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INFLUENCE OF AGE ON AUDITORY GATING

by

Ginny M. Smith

A thesis submitted to the faculty of

Brigham Young University

in partial fulfillment of the requirements for the degree of

Master of Science

Department of Communication Disorders

Brigham Young University

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BRIGHAM YOUNG UNIVERSITY

GRADUATE COMMITTEE APPROVAL

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BRIGHAM YOUNG UNIVERSITY

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ABSTRACT

INFLUENCE OF AGE ON AUDITORY GATING

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Department of Communication Disorders

Master of Science

This study utilized paired tones at 1000 Hz with a 500 ms interpair interval and a 10 s interstimulus interval to assess sensory gating. Forty-two participants, ranging from 3-72 years of age were used to observe maturational changes in amplitude, latency, and suppression ratios of the P50 waveform. Previous research has shown that in normal adults the amplitude in response to the second of the paired tones is significantly suppressed compared to the amplitude in response to the first tone. The current study showed amplitude decreased with age to middle adulthood, where it increased slightly to later adulthood. Latencies decreased with age. Suppression ratios decreased from childhood to adolescence, with an increase from early adulthood to later adulthood. Sensory gating would appear to be a later developing aspect of human sensory physiology. Also similar to many other brain functions, sensory gating decreases in later adulthood.

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Introduction

Sensory gating is a phenomenon that occurs in the normally functioning brain and may be measured using auditory evoked potentials (AEPs). Boutros and Belger (1999) define sensory gating as “the ability of the brain to modulate its sensitivity to incoming sensory stimuli” (p. 917). The processing of sensory input has two stages, the first being stimulus identification, or realizing that there is a stimulus present. The second stage of sensory processing is stimulus evaluation, which allows the brain to regulate the sensory input sent on for higher cortical processing (Boutros & Belger, 1999). The brain is presented with repetitive information when sensory gating is not properly functioning, as has been recognized in various studies with individuals having schizophrenia (Adler, Waldo, & Freedman, 1985; Nagamoto, Adler, Waldo, & Freedman, 1989; Siegel, Waldo, Mizner, Adler, & Freedman, 1984).

The P50 component is a middle latency auditory evoked potential (MLAEP) that occurs between 40 and 80 ms (Arnfred, Chen, Eder, Glenthøj, & Hemmingsen, 2001), and is labeled P50. The P50 scalp recordings are thought to be the summation of multiple intracranial sources, including the superficial temporal, hippocampal, and thalamic sites (Nagamoto, Adler, Waldo, Griffith, & Freedman, 1991). MLAEPs, particularly the P50, are used when studying sensory gating. MLAEPs show abnormalities in how the brain processes repetitive sensory stimuli (Boutros & Belger, 1999). The P50 is relatively insensitive to changes in a participant’s attention (Jerger, Biggins, & Fein, 1992; Nagamoto et al., 1989; Waldo & Freedman, 1986). This feature is beneficial when studying young children or individuals with psychiatric conditions, because their attention levels may be difficult to monitor.

Measurement of the sensory gating process is completed by calculating the ratio of the amplitude of the second response (S2) to the amplitude of the first (S1), and is expressed as S2/S1 (Arnfred et al., 2001). A lower ratio reflects greater inhibitory functioning and a more intact sensory gating function (Boutros & Belger, 1999). According to Boutros, Overall, and Zouridakis (1991), individuals without a history of schizophrenia typically have a P50 suppression ratio of 50% or less.

Maturation development of sensory gating has been an intriguing topic. Freedman, Adler, and Waldo (1987) investigated changes in sensory gating in participants ranging from 18 months to 55 years of age. Freedman, Adler et al. not only observed lower suppression ratios (or greater suppression) in adults vs. children, but observed less variability in the suppression ratios with maturation. For example, children 1-8 years of age showed suppression ratios that ranged from 0-100%. Freedman, Adler et al. observed that suppression ratios decreased with age (indicating more intact sensory gating with age) and that there was less variability with maturation, but used an age group that only extended from 18 months to 55 years of age. There may be further information available about maturational changes in sensory gating, if this type of study were completed using an extended age range that included participants over 55 years of age.

The purpose of the current study was to provide additional information about maturational changes of the P50 response by using participants in a higher age range than previous research. The current study used 42 normally hearing participants without a history of neuropsychiatric disorders, ranging from 3-72 years of age.

Review of Literature

Sensory Evoked Potentials

AEPs are a series of brain waves that vary in voltage as a function of time, and are recorded at the scalp following auditory stimulation. Each of the waveforms occurs along differing portions of the auditory pathway, resulting in varied response times. AEPs are classified depending on their response times (or latencies) and where the responses occur along the auditory pathway (Møller, 1994). Early AEPs, also called auditory brainstem responses (ABRs) or brainstem auditory evoked potentials (BAEPs), occur within the first 10-15 ms following stimulus presentation, and are thought to originate in the VIIIth cranial nerve and in the brainstem. MLAEPs occur between 40 and 80 ms post-stimulus (Arnfred et al., 2001), and are thought to originate in the midbrain. Late auditory evoked potentials (LLAEPs) occur from 75-200 ms and are thought to originate at the level of the cortex. Auditory event-related potentials (ERPs), including the P300, consist of responses recorded between 200 and 389 ms, and involve association areas in the brain (Martin & Clark, 2003).

AEPs can be evaluated through topographic mapping (i.e., brain mapping), latency (time relative to onset of a stimulus), polarity (positive or negative), and amplitude (peak height). Topographic mapping refers to the distribution of brainwaves across the scalp, but not necessarily where the brainwaves are originating. Recordings obtained by placing electrodes on the scalp represent “complex transformations of the summation of the electrical fields of many nerve fibers and nerve cells” (Møller, 1994, p. 23). Latency refers to the time between onsets of the stimulus and when the AEP occurs. Polarity (i.e., phase) refers to the positivity or negativity of the resulting

waveform in reference to the ambient noise baseline at a specific time interval.

Amplitude refers to the magnitude or height of the waveform.

AEPs have small amplitudes and may not be seen amidst the other electroencephalograph (EEG) activity without the use of averaging. Averaging techniques focus on the brain's responses to specific stimuli by removing unrelated activity such as muscle activity and movement artifacts. Averaging is used under the assumption that the unrelated activity occurs randomly while activity related to the stimuli is time related. The random unrelated activity is eliminated when multiple trials are averaged, leaving just the AEP (Misulis & Head, 2003).

The effects of attention are a consideration when obtaining AEPs. There are four levels of attention commonly used when obtaining AEPs. The first level of attention is selective: the participant's attention is maintained by having the participant actively monitor and discriminate between the stimuli (i.e., same-difference tasks). The second level of attention is active: the participant is asked to respond to the stimuli (i.e., pushing a button). The third level of attention is passive: the participant is to be tested while in an awake and alert state, but not necessarily attending to the stimuli. The last level of attention is an ignore condition: the participant is distracted from the stimuli. McPherson and Ballachanda (2000) noted in the MLAEP that active attention may enhance the later components to a small degree, but the effect of the various states of attention on the component being measured can be variable.

The P50 Component and Sensory Gating

Møller (1994) described the labeling process used for evoked potentials: A *P* is used for positive peaks and an *N* is used for negative peaks, while the number that

follows represents the approximate latency at which the evoked potential occurred after a stimulus presentation. Using this system, the P50 component of the MLAEP refers to the positive peak generally occurring at 50 ms after the stimulus is administered.

