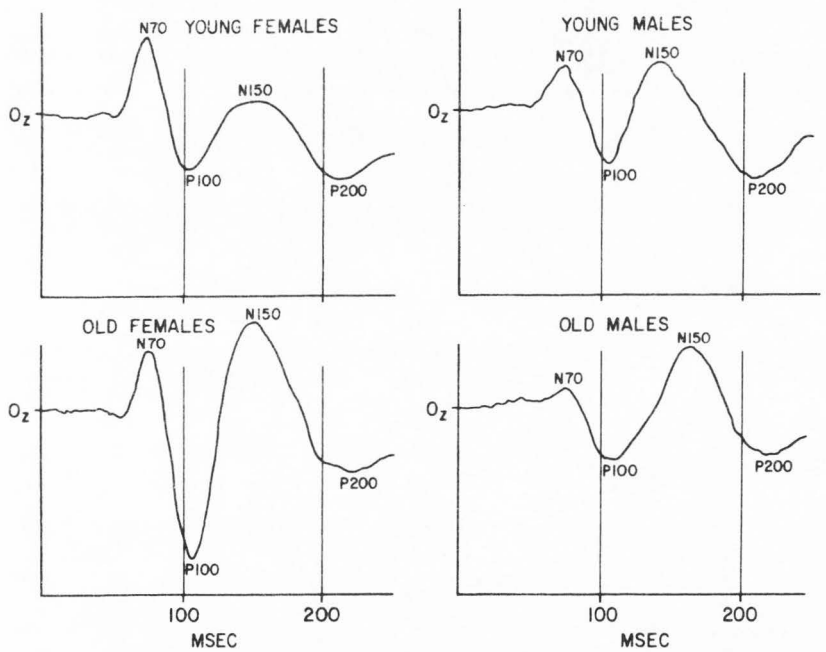

Figure 17. PREP responses.

Table 11

Summary of PREP Latency (msec) Analyses

Component	Age		Sex	
	Young	Old	Female	Male
N70				
MN	74.6	77.1	76.1	75.6
SD	3.9	6.4	4.7	6.1
F	4.84		.17	
P	< .05		NS	
P100				
MN	105.7	108.2	106.4	107.6
SD	6.7	9.7	7.3	9.4
F	1.81		.43	
P	NS		NS	
N150				
MN	142.4	158.0	148.7	151.8
SD	15.7	16.6	15.9	19.8
F	19.1		.75	
P	< .001		NS	
P200				
MN	209.0	213.5	209.3	213.2
SD	25.9	14.4	26.5	13.3
F	.91		.70	
P	NS		NS	

Note. Degrees of freedom for age and sex F-ratios were (1, 76).

Table 12

Summary of PREP Amplitude (uv) Analyses

Component	Age		Sex	
	Young	Old	Female	Male
N70-P100				
MN	9.6	12.10	13.8	7.8
SD	4.3	9.0	8.4	3.8
F	3.21		18.73	
P	NS		< .001	
P100-N150				
MN	8.8	15.1	13.9	10.0
SD	4.8	10.9	11.2	5.3
F	12.94		5.10	
P	< .001		< .05	
N150-P200				
MN	10.1	11.4	11.6	9.9
SD	5.8	7.7	8.0	5.3
F	.74		1.23	
P	NS		NS	

Note. Degrees of freedom for age and sex F-ratios were (1, 76).

PREP Amplitude

Age. Age significantly affected P100-N150. Mean amplitude for the young adults was significantly smaller than that for the older adults (see Table 12).

Sex. Sex was a significant factor for N70-P100 and for P100-N150 amplitudes. The same trend was displayed for both components: amplitude for females exceeded that for males (see Table 12).

Interactions. Analysis of PREP amplitude showed that age interacted with sex for both N70-P100 and P100-N150. The significance of the age X sex interaction for N70-P100 was F (1, 76) = 5.98, $p < .05$; for P100-N150, F (1, 76) = 8.32, $p < .01$. There appeared to be a greater sex difference for the older subjects than for younger subjects. In particular, the older females seemed to differ from other subgroups (see Figure 18). Single factor ANOVAs were computed that compared the four groups of subjects on PREP amplitude for N70-P100 and for P100-N150. These results verified that amplitude for older females was significantly greater than that for young females, young males, and older males, F (3, 76) = 9.31, $p < .001$; 8.79, $p < .001$, respectively. A Duncan's Multiple Range Test showed that means for the older women were significantly larger than means of the remaining three groups, $p < .01$. There were no

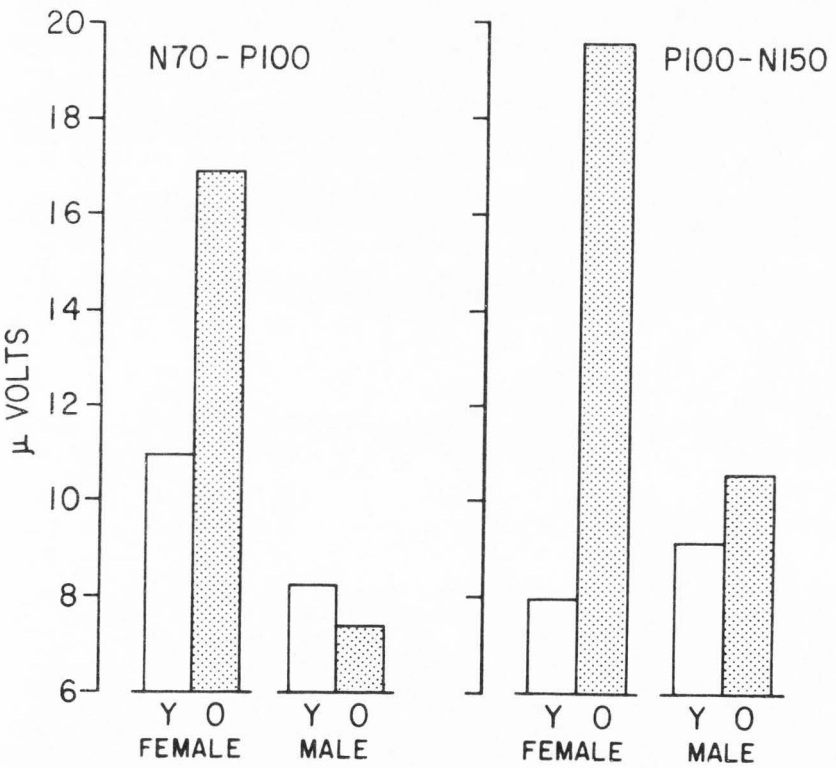

Figure 18. PREP amplitude comparisons.

other group mean differences.

P300

The following P300 components evoked by target stimuli were investigated: for latency, P1, N1, P2, N2, and P3; and for amplitude, P1-N1, N1-P2, P2-N2, and N2-P3. Results were analyzed by an age X sex X area (Fz, Cz, Pz) ANOVA. Figure 19 portrays group averaged P300 evoked potentials recorded from Cz for each of the four subject subgroups, i.e., young females, young males, old females, and old males.

P300 Latency

Age. The age effect was consistent; latencies for young adults were significantly shorter than those for the oldsters for P1, N1, and P3. Main effects are reported in Table 13.

Sex. Gender was not a significant source of P300 latency variance.