P50 gating refers to the attenuation of the AEP in the middle latency range, around 40-80 ms (Arnfred et al., 2001). When two identical stimuli (S1 representing the first stimulus, and S2 representing the second stimulus) are presented with a 500 ms intrastimulus interval and a 10 s interstimulus interval, S2 has a reduced P50 amplitude compared to the P50 amplitude to S1. The reduction in amplitude is expressed as a ratio of the amplitudes to the two stimuli (S2/S1). A lower ratio reflects greater inhibition or greater gating of irrelevant sensory input (Boutros & Belger, 1999). Boutros et al. (1991) found that a typical P50 suppression ratio in individuals without a history of schizophrenia is around 50% or less.

In addition to the term suppression ratio, the term conditioning-testing ratio is used. The first stimulus initiates the conditioning response, and the second stimulus initiates the test response. The response to the second stimuli “is the evidence, or ‘test’ of the action of inhibitory or other gating mechanisms activated or ‘conditioned’ by the first stimulus” (Nagamoto et al., 1989, p. 550). The conditioning-testing ratio is calculated by dividing the testing response by the conditioning response, or as previously described S2/S1 (Arnfred et al., 2001).

The attenuation that occurs during sensory gating reduces the amount of sensory information passed on to the higher cortical processing centers. Individuals with schizophrenia generally have higher suppression ratios, meaning a decreased ability to suppress repetitive information (Adler et al., 1990; Arnfred et al., 2001; Waldo &

Freedman, 1986). Arnfred, Chen, Glenthøj, and Hemmingsen, (2003) found normal P50 gating in unmedicated schizophrenia outpatients, but recognized that this was abnormal and could not find a statistically based explanation for the inconsistency with previous research. The abnormal higher suppression ratios in individuals with schizophrenia are thought to be the neural basis for some of their symptoms (particularly disorganization and delusions), because of the presentation of repetitive sensory information to their higher cortical processing centers (Arnfred et al., 2001).

Interpair Intervals

Dolu, Suer, and Ozesmi (2001) found there were dramatic distinctions in P50 suppression ratios when using different interpair intervals. An interpair interval (also known as an intrastimulus interval) is the time between the first and second presentation of a matched stimulus used in the conditioning-testing P50 paradigms. An interstimulus interval signifies the time between trials.

Dolu et al. (2001) used a 10 s interstimulus interval for each stimulus pair and interpair intervals of 250, 500, 750, and 1000 ms. Dolu et al. showed that the P50 suppression ratios for the 250 and 500 ms intervals were 3.07% and 37.20%, respectively, indicating intact sensory gating mechanisms. The averages of the P50 suppression ratios for the 750 and 1000 ms intervals were 114.35% and 92.92%, respectively, indicating little or no suppression. Most individuals without a history of schizophrenia exhibit a suppression ratio of 50% or less (Boutros et al., 1991). Dolu et al. concluded that the mechanism(s) responsible for sensory gating is activated mostly during the 500 ms after stimulus presentation. In addition an interpair interval of more

than 500 ms used in a sensory gating study may result in abnormally high suppression ratios (indicating less suppression) in neuropsychiatrically normal individuals (Dolu et al., 2001).

Factors Affecting the P50 Response

It has been debated whether gender has an effect on the P50 response. Boutros et al. (1991) have argued that gender and the phase of menstrual cycle have not been found to change the amplitude of the P50 or its degree of attenuation. A study by Hetrick et al. (1996) discovered contrasting evidence about gender affects on the P50 response. Hetrick et al. found that the amplitudes to S1 were not significantly different between men and women at P50, N100, or P180; however, they found that women had significantly higher amplitudes to S2 at P50 and N100. The conditioning-testing ratios were higher in women for the P50 and N100 responses when compared to men, which indicates “less gating (or neuronal inhibition to repeated stimulation)” (Hetrick et al., 1996, p. 55). Hetrick et al. suggest that while “the gating differences [between men and women] are not related to neurophysiological differences in the generator substrates for these components”, it is acknowledged that “the inhibitory mechanisms activated by the conditioning click are different in men and women” (p. 55).

The P50 may be affected by neuropsychiatric disease states (Dolu et al., 2001; Saletu, Itil, & Saletu, 1971), alcohol (Freedman, Waldo, Waldo, Wilson, 1987), slow-wave sleep (Erwin & Buchwald, 1986), extreme degrees of hostility and anger (Waldo & Freedman, 1986), and age (Freedman, Adler et al., 1987). Saletu et al. collected AEP measurements from three groups of participants including schizophrenics with and without a thought process disorder (TPD) and a neuropsychiatrically normal

control group. The participants with TPD showed the greatest variability in their evoked response patterns, and the neuropsychiatrically normal volunteers had the highest stability (Saletu et al., 1971). It has been shown that in addition to individuals with schizophrenia having higher suppression ratios, or decreased sensory gating, their first-degree relatives also had higher suppression ratios (Siegel et al., 1984). Acute alcohol consumption has been shown to decrease the degree of attenuation in response to the second administered stimulus (Freedman, Waldo et al., 1987). The P50 amplitude has been shown to dramatically decrease during slow-wave sleep (Erwin & Buchwald, 1986). Waldo and Freedman (1986) showed that P50 attenuation was lacking in some neuropsychiatrically normal participants when they underwent extreme anger-hostility and tension-anxiety.

Influence of Age on the P50 Response

Surwillo (1977) studied developmental changes in the speed of information processing, but did not specifically target sensory gating changes with age. Surwillo found that younger children had longer latencies than older children did, because they needed more time to process the same amount of information. The results from Surwillo's study about developmental changes in information processing led to an interest in possible developmental changes found in sensory gating. Due to the lack of information about sensory gating at the time of their study, Freedman, Adler et al. (1987) decided to trace the development of the auditory gating mechanism from early childhood through adulthood.

Freedman, Adler et al. (1987) examined the effects of age on the P50 amplitude, latency, and conditioning-testing ratio. Participants included normally hearing males and

females without a medical history of neurologic or psychiatric abnormalities, ranging from 18 months to 55 years of age. Freedman, Adler et al. used a 0.04 ms duration click as their stimulus, and used monopolar recordings made from a single electrode position. Freedman, Adler et al. found longer P50 latencies in young children which decreased around 9-11 years of age. Freedman, Adler et al. found higher P50 amplitudes and higher suppression ratios in children (lesser suppression), which gradually decreased to about 20 years of age, where they leveled off. Not only was there a decrease in the mean suppression ratio (showing greater suppression) as age increased, but also a decrease in variance of the suppression ratio with maturation. Freedman, Adler et al. found that children 1-8 years of age can show suppression ratios which range from 0-100% and that this range decreased with age.

Freedman, Adler et al. (1987) observed that the P50 amplitude, latency, and suppression ratios all decreased with age, and that there was less variability in waveforms with maturation, but their study was completed using an age range from 18 months to 55 years of age. The current study used participants 3-72 years of age to further define maturational changes in sensory gating, focusing on the amplitude, latency, and the suppression ratio of the P50 response.

Method

Participants

Forty-two native English speakers participated in this study, including 21 males and 21 females, ranging from 3-72 years of age. Each individual was placed into one of seven age groups. The age groups consisted of individuals 3-5, 6-9, 10-16, 17-24, 25-39,

40-59, and 60-80 years of age. Each age group included six participants, with equal numbers of males and females in each of the seven age groups.

All participants were otologically normal, which included an absence of signs or symptoms of ear disease and an absence of excessive cerumen. All participants demonstrated normal hearing, bilaterally; either (a) participants had pure-tone thresholds of ≤ 20 dB HL, for the following frequencies: 250, 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz; or (b) participants over 25 years of age had hearing thresholds no higher than the levels designated for their specific age and gender as found in Willott (1991, p. 171). Willott showed age-related changes in auditory sensitivity as a function of age, in males and females separately, using data from eight studies between 1938 and 1963, including rural, random, professional, and industrial populations.