Area. Area had a significant effect on P2 and N2 latencies (see Table 13). Post hoc comparison revealed that,

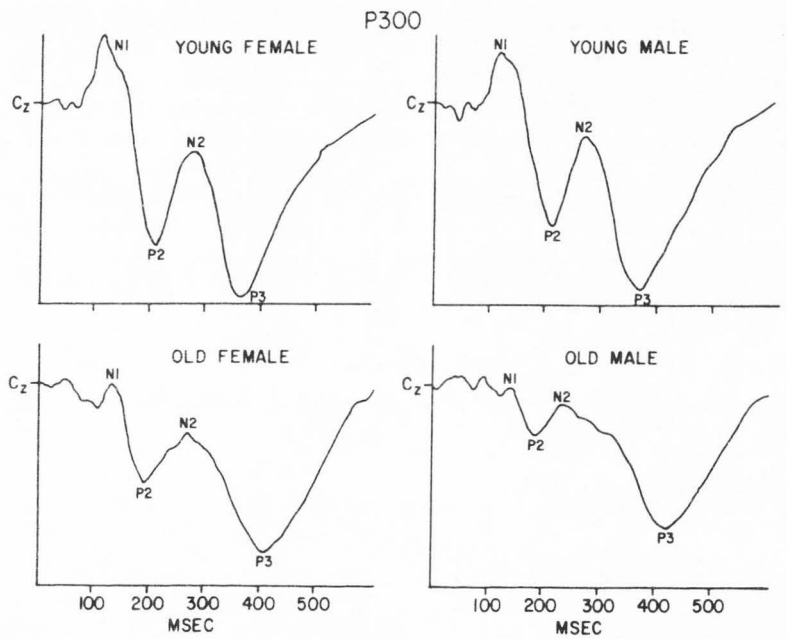

Figure 19. P300 evoked potential responses to target stimuli recorded from central (Cz) scalp.

Table 13

Summary of P300 Latency (msec) Analyses

Component	Age		Sex		Area		
	Young	Old	Female	Male	Fz	Cz	Pz
P1							
MN	86.7	106.6	97.1	96.2	96.5	95.0	98.5
SD	22.9	20.6	22.9	25.0	26.5	24.8	20.3
F	19.85		.04		2.07		
P	< .001		NS		NS		
N1							
MN	130.1	142.9	135.4	137.6	135.0	134.8	139.7
SD	19.9	28.9	22.7	28.2	29.1	26.2	20.7
F	6.77		.19		3.02		
P	< .01		NS		NS		
P2							
MN	214.7	210.9	212.2	213.4	215.3	211.4	211.6
SD	16.1	31.2	20.8	28.4	25.6	24.5	24.6
F	.49		.05		4.97		
P	NS		NS		< .01		
					Fz>Cz,Pz**		
N2							
MN	277.1	280.0	279.2	278.0	283.9	279.0	272.9
SD	24.3	37.3	24.9	37.0	33.3	31.1	29.2
F	.19		.03		13.2		
P	NS		NS		< .001		
					Fz,Cz>Pz**		
					Fz>Cz*		
P3							
MN	375.7	419.5	394.7	400.5	397.2	397.2	398.4
SD	31.3	34.6	37.9	41.1	38.2	39.6	41.3
F	37.46		.64		.25		
P	< .001		NS		NS		

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for electrode site.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for area F-ratios.

for both P2 and N2, responses recorded from the frontal electrode site (Fz) were significantly delayed relative to responses from either central (Cz) or parietal (Pz).

Interactions. Age and area interacted significantly for P1 latency, F (2, 152) = 4.94, $p < .01$. Figure 20 illustrates that age differences were not constant across recording sites. Compared to the older subjects, P1 latency for young subjects occurred 21 msec earlier for Fz and 25 msec earlier for Cz, but only 14 msec earlier for Pz.

P300 Amplitude

Age. Two components, N1-P2 and P2-N2, exhibited significant age effects. In both cases, amplitudes for young adults were larger than amplitudes for older adults, $p < .001$ (see Table 14).

Sex. Significant sex effects were found for N1-P2. Mean amplitude for females exceeded that for males, 17.3 vs. 14.5 uv (see Table 14).

Area. The area effect was highly significant, $p < .001$, for all components. (Mean values are listed in Table 14.)

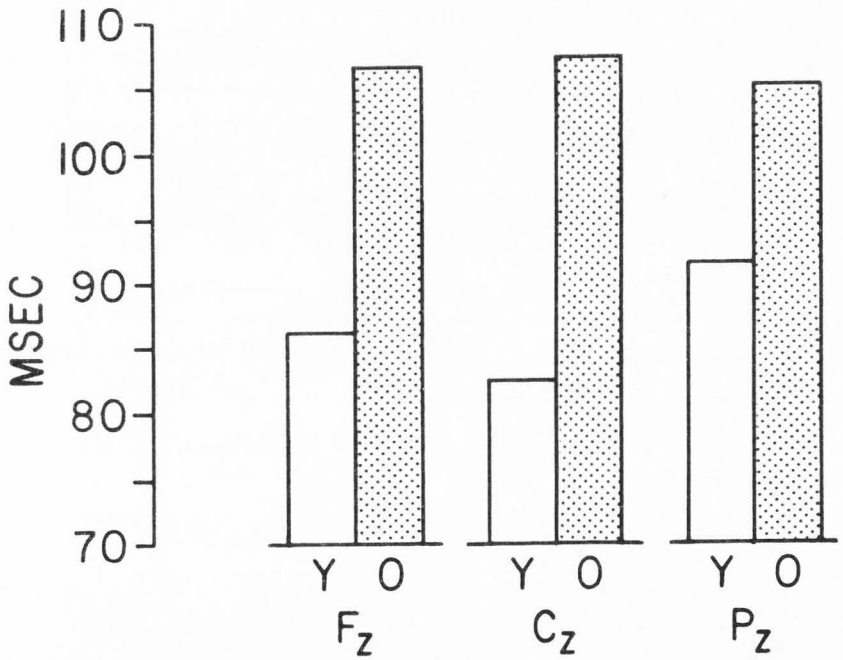

Figure 20. P300 latency for P1: age X area interaction.

Table 14

Summary of P300 Amplitude (uv) Analyses

Component	Age		Sex		Area		
	Young	Old	Female	Male	Fz	Cz	Pz
P1-N1							
MN	8.1	6.8	7.8	7.1	6.1	7.6	8.5
SD	3.9	4.6	4.6	3.9	3.5	4.1	4.8
F	2.93		.77		15.0		
P	NS		NS		< .001		
					Cz, Pz>Fz**		
					Pz>Cz*		
N1-P2							
MN	19.4	12.5	17.4	14.5	13.9	17.2	16.8
SD	7.4	6.6	7.6	7.7	6.2	8.5	8.1
F	26.02		4.73		20.83		
P	< .001		< .05		< .001		
					Cz, Pz>Fz**		
P2-N2							
MN	12.1	8.2	10.5	9.8	9.5	11.1	10.0
SD	4.9	5.4	5.8	5.1	5.1	5.9	5.3
F	13.42		.49		8.59		
P	< .001		NS		< .001		
					Cz>Fz, Pz**		
N2-P3							
MN	17.1	14.6	15.9	15.8	14.1	17.3	16.1
SD	6.5	6.2	5.8	7.1	5.7	6.8	6.5
F	3.58		.00		25.14		
P	NS		NS		< .001		
					Cz>Fz, Pz**		
					Pz>Fz**		

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for electrode site.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for area F-ratios.

Interactions. Analysis of the N1-P2 response component revealed an age X area interaction, F (2, 152) = 4.98, $p < .01$. As illustrated in Figure 21, N1-P2 amplitude was larger for young than for old subjects for all electrode sites, but differences varied considerably across sites. Young vs. old amplitude differences for Fz, Cz, and Pz were 5.3, 8.8, and 6.8 uv, respectively.

Sensory Thresholds

An individualized level of stimulus intensity should provide equivalent receptive stimulus impact, and was thus the preferred method for stimulus administration. Age and sex differences in sensory thresholds were investigated by t-test. No gender differences in sensory performance were found in this subject sample. However, the oldsters had significantly lower levels than the young adults for each sensory function evaluated, i.e., for visual acuity, visual threshold, and auditory threshold (for 78 df, t = 3.24, $p < .01$; 7.95, $p < .001$; 5.93, $p < .001$, respectively). Thus, although the older subjects were considered healthy for their age, they nevertheless displayed sensory deficits associated with old age.

In general, EEG and evoked potential measures of central nervous system processing revealed both age and gender

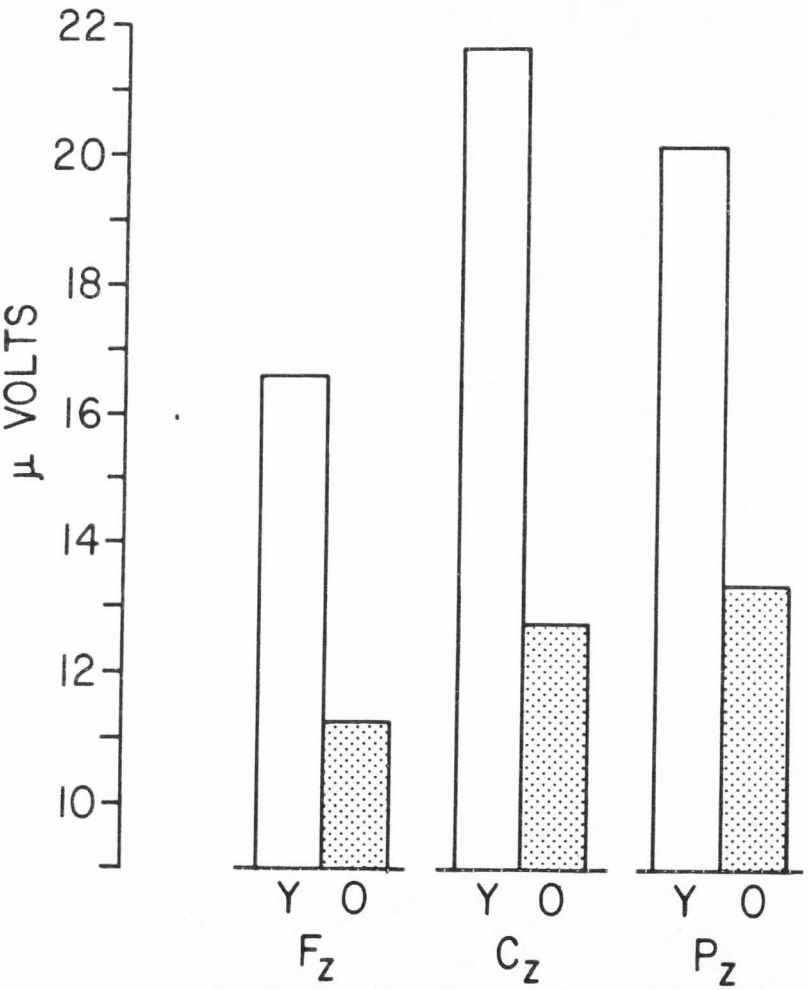

Figure 21. P300 amplitude for N1-P2: age X area interaction.

differences. The most distinguishing patterns were for females to respond with faster, larger waveforms than males, and for older subjects to display slower, larger waveforms than their younger counterparts. Results are summarized in Figure 5 and in Table 15.

Table 15

Summary of Significant Results

	Age	Sex
Power Spectral Analysis	NS	NS
Cortical Coupling	O>Y	NS
BAEP		
latency	NS	M>F
amplitude (I)	Y>O	F>M
(III, V, VII)	O>Y	
IPL	NS	M>F
VEP		
latency	O>Y	M>F
amplitude	O>Y	F>M
A/I	O>Y	NS
PREP		
latency	O>Y	NS
amplitude	O>Y	F>M
P300		
latency	O>Y	NS
amplitude	Y>O	F>M

CHAPTER V

DISCUSSION

In investigating age and sex differences, an interesting consideration arises that has social and political implications. Because a significant difference infers inequality, does a significant electrophysiological result confer a value judgment? If men and women differ, what is the impact? If young adults and oldsters differ, what might be the implications? As a preliminary, cautionary statement, one must realize that statistical significance in research is not necessarily equivalent to a significant difference in daily living skills. Furthermore, within group differences may actually be larger than between group differences, such that an individual's brain information processing cannot be assumed or predicted based on group membership.

In general, variability in electrophysiological responding was affected by age and gender (see Figure 5; Table 15). Both the female response pattern (faster, larger waveforms) and the response pattern for older subjects (slower, larger waveforms) can be discussed in terms of male/female and young/old physiological differences. These differences will be considered separately. As in previous chapters, discussions of findings unique to the different electrophysiological measures

will be found within subsections. Possible explanations accounting for specific results are offered.

Physiology

Age

As previously reviewed, a number of neuropathological changes have been associated with advanced age. A general slowing of function has been typified by electrophysiological latency data, where oldsters responded with longer peak delays than younger subjects. In this study, oldsters displayed longer latency responses than young adults for visual, pattern reversal, and P300 evoked potentials. This trend parallels other deterioration in the aging brain: reduced cerebral blood flow, metabolism, and oxygenation; cell loss in cortical sites; and decreased neurotransmitter function. These pathological changes are likely related to reports of slower reaction time, impaired neuropsychological function, and central inhibitory deficits.

Increased amplitude for oldsters was expressed in BAEP, VEP, A/I slope, and PREP results. However, the opposite effect occurred for the P300 measure: older adults' amplitudes were smaller than those for younger people. Several factors

appeared to contribute to the age effect on amplitude. The logic for a neurochemical explanation of significant results proceeds as follows: monoamine oxidase platelet levels are elevated in old age; because MAO is involved in catecholamine degradation, increased levels of MAO are associated with reduced levels of dopamine; decrease in dopamine, a catecholamine which can act as an inhibitory neurotransmitter, may mean reduced inhibitory function, or relatively greater excitation; cortical excitability has been associated with increased amplitude (Buchsbaum et al., 1977; McGeer, 1981; Robinson et al., 1977; Scheibel & Scheibel, 1975; Shafer & McKean, 1975; Vaccari, 1980). Neuropathological changes accompanying old age could thus result in heightened CNS excitability, with resulting increase in evoked potential amplitude. Clinical support can be found in Down's syndrome, phenylketonuria, and Parkinson patients, all with catecholamine deficiencies, and all with evoked potential amplitudes larger than those of control subjects (Callner, Dustman, Madsen, Schenkenberg, & Beck, 1978; Glidden, Busk, & Galbraith, 1975; Shafer & McKean).

As might be anticipated from anatomical knowledge, there were differences in responses measured from different areas of the brain. Interestingly, a topographical anterior/posterior dichotomy was characteristic of power spectral analysis, cortical coupling, and VEP data. Results were apparently confounded by the aging process: in general, the response

recorded from frontal cortex was greater for the oldsters, whereas the response recorded from occipital cortex was greater for the young people. It seems possible that anterior/posterior differences may be influenced by an age-related loss of inhibitory function perhaps related to earlier and/or greater decrements in frontal cortex (neuropathological and neuropsychological studies have shown that the frontal cortex appears more sensitive to age-related decline [Bondareff, 1977; Brody, 1973]). For young adults in this study, there was a greater variability of cortical coupling, power spectral analysis, and visual evoked potential measures across recording sites than for the older subjects whose level of responsivity was more homogeneous. This relative interarea homogeneity for the oldsters may be hypothesized to relate to a loss of inhibitory function, and a concomitant degradation of functional autonomy. The combination of greater amplitude and greater dispersion found in this study offered further credibility to the relationship between decreased inhibitory functioning and increased age.

Similarly, Gaches (1960) found greater spread of alpha activity across electrode sites for older subjects than for younger subjects; Drechsler (1978) reported more generalized responses for somato-sensory evoked potentials for oldsters compared to younger adults. Noting particular frontal and temporal decrements, Obrist (1976) speculated that cerebral ischemic and metabolic deficits may exaccerbate functional

losses in the elderly. The general observation of breakdown of functional autonomy of older people may reflect a loss of central inhibitory function, supported by the finding of greater interarea similarity of EEG power for older people. The relatively greater variability demonstrated by the young may be related to specialization of functional areas of the "healthy" brain, compared to a degredation of autonomy across electrode sites for oldsters.

Sex

As previously discussed, numerous gender differences have been postulated to account for significant electrophysiological differences between males and females. The sex-related report of shorter latencies for female responses was supported by BAEP peak latency and IPL results as well as those for VEPs. Despite several studies which found factors of head size, body weight, and length of conduction pathways to be insignificant, others still attributed shorter latency for females to the smaller anatomical structures of women (see Review of Literature). Furthermore, females are known to have higher catabolism and metabolic turnover rates than males, which may well contribute to greater efficiency in neural transmission.