Additionally, all participants had a bilateral hearing threshold difference no greater than 5 dB at 1000 Hz, the stimulus frequency used for this study. Participants had normal middle ear function consisting of tympanometric peak pressure between 80 and 159 daPa for children, and between 51 and 114 daPa for adults (Margolis & Hunter, 2000). Static acoustic admittance between .25 and 1.05 cm³ for children, and between .30 and 1.70 cm³ for adults, was required (Margolis & Hunter, 2000). Any individual reporting a history of autism, attention deficit disorder, psychosis, blindness, severe language disorder, aphasia, gross neurological disease, or other central nervous system deficits was excluded from the study.

Each participant signed a consent form approved by the Brigham Young University Institutional Review Board for Human Subjects prior to inclusion in this study. Participants under 18 years of age also had a parent or guardian sign a consent

form giving permission for their child to be included in this study (See Appendixes A, B, C, and D).

Instrumentation

A Grason Stadler[®] 1761 audiometer was used for the hearing screenings and a Grason Stadler[®] 1733 Version 2 Middle Ear Analyzer was used to obtain tympanometric measures. The Grason Stadler[®] 1761 audiometer was calibrated according to ANSI S3.6-2004 Specifications for Audiometers (American National Standards Institute, 2004) and the Grason Stadler[®] 1733 Version 2 Middle Ear Analyzer was calibrated in accordance with manufacture specifications, at the outset of the study and at the completion of the study.

An electrode cap from NeuroScan Laboratories with 32 silver-silver chloride electrodes was used to record the AEPs. The electrodes were positioned according to the 10-20 International System (Jasper, 1958) and referenced to both earlobes. The ground electrode was placed at Fz. Eye movement was monitored by placing electrodes on the outer cantha, and above the supra-orbital foramen, on both eyes. Electrode impedance was measured at or below 10 k Ω .

Stimuli

Matched pairs of 1000 Hz tone pips (S1 and S2; see Figure 1) were delivered through Etymotic ER-3A insert phones with an intensity of 80 dB HL. Each tone pip had a 3 ms rise and fall time and a 5 ms plateau time. The tone pips had an intrastimulus interval (i.e., between S1 and S2) of 500 ms, and an interstimulus interval (i.e., between trials) of 10 s. The sound pressure of the acoustic stimulus was calibrated using a Larson-Davis[®] 800B sound level meter according to the procedure outlined by Burkard

(1984). The audiometer was calibrated at the outset of the study and at the completion of the study. No significant variations were noted.

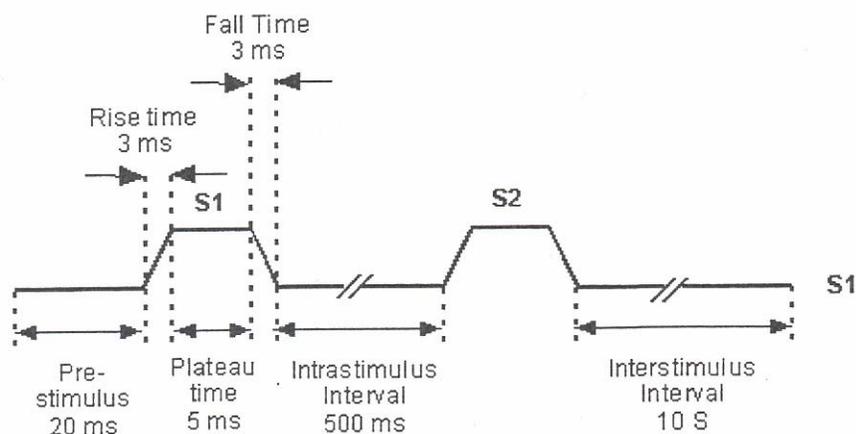


Figure 1.

Schematic of the stimulus presentations (S1 and S2) and sequencing.

The data were collected in an acoustically treated sound suite. Ambient noise levels were measured using 1/3 octave band filters with a Larson-Davis[®] 800B sound level meter and were in compliance with the ears covered condition of ANSI S3.1-1999 Standard for Maximum Permissible Ambient Noise Levels for Audiometric Test Rooms (American National Standards Institute, 1999).

Procedure

Each participant was fitted with an electrode cap. Each electrode was filled with ECI Electro-gel to reduce impedance to 10 k Ω or less. Participants were then seated comfortably in a sound treated room. Children under 6 years of age were able either to sit on a parent's lap, or to cuddle with a blanket or toy (Freedman, Adler et al., 1987). Etymotic ER-3A insert phones were situated in each ear canal. Participants were

instructed to relax, keep their eyes open and fixed on a distant point, or to keep their eyes closed during the auditory stimulus presentations. Participants were also asked to respond to each pair of stimuli by pressing a hand held button. Pressing the hand held button was primarily used to help keep the participants alert during the recording session.

Two trials of fifty 1000 Hz tone pairs were presented and samples were taken using a Neuroscan signal averager from Neuroscan Laboratories. A 20 ms pre-stimulus and 100 ms post-stimulus sample was obtained in order to record the P50 response. The evoked responses were amplified 10,000 times and band-passed between 1 and 200 Hz (3 dB down, 6 dB/octave) with a rejection rate of 6 dB/octave. Averaging was performed using Scan 4.2 averaging software on a Neuroscan System.

Data Analysis

Peak-to-peak amplitude and latency of the P50 waveforms were measured on individual-average waveforms, and grand-average waveforms for each age group. Two trials were recorded for each subject. The best trial was defined as the trial in which the least number of artifact rejects occurred during the averaging process. All components were analyzed at the Cz electrode site. The P50 response to S1 was identified as the largest peak occurring between 40 and 80 ms post stimulus. If two peaks of the same amplitude were present, the later occurring wave was selected. The P50 response to S2 was defined as the largest positive peak occurring within ± 5 ms of the P50 response to S1 (Adler et al., 1985; Freedman, Adler et al., 1987).

The peak-to-peak amplitude of the P50 response was measured from positive peak to the preceding negative trough. The latency was measured from stimulus onset to the P50 response. The suppression ratio of the P50 response was calculated as follows:

the amplitude of the P50 in response to S2 divided by the amplitude of the P50 in response to S1, and is given as S2/S1 (Adler et al., 1990).

Results

The current study used 42 participants ranging from 3-72 years of age to observe maturational changes in sensory gating, particularly the amplitude, latency, and suppression ratio of the P50.

A repeated measures ANOVA was completed to determine significant differences between trials using a significance level of $p \leq .05$. Because the ANOVA failed to show significant differences between Trial 1 and Trial 2, $F(3, 2) = 1.777, p = .154$, the best trial was chosen for each subject and used for statistical comparison and representation throughout this manuscript.

Table 1 shows descriptive statistics for the amplitudes, latencies, and suppression ratios for each of the seven age groups, using the best trial for each subject. The S2 amplitudes were smaller than the S1 amplitudes, $F(1, 2) = 26.876, p \leq .001$. Similarly, the S2 latencies were shorter than the S1 latencies, $F(1, 2) = 6.244, p = .015$, with the exception of the latency for individuals 3-5 years of age, which showed the S1 latency was shorter than the S2 latency. Individuals 17-24 years of age had the lowest mean suppression ratio (showed greatest suppression). There is also a general trend that as age increases, amplitude and latency (between the minimum and maximum) decrease. Furthermore, post-hoc testing of the amplitudes of S1 and S2 (see Table 1) showed that there were significant differences between the amplitudes of S1 and S2 for age groups 10-16, 17-25, 25-39, and 40-59.