Additional gender differences which may impact evoked potential amplitude as well as response latency include: a

longer life span, faster blood flow, higher deep body temperature, a larger splenium, and higher levels of MAO (platelets and plasma). An association between MAO level elevations for females and enhanced amplitudes follows the same logic as described for the oldsters' increased amplitudes. The finding of greater amplitude for females than for males was consistent across evoked potential measures, occurring for BAEPs, VEPs, PREPs, and P300s.

A cursory observation might suggest that females and oldsters shared a common factor which increased amplitude. However, a finer examination showed separate response patterns. Female amplitude was greater for earlier waves for VEPs from occipital scalp (Oz), presumably associated with direct sensory reception. Amplitude for oldsters was greater for anterior cortex (Fz) for earlier waves, as if sensory reception was more dispersed, or generalized, as might be associated with loss of functional autonomy. Speculatively, the female response pattern (faster, larger waveforms than males' responses) may represent a "healthier" process than the older subjects whose responses were larger but slower, possibly representing the pathological compromise of the aging process.

EEG

Power Spectral Analysis

EEG power spectral analysis showed no sex or age effects in any of the frequency bands studied. These results can be considered supportive of the normalization phenomenon whereby age and sex differences characteristic of childhood diminish with maturation, disappearing by adulthood (Eeg-Olofsson 1971; 1980). This trend may reverse so that in old age variability may again increase. Although some researchers such as Rodin et al. (1965) have reported alpha wave slowing associated with aging, and alpha slowing more for males than for females, the present study did not find power spectral analysis sensitive to such differences. In comparing results, methodological differences may account, in part, for lack of replication. Rodin and his colleagues analyzed EEGs which were the background rhythms to visual evoked responses (alpha correlated positively with amplitude); present EEGs were recorded while subjects were at rest with no stimulation. EEG rhythms have been reported to vary as a consequence of task (Walker, 1980). Actual data analysis procedures differed as well (frequency analysis vs. power spectral analysis). Furthermore, because many oldsters in good health may show no EEG changes (Mankovsky & Belonog, 1971), absence of age-related EEG changes may have

reflected the relatively good health of the older subjects in the present study.

Investigation of the age X area interaction for PSA, however, did reveal significant age differences. Computing a homogeneity score (the standard deviation of power spectral analysis loadings across the four electrode sites), as compared to the younger subjects, power was significantly more homogeneous for the older adults for all frequency bands (see Figure 4).

The significance of the area effect in EEG power spectral analysis highlights the need to report electrode site. Replication of results necessitates similar recording measures.

Cortical Coupling

Age. Analysis of EEG by cortical coupling revealed that age was a highly significant factor. Older adults produced higher group cortical coupling values, indicating greater similarity among areas than did the younger adults. For eight of the ten EEG electrode pairings, cortical coupling values of the older adults were significantly higher, suggesting greater homogeneity of EEG patterns than for the younger group. These results parallel those obtained from the EEG power spectral analysis and lend support to a theory of weakened inhibitory function in old age.

Electrode site was also a significant factor. Looking at the cortical coupling means, ranked pairs which seemed to be most "communicative" are primarily frontal and central sites (Fz-Cz; Cz-Pz; Cz-C3) while the least "communicative" are notable in their distance from the occipital site (Fz-Oz; Cz-Oz; Oz-C3). Such disparity in location of "communicative activity" may reflect differential aging in cortical sites; e.g., frontal and central areas demonstrating greater and/or earlier inhibitory decrements than the occipital area for brain information processings. Age-related topographical differences are consistent with speculation of diminished inhibitory capacity in senescence (Beck et al., 1980; Dustman et al., 1981; Dustman, 1984). It seems possible that the visual system may retain its specialized function, whereas anterior cortical areas may decline relatively more rapidly by the sixth decade. This would be consistent with known age-related decrements in frontal cortex.

Brainstem Auditory Evoked Potential (BAEP)

Age

There have been some contradictory reports regarding the effect of age on BAEP latency. The present findings of no

age-related latency or IPL effects supported the study by Otto and McCandless (1982), and contrasted with reports of longer latency for older subjects (Jerger & Hall, 1980; Kjaer, 1980a). Differences in laboratory conditions and experimental paradigms may influence results. As noted by Campbell et al. (1981), BAEP stimuli may be presented at different intensities, frequencies, durations, distances, and phases. Presentation may be at hearing level (HL), sound pressure level (SPL), or sensation level (SL). Subjects in the present study were carefully screened for general medical history, medications, and auditory thresholds. However, formal audiometrics were not possible under present laboratory conditions. This may have been a limitation for the BAEP wave latency and amplitude analyses, although IPLs would not have been affected since they have been shown to be independent of stimulus intensity and conductive hearing loss (Otto & McCandless; Rowe, 1978). Furthermore, the absence of age-related latency effects may have reflected the general state of health of this elderly sample.

A wave-specific, age-related effect occurred for BAEP amplitude. For Wave I, amplitude was greater for young adults, while for Waves III, V, and VII, amplitude was greater for older adults. Wave I is believed to reflect activity from the acoustic nerve, more related to direct sensory perception, whereas Waves III, V, and VII are more associated with information transmission and processing functions (superior

olivary complex, inferior colliculus, auditory cortex). Speculatively, the increased amplitude for the earlier Wave I may relate to sensory responsivity for the young adults, considered indicative of efficient functioning, compared to the increased amplitude for later waves for the older subjects, possibly reflecting loss of inhibitory function. The component-specific BAEP response pattern may be worthy of further investigation.

As with many EP measures, it is important to evaluate the same response component when comparing research studies. Some studies reported only Wave V results (e.g., Jerger & Hall, 1980); other studies have used IPLs for comparisons (e.g., Stockard et al., 1979).

Sex

Amplitude for females exceeded that for males for Waves I, IV, V, and VII. Females displayed shorter latency responses for Waves II, III, IV, V, and VII. Thus, females' responses were generally larger and faster than males', confirming the conclusions of other investigators (see Review of Literature). Summarizing previous studies of sex effects, Stockard et al. (1978) theorized that conduction differences may be due to a shorter female posterior fossa auditory pathway, independent of observable physical measures (head size, body length, etc.).

The significant IPL sex effect (males with slower conduction times) found for Waves I-III and I-V replicates those from other laboratories. Since the IPL is not affected by body length (Kjaer, 1979) or head size (Edwards et al., 1983), it is a particularly useful comparator for sex differences. In fact, the replication of mean IPL time is remarkable: mean IPLs (I-V) from 8 different laboratories averaged 4.05 msec (Kjaer; Rowe, 1978) compared to the present finding of a 4.06 msec mean IPL. The published I-III IPL mean of 2.02 msec for females and 2.13 msec for males (Campbell et al., 1981) was closely replicated by the present 2.04 msec mean IPL for females and 2.16 msec for males.

Visual Evoked Potential (VEP)

Age

A graphic summary of VEP results was presented in Figure 6. The direction of the age effect was consistent: latency was significantly shorter for young adults than for older adults. In general, the effect of age on amplitude was also consistent: young adults expressed smaller amplitudes than oldsters. Thus, the characteristic young people's VEP responses were faster and smaller than those for older

individuals.

More specific consideration of recording areas and of components yielded secondary observations. Recordings from frontal scalp showed age effects for P100, N130 and P200 latency, and for N90-P100 and P100-N130 amplitude, compared to recordings from occipital scalp which showed similar age effects for P200 latency and for P100-N130 and N130-P200 amplitude. Beck and Dustman (1975) found similar age-related increases in latency for later VEP components which they related to an increase in sensory processing time. The impact of age appeared to occur for relatively more and for earlier response components for frontal than for occipital cortex. Such results are compatible with theories that frontal associative cortex may be more affected by the aging process (decrements in inhibitory functioning, etc.), or that perhaps the visual system is more resistant to decline.

Analysis of the age X intensity interactions for VEP P100 and N130 latencies at Oz revealed an interesting age effect. The young adults responded with relatively greater change in latency across intensity than did the older adults (see Figure 8), again suggesting greater homogeneity for the responses of the oldsters.

Sex

Latency for females was consistently shorter than that for males; the effect was more extensive for the frontal area. That is, all components recorded from Fz showed gender effects whereas only N80 and P200 were the affected components for Oz. Female VEP amplitude for frontal recordings for N130-P200 and for occipital recordings for N80-P100 exceeded that for males. Thus, the gender effect for amplitude was area and component specific.

Results from Shagass & Schwartz's (1965) pioneering study revealed a similar gender finding: females had larger, faster responses than males. They rejected the explanation of shorter female conduction pathways as insufficient to account for sex differences. Amplitude was discussed in terms of a combination of excitatory and inhibitory functions: females may have demonstrated greater excitation, or less inhibition, or both. The debate continues, however, as Allison et al. (1983) assessed sex differences in VEP P100 latency by calculating a male/female brain volume ratio and concluded that brain size, and assumed proportional sensory pathway differences, could account for shorter female latency responses.

In summary, visual evoked potentials were sensitive to effects of age and sex. The characteristic differences were consistent: young adults responded with greater amplitudes and shorter latencies than older adults; females responded with

greater amplitudes and shorter latencies than males. Brighter flash intensities were also associated with larger, faster responses.

A surface observation might suggest that females and oldsters shared a common factor which increased amplitude. However, a closer examination showed separate site and component response patterns. VEP amplitude for oldsters was greater for earlier waves at anterior cortex (Fz) but later waves at posterior cortex (Oz). For females, however, amplitude was greater for later waves at anterior cortex (Fz) and for earlier waves at posterior cortex (Oz), as detailed in Tables 7 and 9. Because of the localization of function of the brain, VEPs from Oz have been considered reflective of sensory processes while recordings from Fz have been thought to reflect more associative processes.

This anterior/posterior difference in age-related response may be considered supportive of the power spectral homogeneity and cortical coupling findings of greater interarea coherence for older adults. The earlier portions of the response component (stimulus onset-P100) have been associated with sensory receiving activity, while later components (P100-N250) have included information transmission correlates (Beck, 1975). Lesevre & Joseph (1980) theorized the following component origins: N65 in primary visual cortex area 17; P100 in visual association area 19; and N150 and P200 in visual association area 18.

General physiology suggests that the females may have demonstrated a more excitatory response directly related to stimulus presentation (greater Oz amplitude for earlier components), whereas the oldsters may have reflected a loss of inhibitory function associated with the more generalized response (increased Fz amplitude for earlier components).

Amplitude/Intensity Slope

As noted previously, some individuals respond to increased stimulus intensity with an increase in VEP amplitude (augmenters), while others respond with a relative lack of an increase or a decrease in VEP amplitude (reducers).

The age X sex X area ANOVA calculated on A/I slope revealed that oldsters exhibited greater increase in amplitude across intensity for the P100-N130 component than did the young people. In addition to the age-related augmentation, the N130-P200 area effect generally supported the relationship between age and area found by Dustman et al. (1982) and Dustman and Snyder (1981). Oldsters seemed to display greater augmenting than did young adults for frontal recordings, but for recordings from occipital scalp, the young adults appeared to show a greater augmenting response.

However, when analyzed by an age X sex ANOVA, the present data did not reveal significant differences for young vs. old

at either frontal or occipital findings. Dustman and his colleagues found that oldsters were augmenters for anterior electrode sites, although no age effect occurred for occipital electrode site. Thus, both studies seemed to suggest that anterior cortical areas may appear more susceptible to the influences of aging as measured by A/I slope. As explained by Knorring and Perris (1981), the neurophysiological mechanisms related to the augmenting/reducing phenomenon seem two-fold: direct visual reception of sensory stimulation (occipital); and a modulating system activated by corticofugal impulses which are believed to inhibit afferent relays and the ascending reticular activating system (anterior). Data from this study is compatible with the assumption of age-related deficits in central inhibitory function that may relate to problems in anterior cortex which has an inhibitory influence over the ascending reticular formation (Scheibel & Scheibel, 1976). Thus, results can be interpreted as further support of the association between deterioration of inhibitory mechanisms and age. Evidence of a similar expression of inhibitory deficits has been reported clinically for alcoholics (Knorring & Oreland, 1978) and Down's syndrome patients (Callner et al., 1978; Glidden, et al., 1975); both groups have been compared to the aging population in loss of inhibitory functions and anterior cortical decrements, and both evidence larger amplitude excursions than normals.

Two main effects emerged from analyses of A/I slope for

occipital VEPs: oldsters were augmenters relative to young adults, and women were augmenters relative to men (see Figure 13). Knorring (1978) and others have postulated that EP augmentation is related to levels of excitatory/inhibitory neurotransmitters, expecially the catecholamines. Increased levels of the enzyme monamine oxidase have been associated with an augmenting VEP response (Knorring & Perris, 1980). Females are also known to have higher catabolism and turnover rates than males (Knorring & Perris; Vaccari, 1980) which may well contribute to enhanced levels of excitation.

VEPs Elicited by Patterned versus Unpatterned Stimuli

Shafer & McKean (1975) provided balanced stimulation of monoaminergic activity in phenylketonuria patients, known to have a catecholamine depletion, and thereby increased their ability, as determined from VEPs, to differentiate patterned from unpatterned stimulation. The relationship was thus established between visual differentiation and monoamines. The Shafer & McKean model was supported by present results and similar findings obtained by Dustman et al. (1981), who related life-span alterations in the visual evoked potential to the reduction in inhibitory functioning associated with aging.

An interesting pattern of age differences was observed in the present study: compared to older subjects, younger

subjects had less differentiation for VEPs measured from Fz, but greater differentiation for VEPs recorded from Oz (see Figure 16). In the sensory receiving area, then, the young people displayed greater contrast sensitivity, while the older group displayed greater differentiation at associative cortex. This anterior/posterior pattern of age effects on pattern sensitivity also supports findings reported from other measures (EEG, VEP). Again, the young people revealed a greater sensory response hypothesized to represent a healthier central nervous system; the oldster's greater frontal response was compatible with inhibitory deficits.

Pattern Reversal Evoked Potential (PREP)

When previous research has noted age and gender differences, the most common finding has been that females demonstrated larger, faster responses than males, and that increasing age was associated with increasing latency. PREP findings from this study displayed similar overall trends, with an interesting exception.

Age

Latencies for N70 and N150 showed oldsters with slower waves. Explanations for delayed PREP latency associated with aging, reviewed by Stockard et al. (1979), include factors previously mentioned: demyelinization, axonal loss, slowed conduction velocity in the optic nerve and/or optic pathways, increased synaptic delay, neuronal cell loss, decreased pupil diameter (senile miosis), and dendritic loss. Throughout this study, increased latency for older subjects seemed to be a consistent finding from electrophysiological measures of the age effect. These results generally replicate PREP latency findings by Celesia and Daly (1977), Kriss et al. (1982), Sokol et al. (1981), and Shearer and Dustman (1980).

Sex

A highly significant sex effect occurred, but only in the older people: PREP amplitudes were substantially larger for old females as compared to old males (see Figures 17 and 18). Moreover, mean amplitude for the older females was significantly higher than the mean amplitudes for each of the other groups. This finding seemed anomolous, particularly as Kjaer (1980), Kriss et al. (1982), and others have reported a decrease in PREP amplitude associated with increased age.

However, the present results apparently represent the PREP study that investigated both age and sex, therefore identifying older females as a specific subgroup.

The reason for this anomalous finding was not clear. Because PREPs have been used clinically to assess visual system pathology, one might predict that the gender differences found in the elderly represented differences in visual acuity or visual threshold. However, t-tests on these thresholds (see Results) revealed no such differences between older females and older males. Theoretically, if the visual detection of lines, edges, and contours were defective in this sample of older women, there would also have been a sex difference for the VEP measure comparing EPs elicited by patterned vs. unpatterned stimuli; there was no significant difference between females and males. Thus, simple visual pathology did not seem to account for the unique response of the older women.