Table 1

Descriptive Statistics for Amplitude (μ V), Suppression Ratio, and Latency (ms) of the P50

Response

Age range (years)	<i>N</i>	<i>M</i>	<i>SD</i>	Minimum	Maximum
3-5					
S1 Amplitude	5	3.26	1.55	1.62	4.83
S2 Amplitude	6	1.62	1.18	0.69	3.71
Suppression Ratio	5	0.41	0.19	0.15	0.63
S1 Latency	5	53.20	4.92	49.00	61.00
S2 Latency	6	45.67	5.57	50.00	63.00
6-9					
S1 Amplitude	6	4.89	6.31	1.46	17.67
S2 Amplitude	6	1.16	0.87	0.25	2.49
Suppression Ratio	6	0.44	0.55	0.11	1.54
S1 Latency	6	61.67	8.09	49.00	69.00
S2 Latency	6	53.83	7.00	44.00	64.00
10-16 ^a					
S1 Amplitude	6	6.03	3.20	1.63	10.75
S2 Amplitude	6	2.34	2.02	0.49	4.99
Suppression Ratio	6	0.41	0.29	0.09	0.85
S1 Latency	6	60.50	6.22	51.00	69.00
S2 Latency	6	59.17	6.18	54.00	67.00
17-24 ^a					
S1 Amplitude	6	3.28	2.40	1.07	7.91
S2 Amplitude	5	0.75	0.47	0.28	1.37
Suppression Ratio	5	0.26	0.22	0.08	0.56
S1 Latency	6	57.83	3.87	52.00	63.00
S2 Latency	5	54.00	3.94	48.00	58.00
25-39 ^a					
S1 Amplitude	6	4.36	2.52	2.29	9.07
S2 Amplitude	6	1.29	1.03	0.09	2.89
Suppression Ratio	6	0.32	0.26	0.02	0.64
S1 Latency	6	60.83	3.87	55.00	65.00
S2 Latency	6	55.83	4.36	52.00	64.00

Age range (years)	<i>N</i>	<i>M</i>	<i>SD</i>	Minimum	Maximum
40-59 ^a					
S1 Amplitude	6	3.09	2.37	0.48	6.83
S2 Amplitude	6	1.29	1.07	0.20	3.16
Suppression Ratio	6	0.52	0.35	0.16	1.11
S1 Latency	6	57.67	4.76	50.00	63.00
S2 Latency	6	56.17	4.49	49.00	59.00
60-80					
S1 Amplitude	6	4.36	2.90	0.58	8.69
S2 Amplitude	6	1.13	0.70	0.19	1.86
Suppression Ratio	6	0.37	0.28	0.50	0.79
S1 Latency	6	54.33	2.50	51.00	58.00
S2 Latency	6	52.17	2.23	49.00	55.00

Note. Descriptive statistics for each age group, using the best trial for each participant. S1 refers to stimulus one. S2 refers to stimulus two. Suppression ratio is defined as the S2 amplitude divided by the S1 amplitude. Suppression ratios were derived from individual ratios from each subject and not from the mean S1 and S2 values.

^a Significant S1-S2 (ANOVA) amplitude differences were seen for the following age groups: 10-16 years of age, $F(1, 2) = 5.686, p \leq .038$; 17-24 years of age, $F(1, 2) = 5.303, p \leq .047$; 25-39 years of age, $F(1, 2) = 7.612, p \leq .020$; and 40-59 years of age, $F(1, 2) = 7.043, p \leq .024$.

Figures 2 and 3 show the trends for the P50 amplitude (in μV) when compared to age (in years). A third order polynomial was used as a means of constructing a best fit regression in figures 2 and 3. Figure 2 shows a decrease in amplitude to about 40 years of age, with a slight upward trend after 55 years of age. Figure 3 shows a similar curve, with a decrease in amplitude to about 45 years of age, and an increase in amplitude after 45 years of age. Figure 4 compares the mean amplitudes for S1 and S2 for each of the seven age groups, and clearly demonstrates that the S2 amplitudes were lower than the S1 amplitudes for each of the seven age groups.

Figures 5 and 6 used first order polynomials to illustrate the trends of the P50 latency (in ms) when compared to age (in years). These figures show a decrease in latency as age increases, with a slightly greater slope for the latency of S1 than for S2. Figure 7 compares the mean latencies for S1 and S2 for each of the seven age groups. This bar graph shows that the S2 latencies were shorter than the S1 latencies for all of the age groups except for individuals 3-5 years of age, which showed the S1 latency was shorter than the S2 latency.

Figure 8 shows the trend using a third order polynomial for the P50 suppression ratio when compared to age (in years). Figure 8 indicates there is a decrease in the suppression ratio (greater suppression) to about 30 years of age, with an increase in the suppression ratio (lesser suppression) after 35 years of age. Figure 9 compares the mean suppression ratios of each of the seven age groups. Figure 9 shows that individuals 17-24 years of age had the lowest mean suppression ratio (greatest suppression), which can be explained by that age group's low mean amplitudes, as seen in Figure 4. Individuals 17-24 years of age had one of the smallest amplitudes for S1 (one standard deviation away

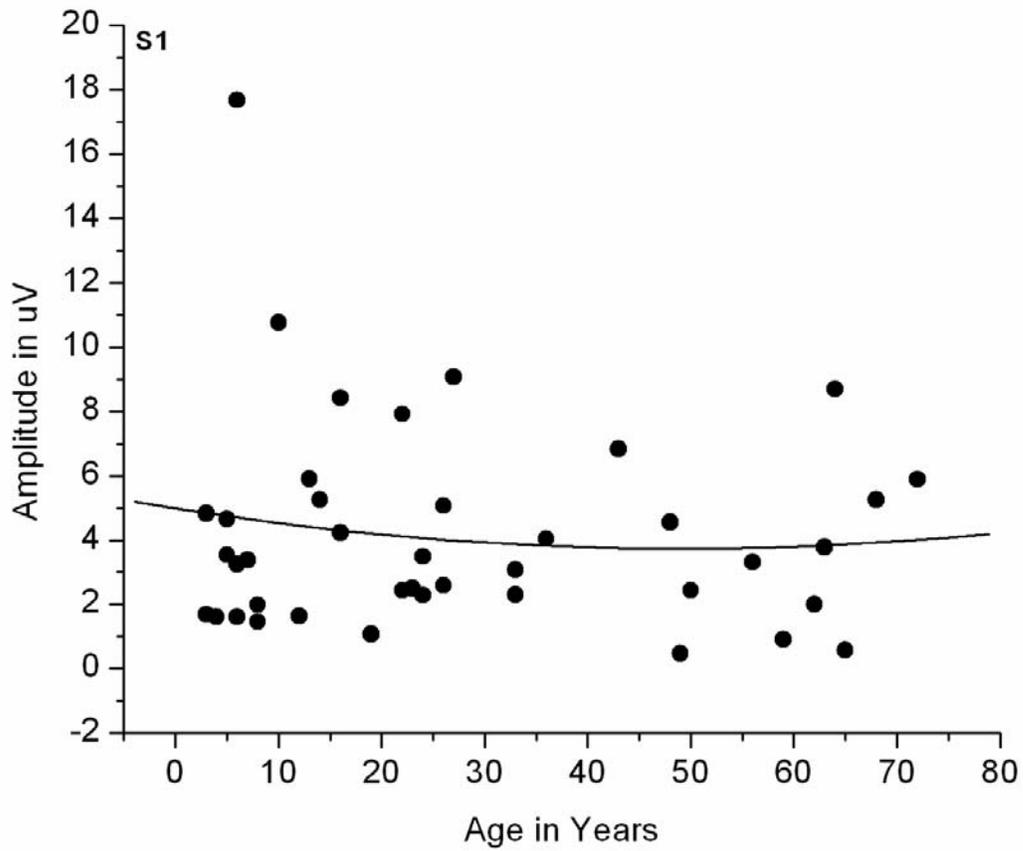


Figure 2.