The factors which comprise this anomalous result may represent compounding of aging and gender factors and/or may result from particular subgroup characteristics not previously identified. Certainly this interesting finding would seem to bear further study.

P300

Summarizing significant P300 results, young adults responded with larger, faster responses than their older counterparts; women displayed greater amplitude responses than men (see Figures 19 and 20).

Age

In general, evoked potential measures from this study have indicated that latency was generally slowed for the older population and amplitude was usually increased. For the P300 results, latency was again longer; however, the oldsters showed a smaller amplitude response than the young adults. Although the decrease in amplitude response for the elderly group might appear contradictory to other evoked potential results, decline in cerebral functioning, particularly the loss of inhibitory functioning, is nevertheless conjectured to contribute to the age effect. Because the P300 has reflected more central and subcortical mechanisms not directly sensory in nature, the mechanism of inhibitory influence is different for the P300 than for other EP measures. Beck et al. (1980), noting that amplitude reductions have been previously reported for the P300, attributed increased latency and decreased amplitude for

the P300 to a similar neural mechanism. They reviewed the aging process as described by the neural circuitry model of Desmedt & Debecker (1979) and the histopathology changes described for frontal cortex by Scheibel & Scheibel (1975) to formulate the following theory. An interactive loop from frontal cortex modifies the mesencephalic reticular formation which in turn reduces cortical negativity and registers electrophysiologically as the P300 evoked potential. In the aging population, frontal cortex is known to suffer earlier degredation, lessening its impact on the MRF, resulting in amplitude reduction and latency increase. An interactive loop is not incompatible with other explanations of increased P300 latency. Blom, Barth, & Visser (1980) attributed the origin of the visual late component to reticulo-cortical or thalamo-cortical influences associated with recognition and information processing; a generalized autonomic explanation is favored by Wood et al. (1980), relating P300 activity to subcortical functioning. As noted by Yakovlev & Lecours (1967), the reticular formation and certain parts of the thalamus shrink and demyelinate with increased age. Podlesny and Dustman (1982) reported a correlation between slowed reaction times, heart rate deceleration, and decrease in P300 amplitude, suggesting that a cluster of physiological indicators may be related to age deficits in sensorimotor and cognitive performance. Cognitive processes are known to decline differentially with increasing age (Botwinick, 1981;

Woodruff & Birren, 1975). For example, neuropsychological tests such as the Memory-for-Designs and some of Wechsler Adult Intelligence Scale subtests indicate cognitive decline with increased age (Blusewicz, Dustman, Schenkenberg, & Beck, 1977; Blusewicz, Schenkenberg, Dustman, & Beck, 1977; Dustman & Beck, 1980). It has been suggested that the association of greater P300 peak delay with aging may result from loss of ability to cope with cognitive demands, or increased time needed to evaluate stimuli (Ford et al., 1982).

The finding of significant age X area interactions offered further support for a theory of differential aging, i.e., decrements associated with the aging process may be location and/or function specific.

Sex

The gender finding for P300 evoked potential, i.e., amplitude for females was greater than that for males, has been typical of other sensory measures found in this study. When reported from other research, amplitude sex effects have been consistently in this direction. Most recently, Picton et al. (1984) noted similar gender effects, female subjects producing greater amplitude response compared to males. Previously discussed factors of the excitation/inhibition balance may also be speculated to account for P300 amplitude

gender differences.

Limitations

As a specific limitation, the lack of audiometry available under current laboratory conditions may have influenced the amplitude and latency results for the brainstem auditory evoked potentials.

As a more general concern, the definition of "healthy, aging" subjects is somewhat debatable. Subject selection in an older age range, such as 70-80 years, might have yielded greater significant differences when compared to young adults. However, the covariance of age and health raises the question of whether health should be considered relative to age. If, for example, selection is limited to oldsters who have never been hospitalized, or take no daily medication, the sample would probably not be representative of the general population in that age range. Such subjects would be "super-healthy," rather than typical.

Theoretically, a researcher is limited only by lack of creativity. Pragmatically, however, there are resource constraints of time, money, and energy availabile. In realistically reviewing the implementation of the present study, the limitations seemed primarily pragmatic ones.

Certainly, a larger sample size would have been desirable. Additional measures such as auditory P300, auditory amplitude/intensity slope, and somatosensory evoked potentials would have complemented the current results and allowed further interpretation.

Suggestions for Future Research

As previously mentioned, the effects of age and gender are not yet well documented for a number of electrophysiological measures. The variety of experimental paradigms and laboratory conditions have complicated replication efforts. Further investigation into the individual differences which may influence research results and clinical applications is necessary.

An area for further research which seems specifically suggested by the present study would be the anomalous PREP amplitude response for the older females, which exceeded that for all other groups rather dramatically. Additional investigation might indicate whether the increased amplitude could be considered typical of this subgroup, and possibly offer speculation regarding contributory factors.

An analysis of the variability of responses, perhaps as evaluated by analysis of standard deviations, would offer

additional information regarding not only age and sex differences, but also site and component differences. Such analysis might extend the exploration of the concept of differential aging, particularly as a means for understanding the factors and processes associated with advancing age.

Another interesting area for future research would be the assessment of information processing asymmetry, comparing responses measured from the left hemisphere of the brain with those from the right hemisphere (e.g., F3, F4; C3, C4; O3, O4) in an age X sex paradigm. Do females and males process information differently, as preliminary research suggests? Are there age X sex interactions which might indicate that females and males age differently, as some biological research suggests?

While investigators continue to unravel the mysteries of individual differences, a complex, interactive theory would seem more plausible than a limited, simplistic explanation to account for the aging and gender differences found in this study. Nevertheless, exploring the relationship of neurochemical factors to the electrophysiological excitation/inhibition balance appears to be a generally promising avenue for future investigation. Speculatively, appropriate biochemical intervention may have the capacity to enhance function. The human brain is so sophisticated that electrophysiology can reflect levels of sensory as well as processing functions. Particularly in reviewing the P300

measure, the domain of cognition can also be included.

Not only deficits, but also rehabilitative increments can be investigated by EEGs and evoked potentials. For example, Dustman et al. (1984) found improved neuropsychological function in older individuals who participated in aerobic exercise training. Recently, Gummow, Dustman, and Keaney (in press) used electrophysiological measures to assess remote effects of cerebrovascular accidents.

Other directions for electrophysiological investigation include: brain dysfunction and clinical diagnostic applications; topographical mapping of brain function; normal and pathological psychophysiology; biocybernetics (a psychophysiological communication link between humans and computers); and transdermal stimulation to enhance nerve regeneration.

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APPENDICES

Appendix A

Patient's Personal History

PATIENT'S PERSONAL HISTORY

Patient No. _____

Date _____

Confidential Record: Information contained here will not be released except when you have authorized us to do so.

Last Name	First	Middle	Birth Date	Birth Place

Address	City	State	Zip	Home Phone	Business Phone

Occupation	Medicare No.	Medicaid No.	Sex	Marital Status	Religion
			M F		

Insurance Company	Insurance No.

Person to Notify _____ Relationship _____

Address _____ Phone Number _____

Date of Last Physical Examination _____ Doctor _____

Family or Referring Physician _____ Address _____

FAMILY HISTORY		If Living		If Deceased	
	Sex	Age	Health	Age at Death	Cause
Father					
Mother					
Brothers/Sisters* *(Circle Sex)*					
	M F				
	M F				
	M F				
	M F				
	M F				
Husband/Wife					
Sons/Daughters* *(Circle Sex)*					
	M F				
	M F				
	M F				
	M F				
	M F				

**Since some names may be used for either men or women, please circle sex for each Brother, Sister, Son or Daughter.*

Do you know of any blood relative who has or had: (Circle and give relationship)

Stroke	_____	Epilepsy	_____	Heart Attack	_____	Nervous breakdown	_____
Cancer	_____	Suicide	_____	Stomach ulcers	_____	Rheumatic heart	_____
High blood Pressure	_____	Migraine	_____	Kidney disease	_____	Insanity	_____
Tuberculosis	_____	Asthma	_____	Goiter	_____	Congenital heart	_____
Diabetes	_____	Hay fever	_____	Arthritis	_____		
Leukemia	_____	Bleeding tendency	_____	Colitis	_____		

PERSONAL HABITS: (Circle)

Yes No Do you regularly smoke? Cigarettes ☐ Pipe ☐ Cigars ☐ For how many years? _____

Yes No Do you usually drink over 6 cups of coffee per day?

Yes No Do you regularly drink alcohol? 1 oz. per day ☐ 2 oz. per day ☐ 4 oz. per day ☐ over 6 oz. ☐;
BEER: 1 bottle per day ☐ 2 bottles per day ☐ over 4 bottles per day ☐.

Yes No Do you have difficulty in falling asleep?

Yes No Do you awaken early in the morning without apparent cause?

MEDICATIONS:

Are you presently taking any of the following medications? (Circle)

Yes	No	Aspirin, bufferin, anacin	Yes	No	Tranquilizers
Yes	No	Blood pressure pills	Yes	No	Weight reducing pills
Yes	No	Cortisone	Yes	No	Blood thinning pills
Yes	No	Cough medicine	Yes	No	Dilantin
Yes	No	Digitalis	Yes	No	Shots
Yes	No	Hormones	Yes	No	Water pills
Yes	No	Insulin or diabetic pills	Yes	No	Antibiotics
Yes	No	Iron or poor blood medications	Yes	No	Barbiturates
Yes	No	Laxatives	Yes	No	Birth control pills
Yes	No	Sleeping pills	Yes	No	Phenobarbital
Yes	No	Thyroid medicine	Yes	No	Other drugs not listed

Write in the names and year of any operations which you have had:

Name any drugs to which you are allergic:

Write in the names of any diseases you have had which required hospitalization:

Serious Illnesses which you have had: (not requiring hospitalization)

Serious injuries or accidents:

To be answered by WOMEN only: (Circle)

Yes	No	Are you still having regular monthly menstrual periods?	
Yes	No	Have you ever had bleeding between your periods?	When? _____
Yes	No	Do you have very heavy bleeding with your periods?	When? _____
Yes	No	Do you feel bloated and irritable before your period?	
Yes	No	Are you now on or have you ever taken the birth control pill?	When? _____
Yes	No	Have you ever had a miscarriage?	When? _____
Yes	No	Have you ever had a discharge from the nipple of your breast?	When? _____
Yes	No	Do you regularly have the cancer test of the cervix?	Date of last test _____

How many children born alive _____
How many stillbirths _____
How many premature births _____
Date of last menstrual period _____

How many miscarriages _____
How many cesarean operations _____
Any complication of pregnancy _____

To be answered by men and women: (Circle)

Yes	No	Do you frequently have severe headaches? (If yes, answer the following):
Yes	No	Do they cause visual trouble?
Yes	No	Do they occur on one side of the head?
Yes	No	Do they awaken you at night from sleep?
Yes	No	Do they feel like a tight hat band?
Yes	No	Do they hurt most in the back of the head and neck?
Yes	No	Does aspirin relieve them?

Yes	No	Have you ever fainted?	Yes	No	Have you ever had a convulsion?
Yes	No	Spells of dizziness?	Yes	No	Double vision?
Yes	No	Spells of weakness of an arm or leg?	Yes	No	Pains in ear?
Yes	No	Ringing in ears?	Yes	No	Nosebleeds?

Yes	No	Do you frequently have bleeding gums?	Yes	No	Do you frequently have a sore tongue?
Yes	No	Do you frequently have trouble swallowing?	Yes	No	Do you frequently have nausea and vomiting?
Yes	No	Do you frequently have hoarseness?			

Have you ever had shortness of breath?: (Circle)

Yes	No	Doing your usual work?	Yes	No	Which causes you to cough?
Yes	No	Climbing a flight of stairs?	Yes	No	Accompanied by wheezing?
Yes	No	Which awakens you at night?	Yes	No	Have you ever coughed blood?
Yes	No	Do you have a chronic cough?	Yes	No	Do you cough up much sputum?

Have you ever had chest pain or tightness in the chest which begins when: (Circle)

Yes	No	When exerting yourself?	Yes	No	Radiates down the arm?
Yes	No	When walking against a wind?	Yes	No	Disappears if you rest?
Yes	No	When walking up a hill?	Yes	No	Occurs only at rest?
Yes	No	After a heavy meal?	Yes	No	When walking fast?
Yes	No	When upset or excited?	Yes	No	When walking in cold weather?
Yes	No	Palpitations	If you have chest pain or tightness please explain _____		
Yes	No	Do you sleep on more than one pillow?			

Have you recently had pain in the stomach which: (Circle)

Yes	No	Occurs 1 – 2 hours after a meal?
Yes	No	Is brought on by eating fried foods, gassy foods?
Yes	No	Awakens you at night?
Yes	No	Is relieved by antacid medications?
Yes	No	Is relieved with milk or eating?
Yes	No	Occurs while eating or immediately after?
Yes	No	Is relieved by a bowel movement?
Yes	No	Loss of appetite?

If you have had a change in bowel habit recently answer the following: (Circle)			When or since when?
Yes	No	Crampy pain in the abdomen?	
Yes	No	Alternating diarrhea and constipation?	
Yes	No	Pain during or after bowel movement?	
Yes	No	Mucous in the stool?	
Yes	No	Blood in the stool?	
Yes	No	Ribbon-like stools?	
Yes	No	Black stools?	
Yes	No	Require use of strong laxatives or enemas?	
Have you had: (Circle)			
Yes	No	Burning when urinating?	
Yes	No	Loss of control of bladder?	
Yes	No	Blood in the urine?	
Yes	No	Dark colored urine?	
Yes	No	Trouble starting to urinate?	
Yes	No	Trouble holding the urine?	
Yes	No	Getting up frequently at night?	
Yes	No	Passed a kidney stone?	
Have you recently had: (Circle)			
Yes	No	Pains in calves of legs when walking?	
Yes	No	Cramps in legs at night?	
Yes	No	Pain in the big toe?	
Yes	No	Varicose veins?	
Yes	No	Phlebitis or inflamed leg veins?	
Yes	No	Swelling in the ankles?	

To be answered by MEN only: Have you ever had: (Circle)

Yes	No	Loss of sexual activity? For how long? _____
Yes	No	Treatment for genitals (private parts)?
Yes	No	Discharge from penis?
Yes	No	Hernia (rupture)?
Yes	No	Prostate trouble?

Describe briefly your present medical symptoms:

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Appendix B

Procedural Protocol

Calibrate Sine Wave

1. turn computer on; load disk, 2 control "C"s
2. load tape; set tape speed @ 1 7/8
3. turn on: polygraph, oscilloscope, multi-stimulator, tape recorder
4. turn oscillator (sine wave generator) on; set @ 20 Hz; 4.0 on large dial, X5 switch; let warm up for 5 minutes
5. check EEG amplifier settings: channels 1-6 Lo @ 1 HZ, Hi @ .1 kHz, sensitivity @ 7.5, 60 Hz filter on; channel 7 Lo @ 30, Hi @ 3 kHz, sensitivity @ 20, 60 Hz off; sec/div @ 20m; chart paper @ 15; pens @ 90; upper scope @ .5, lower scope @ 1.0.
6. connect oscillator output to jackbox; red wire into ground; silver jack in channel 7; Y-F8 on channels 1-6
7. check sine wave output on amplifiers of polygraph (J6 output), including trigger pulses for channel 7
8. check sine wave on tape recorder channels, including trigger pulses for channel 7
9. turn off subject's room lights
10. record start point number; record sine wave, including 7 trigger pulses on channel 7; record stop number.
11. turn lights back on
12. record calibration on computer: run ATD; E; sine: escape (altmode). Push trigger pulse on stimulator. "Are you sure?" - (do not push Y) - return - (will say aborted)
13. turn oscillator off
14. set VEP filters (4.0, horizontal bar, diffuse)
15. prepare equipment for subject
16. disconnect oscillator output from jackbox
17. connect jackbox to impedance meter
18. sign subject on wall list