Mean P50 amplitude in μV , compared to age in years, for S1 (stimulus one). The line illustrates the trend of the P50 amplitude when compared to age using a third order polynomial.

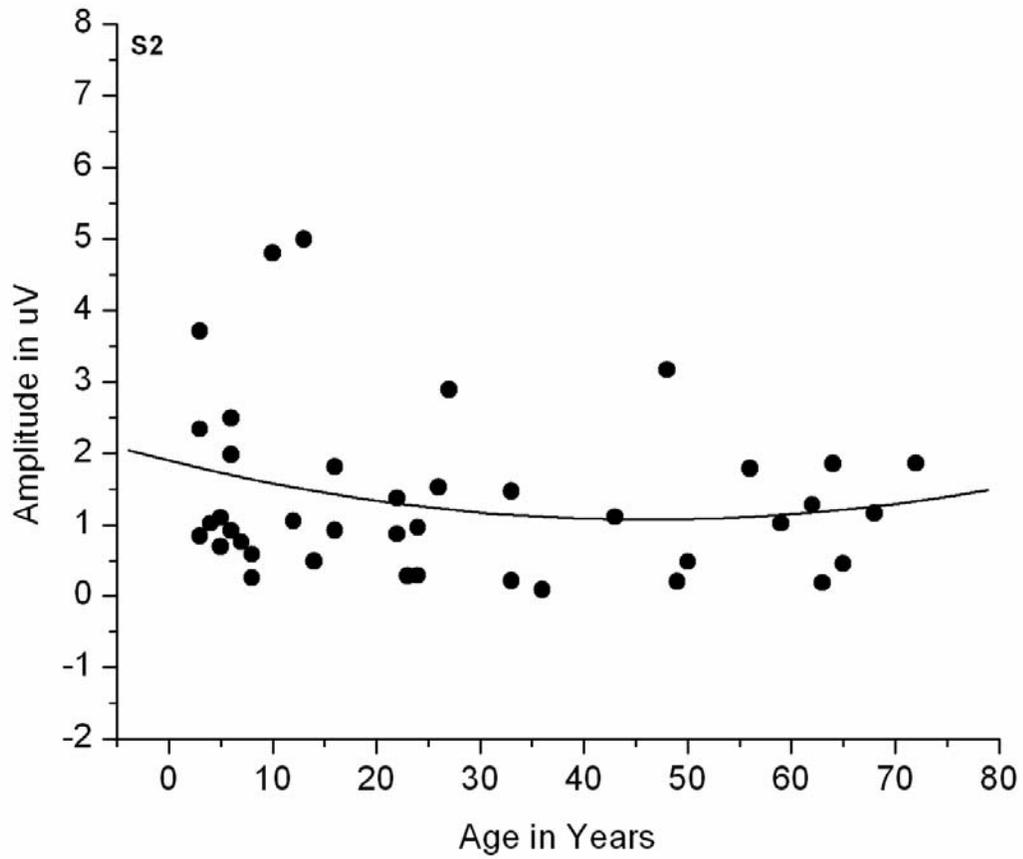


Figure 3.

Mean P50 amplitude in μV , compared to age in years, for S2 (stimulus two). The line illustrates the trend of the P50 amplitude when compared to age using a third order polynomial.

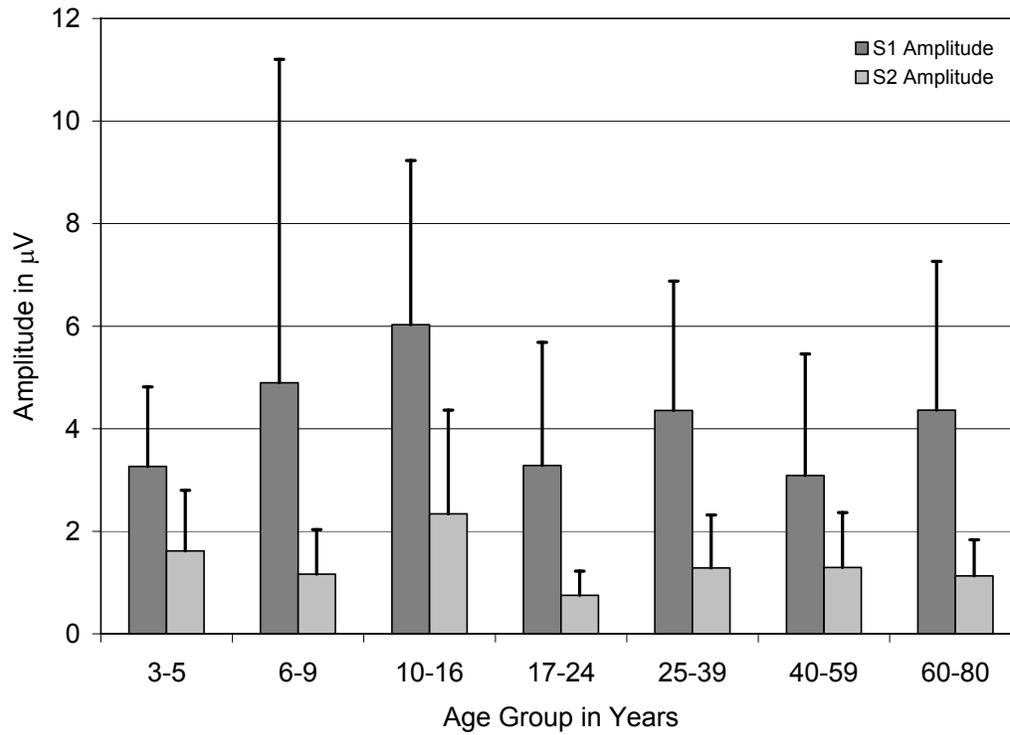


Figure 4.

Mean and SD of P50 amplitude in μV for S1 (stimulus one) and S2 (stimulus two) for each of the seven age groups.