Subject Preparation

-ask subject if would like to use restroom before begin-

1. read and sign consent forms
2. administer Bausch and Lomb vision test; record Snellen acuity.
3. introduce subject to lab and seat. Ask subject to complete physical information. Take temperature
4. glasses/contacts on; earrings off
5. measure and record head size
6. apply montage: Fz, Cz, Pz, Oz, Cz, A1, A2, FP1, FP2, ground @ C4 (test ground C4)
7. replace ground; "disconnect" on impedance meter; reconnect jackbox to chamber connector
8. unplug air hose
9. set photostimulator (reflection of eyes at dot level, centered)
10. place headset on subject (headphone jack plugged into auditory stimulator)

BAEP

1. change channels 1 & 2 behind polygraph

2. turn auditory stimulator on
3. turn fans off; turn off all lights; turn on red lamp only henceforth
4. get threshold; use multi-stimulator manually (really relax and concentrate); record
5. set threshold +70
6. change channels 1 & 2 on selector panel; best ear (A1 or A2) to Cz
7. change amplifier settings on channels 1 & 2 to 100Hz, 3kHz
8. set tape speed @ 7 1/2; leave tape recorder on channel 7 to see stimuli
9. multi-stimulator rate @ X10 (1.15)
10. (don't record on EEG paper or tape recorder)
11. instruct subject: eyes closed, may even sleep, jaw and facial relaxation - need 2,000 good trials, about 3 minutes
12. give subject sample of threshold + 70; ask if S can hear it
13. Terak: (prepared during calibration) ATD; name file, e.g., FD1:SDF01.70D; esc. If Terak dies or is stopped, reset by .run ATD; E; sinec; ATD; file name; esc
14. set multi-stimulator to repetitive
15. computer will stop after 2,000 good ones
16. turn fans on
17. with flashlight, remove headset
18. turn auditory stimulator off
19. reconnect channels 1 & 2 of polygraph
20. reset selector panel: 1, FP1-FP2; 2, Fz-A1&A2; 3, Cz-A2; 4, Pz-A2; 5, O1-A1; 6, C3-A1; 7, blank; 8, X-F7
21. reset channels 1 & 2 amplifier settings @ 1; .1
22. reset tape recorder to 1 7/8

3' EEG

1. label graph paper
2. instruct to close eyes and relax (not sleep)
3. check EEG recording - blink, swallow, etc.
4. check scope and punch through recorder channels
5. record start
6. record 3'; record stop
7. turn photostimulator on

VEP

1. instruct subject and find threshold (ask subject 1. if can see bar?; 2. is bottom on left or right? remind "I can control and change the orientation of line")
2. check randomizer chart: if threshold is 4.5, 1 log is 3.5; 2 logs is 2.5; 3 logs is 1.5
3. give subject trigger switch and instruct: depress every 10, focus on dot, no blinks, swallows, etc., relax
4. record start; begin after samples
5. manual stimulation - 50 good at 2 sec rate for each log condition
6. record stop

PREP

1. turn on my lights only; patially plug in TV; 90cm, centered screen placement
2. turn on Teleray and Terak; turn on additional fan
3. turn both stimulators off
4. replace silver jack with black jack in channel 7
5. instruct subject: focus on X in center of screen, no blinks, etc., relax
6. load Terak: control "C"s; run:patrev (screen will go blank)
7. Teleray prompt: P; (if it doesn't ask for distance, 2 control "C"s, run:patrev) 90 cm; 30'; (wait to build pattern) on-30; off-30 don't return
8. EEG paper on (1 7/8), record start number.
9. begin by turning on recorder and "return" on Teleray (look for pulse on lower trace); ask for two eyeblinks at very start to set parameters
10. then time 2 1/2 minutes of few artifacts
11. 2 control "C"s when done
12. record stop number

P300

1. change channels 1-4; Lo filters to .1 Hz; change chart paper to 30
2. reboot Terak; run QX:P300 (tiny center dot)
3. on Teleray, when "default?," Y; background = 0; target = X; duration = 3
4. instruct subject, expecially count Xs, ignore Os, report total Xs at end
5. EEG paper on; record start number; begin recorder; number by tens
6. push Teleray "return" to start sequence. If need more trials, enter 0 skips; stop when enough
7. record stop number; ask for total Xs

Clean Up

1. debrief and unhook subject; remind subject that check will come in about 6 weeks
2. replace black with silver jack; clear selector panel
3. rewind and remove tape
4. clean syringes and scalp electrodes
5. turn off all machines except computer
6. back up disk; turn off computer
7. record (log) all information, ready for analog-to-digital

Appendix C

VEP Recordings from Central Scalp

Table 16

Summary of Age X Sex X Intensity ANOVAs
for VEP Latencies (msec) from Central (Cz) Scalp

Age

Component	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
N80	82.7	(13.4)	84.1	(12.1)	.65	NS
P100	100.4	(13.1)	107.0	(14.2)	9.50	< .01
N130	125.1	(13.5)	135.9	(17.3)	16.36	< .001
P200	196.3	(18.8)	207.2	(24.9)	10.74	< .01

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
N80	80.7	(12.7)	86.0	(12.3)	8.99	< .01
P100	99.9	(14.1)	107.5	(13.0)	12.48	< .001
N130	125.8	(14.0)	135.2	(17.2)	12.60	< .001
P200	196.0	(21.1)	207.5	(22.8)	11.88	< .001

Intensity

	1 $\bar{X}$	(SD)	2 $\bar{X}$	(SD)	3 $\bar{X}$	(SD)	F	p	
N80	86.3	(14.1)	84.0	(13.2)	79.8	(9.9)	6.44	< .01	1>3** 2>3*
P100	106.3	(15.2)	103.1	(14.4)	101.8	(12.1)	3.62	< .05	1>3*
N130	134.6	(16.8)	128.4	(16.5)	128.4	(15.2)	9.54	< .001	1>2,3**
P200	207.9	(23.7)	205.4	(20.6)	192.0	(20.5)	21.42	< .001	1>3** 2>3**

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for stimulus intensity conditions.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for intensity F-ratios.

Table 17

Summary of Age X Sex X Intensity ANOVAs
for VEP Amplitudes (uv) from Central (Cz) Scalp

Age

Component	Young $\bar{X}$	(SD)	Old $\bar{X}$	(SD)	F	p
N80-P100	2.2	(2.1)	4.4	(3.2)	24.28	< .001
P100-N130	4.3	(3.4)	7.6	(5.1)	18.53	< .001
N130-P200	13.5	(5.9)	11.7	(5.8)	2.74	NS

Sex

	Female $\bar{X}$	(SD)	Male $\bar{X}$	(SD)	F	p
N80-P100	3.2	(2.8)	3.4	(3.0)	.27	NS
P100-N130	6.6	(4.9)	5.3	(4.1)	2.70	NS
N130-P200	14.3	(6.5)	10.9	(4.7)	9.80	< .01

Intensity

	1 $\bar{X}$	(SD)	2 $\bar{X}$	(SD)	3 $\bar{X}$	(SD)	F	p
N80-P100	2.4	(2.2)	3.2	(2.9)	4.3	(3.2)	22.95 3>1,2** 2>1**	< .001
P100-N130	5.5	(3.8)	5.9	(4.4)	6.5	(5.5)	NS	
N130-P200	11.7	(5.5)	12.0	(5.8)	14.1	(6.2)	11.66 3>1,2**	< .001

* $p < .05$; ** $p < .01$; Duncan's Multiple Range Test for stimulus intensity conditions.

Note. Degrees of freedom for age and sex F-ratios were (1, 76) and (2, 152) for intensity F-ratios.

VITA

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