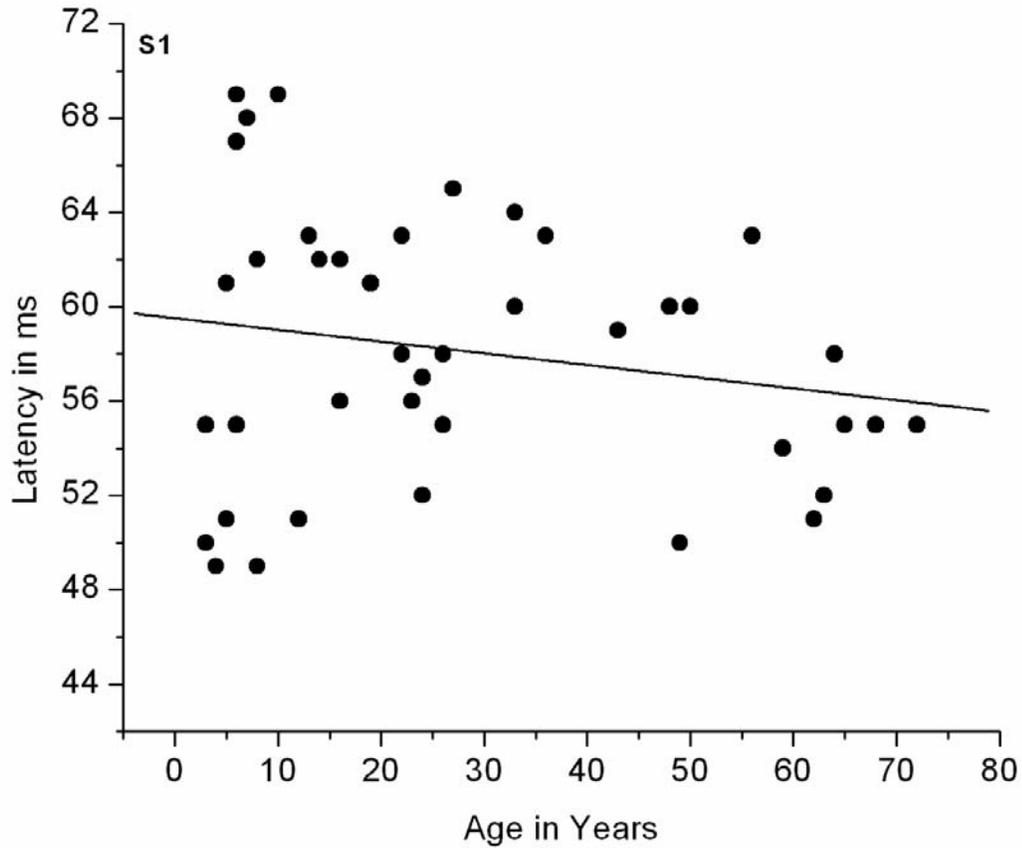
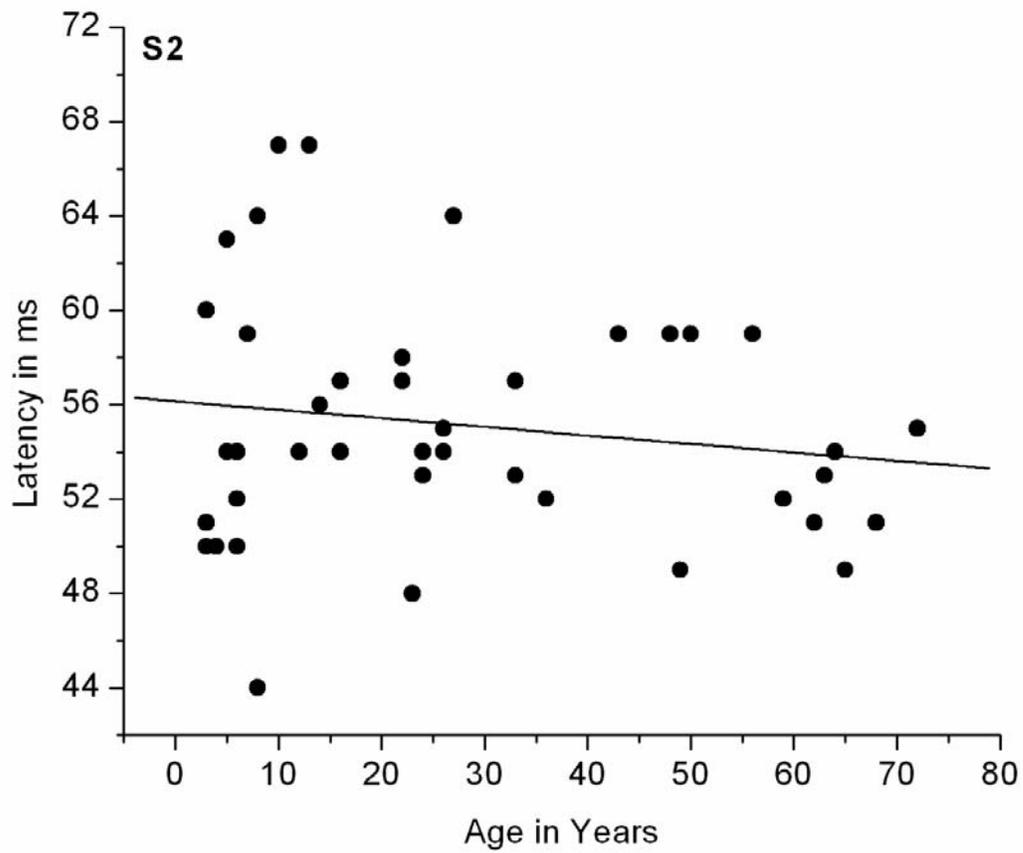


Figure 5.

Mean P50 latency in ms, compared to age in years, for S1 (stimulus one). The line illustrates the trend of the P50 latency when compared to age using a first order polynomial.



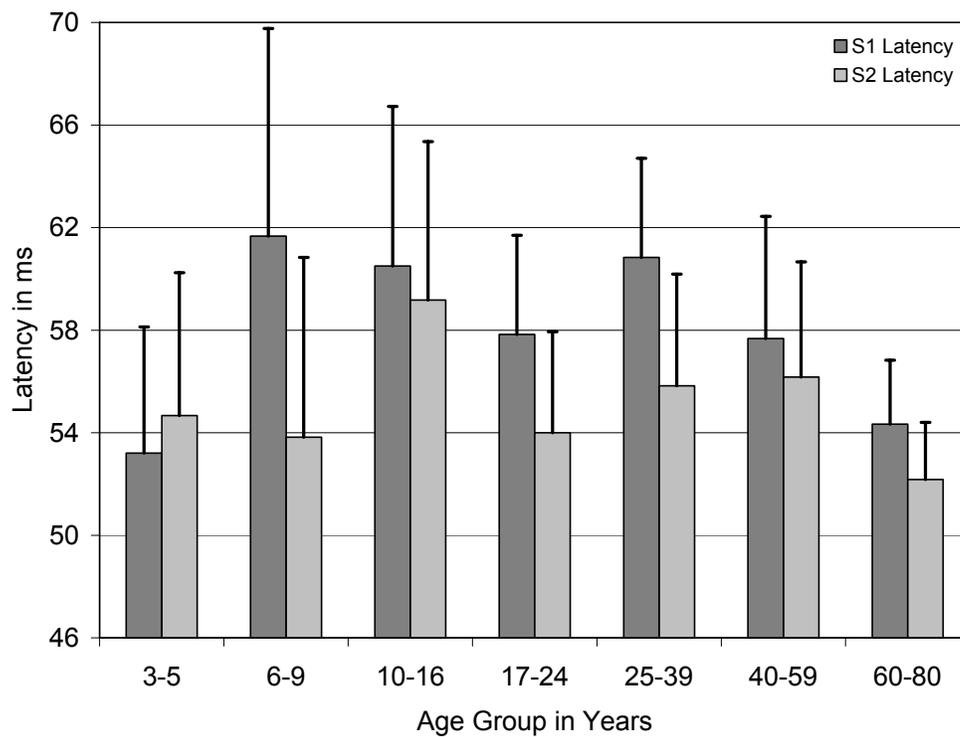


Figure 7.

Mean and SD of P50 latency in ms for S1 (stimulus one) and S2 (stimulus two) for each of the seven age groups.

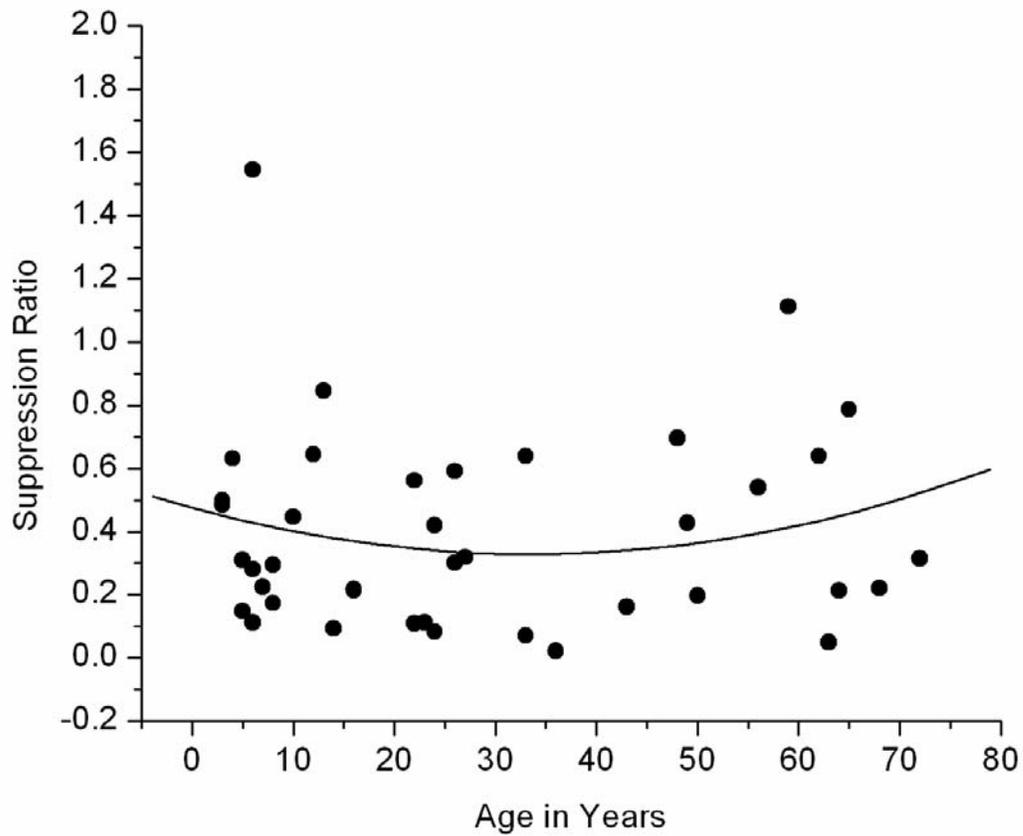


Figure 8.

Mean P50 suppression ratio, compared to age in years. Suppression ratio is defined as the S2 (stimulus two) amplitude divided by the S1 (stimulus one) amplitude. The line illustrates the trend for the P50 suppression ratio when compared to age using a third order polynomial.

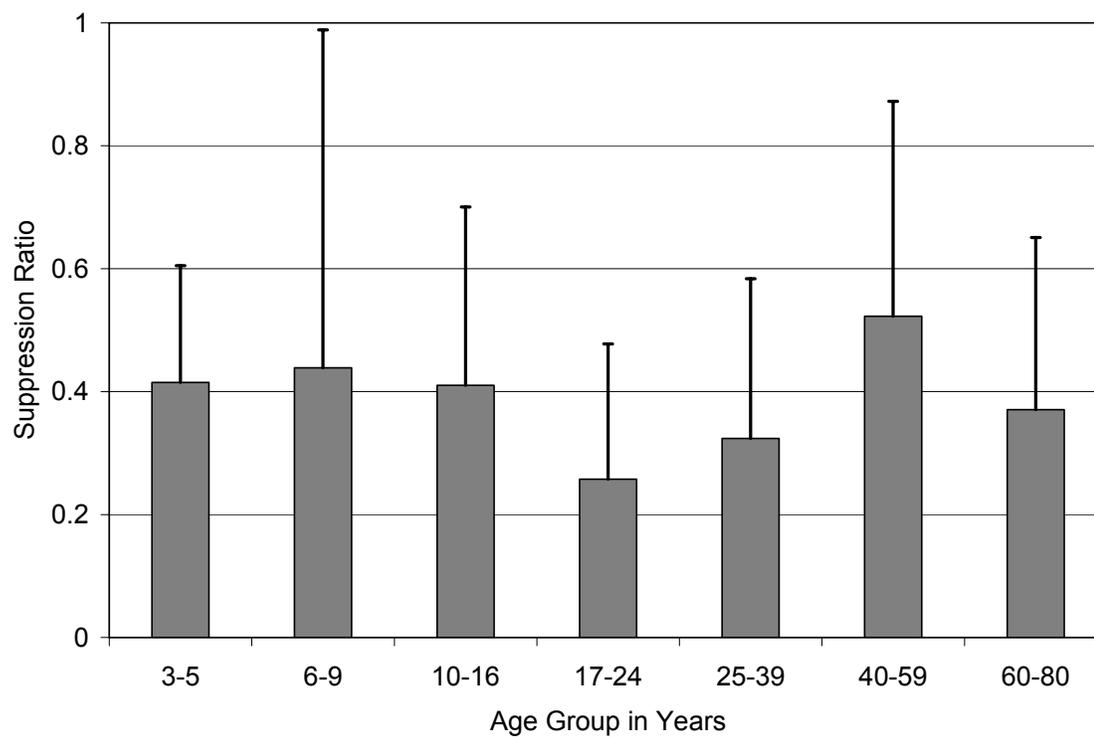


Figure 9.

Mean and SD of P50 suppression ratios for each of the seven age groups. Suppression ratio is defined as the S2 (stimulus two) amplitude divided by the S1 (stimulus one) amplitude.

from the two smallest amplitudes for S1, which were observed in individuals 3-5 years of age and 40-59 years of age, respectively), and individuals 17-24 years of age also had the smallest amplitude for S2. This combination of amplitudes resulted in individuals 17-24 years of age having the lowest suppression ratio, or greatest level of suppression.

Discussion

Amplitude

The current study showed that the S2 amplitudes were lower than the S1 amplitudes for ages 10 through 59 years of age. This is consistent with previous findings from Boutros and Belger (1999) who stated that MLAEPs are used with sensory gating and are known to decrease in amplitude with repetition at short intervals. Freedman, Adler et al. (1987) asserted that the cause of this decrement was the activation of recurrent inhibitory neuronal pathways.

The trends for amplitude in the current study are consistent with prior observations of maturational effects on the P50. The current study showed the amplitudes of P50 for S1 and S2 were higher for young children and decreased in amplitude, with minimum average amplitude observed between 40 and 45 years of age (see Figures 2 and 3). This is similar to the results observed by Freedman, Adler et al. (1987), who found higher S1 amplitudes for the P50 in childhood, with a gradual decrease in amplitude for individuals 20-29 years of age. The current study showed an increase in amplitude for both S1 and S2 for adults through 72 years of age, which was not noted in the study by Freedman, Adler et al., since their highest age was 55 years. This prevented Freedman, Alder et al. from observing the trends in the amplitude of the P50 seen in the current study.

Latency

The trends for latency in the current study were different from those of previous studies of maturational effects on the P50. Both the current study and the study by Freedman, Adler et al. (1987) showed longer latencies in childhood, with either a decrease or steadiness in latencies into adulthood. The current study showed the latencies of P50 for S1 and S2 were longer during childhood and decreased in latency with age, except for individuals 3-5 years of age, who showed shorter latencies (for both S1 and S2) than the other children age groups. As seen on Figure 7, there was a noticeable decrease in latency around 17-24 years of age. In contrast, the study by Freedman, Adler et al. showed longer latencies for S1 for individuals 1-8 years of age, with a stabilization of latencies for individuals 9-11 years of age through individuals 45-55 years of age.

Suppression Ratio

The trends for mean suppression ratio in the current study are consistent with prior observations of maturational effects on the P50, up to 55 years of age. Additionally, the current study showed less suppression for adults through 72 years of age, which was not noted in the study by Freedman, Adler et al. (1987), since their highest age was 55 years. Woods and Clayworth (1986) found higher amplitudes and longer latencies in individuals 60-80 years of age when looking at MLAEPs. They reported that these differences in MLAEP waveforms appeared to be a consequence of age-related central modifications in auditory processing, and were not caused by changes in peripheral auditory structures. While Woods and Clayworth did not study sensory gating or use

suppression ratios, they did find changes in amplitude with age, which would affect suppression ratios in a similar manner.

Figure 9 shows suppression ratios as compared to age groups for the current study. The mean P50 suppression ratio was higher for individuals 3-5 years of age (showing decreased suppression), then suppression ratios decreased to the minimum for individuals 17-24 years of age (showing increased suppression), before the suppression ratios increased again for individuals 25-39, 40-59, and 60-72 years of age (showing decreased suppression). This is similar to the results observed by Freedman, Adler et al. (1987), who found a decrease in the suppression ratio from childhood to individuals 45-55 years of age, showing greater suppression with age. While Freedman, Adler et al. found increased suppression in adults, the association with age was not linear before 20 years of age.

Both Freedman, Adler et al. (1987) and the current study showed mean suppression ratios no higher than 60% which is consistent with Boutros et al. (1991), who found that most individuals without schizophrenia exhibit a suppression ratio of 50% or less. Figure 9 shows the highest mean suppression ratio (or least amount of suppression) from the current study was 52% for individuals 40-59 years of age followed closely by individuals 3-5, 6-9, and 10-16 years of age. The highest mean suppression ratio (or least amount of suppression) from Freedman, Adler et al. was around 60% for males 1-8 and 9-11 years of age. Figure 9 shows that the lowest mean suppression ratio (or greatest amount of suppression) from the current study was for individuals 17-24 years of age, which correlates with Freedman, Adler et al., whose lowest mean suppression ratio was for males and females 20-29 years of age.

Maturation of the Brain

Woods and Clayworth (1986) discussed the nature of the amplitude and latency changes seen in MLAEPs in their study. They found that elderly participants, when compared to younger adult participants with similar hearing levels, had higher Na-Pa and Pa-Nb amplitudes and longer Pa latencies. Woods and Clayworth believe that the Pa amplitude trends seen in their study were caused by changes in the central nervous system, such as structural or neurochemical changes, and were not caused by changes in the peripheral auditory structures. This observation may explain the results from the current study, which showed a gradual increase in P50 amplitudes after approximately 40-45 years of age. It is foreseeable that the changes in amplitude in older participants are due to modifications in the nervous system's structure and neurochemical makeup.

Suzuki and Hirabayashi (1987) studied age-related morphological changes in MLAEPs. They found that the Pa component of the MLAEP approached the adult waveform pattern earlier than the later part of the response, which was not complete until after 12-14 years of age. Suzuki and Hirabayashi reported, "maturation of nonspecific thalamocortical pathways and sensory cortex was not complete until the onset of puberty" (p. 318). Suzuki and Hirabayashi observed that children 8-11 years of age had initial portions of the MLAEP waveforms similar to the adult pattern, but that the complete adult pattern was not reached even by 12-14 years of age. Consistent with Suzuki and Hirabayashi's observations, the current study showed adult patterns for amplitude, latency, and the suppression ratio in participants by around 17 years of age. Suzuki and Hirabayashi also propose that evoked responses with longer latencies may mature slower since they are likely generated from higher levels of the brain.

The finding of more intersubject as well as intrasubject variation in waveforms (including amplitude, latency, and suppression ratio) for younger children than there were for adults in the current study is consistent with results from Suzuki and Hirabayashi (1987) and Freedman, Adler et al. (1987). Suzuki and Hirabayashi suggested that the higher variability in waveforms in younger participants could be due to changes in the brain over the course of natural development. Freedman, Adler et al. reported that the changes in both mean and variance of the suppression ratio point to multiple neurobiologic mechanisms being responsible for the development of adult patterns of the suppression ratio.

Future Research

Upon completion of this study and reviews of previous research, it appears that there are intersubject and intrasubject variations of the P50 waveforms for amplitude and latency and in the P50 suppression ratio, which change as a function of age. Further studies are necessary to systematically categorize the changes seen in the P50 sensory gating waveforms.

As has been shown by Wible, Nicol, and Kraus (2005), several speech and language disorders are known to have abnormalities in AEPs. It is unknown whether these disorders also show abnormalities in P50 sensory gating. A more complete understanding of how these communication disorders relate to sensory gating awaits further investigation.

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Appendix A

Consent to Act as a Human Research Subject
 Scalp Distribution of Sensory Gating
 David L. McPherson, Ph.D.
 Department of Audiology and Speech Language Pathology
 Brigham Young University
 (801) 422-6458

Name of Participant: _____ Date of Birth: _____

Purpose of Study:

This investigation is designed to study how the brain reacts when presented with pairs of identical sounds. Participation in this study will help scientists better understand how the brain processes sounds in children and adults.

Procedures:

I have been asked to participate in a research study being conducted by Dr. David L. McPherson and/or such assistants as may be selected by him.

The study will be conducted in room 109 and 111 of the John Taylor Building on the Brigham Young University campus. I will be asked questions but do not have to answer any questions that I do not want to answer.

Participation in this study, including orientation and testing, requires two sessions, equaling about 2 hours, total. These two sessions will be scheduled on separate days, but it is possible for the two sessions to be combined into one visit. The first session will be for a hearing screening and a question and answer session, which should last approximately 20 minutes. The second session will be for the actual data collection, which should last about 1 ½ hours. I have been advised that these times are an estimate, and it may take more or less time, depending on how easy it is to set up the equipment. I may ask for a break at any time during testing.

I will be given a standard hearing test screening. My ears will be looked into with a scope, my eardrums will be tested to see how they move, and my hearing will be tested.

Surface electrodes (metal discs about the size of a dime) will be used to record electrical activity of the brain. These discs will be applied to the surface of the skin with a gel and are easily removed with water. Blunt needles will be used as a part of this study to help apply the electrode gel, but they will *never* be used to puncture the skin.

Electrical activity of my brain will be recorded, but no electrical impulses/shocks will be applied to the brain. This is similar to what is known as an “EEG” or brain wave test. These procedures show actual measurements of normal, continuous, electrical activity in the brain.

Some sounds will be presented through earphones. I will be asked to push a button when I have heard a sound through the earphones. These clinical procedures are routine, similar to those used to test hearing, although some techniques of analysis are experimental.

Participant Initial _____, Page 1 of 2

Risks:

There may be some local skin irritation resulting from the electrode discs. This will be treated in the usual manner by removing the discs and exposing the area to air, which results in alleviation of the irritation. Another possible, but unlikely, discomfort would be if the scalp received an abrasion when the blunt needle is used to place the electrode gel. The electrodes would be removed immediately, and any gel on the injured site would be removed. If this were to occur I would be given the option to discontinue my participation in the study. There are no other known risks with this procedure.

Benefits:

Possible benefits from participating in this study will be the assessment of my hearing. I will be notified of any irregularities in my ears (structures) and/or hearing abilities found during the routine hearing tests. If irregularities are discovered, I may be advised to have a professional examine my ears/hearing, or I may be advised of possible treatments (if any). These procedures will benefit me by providing (possible) early treatment. I also understand there may be no direct benefit to me. However, the information obtained will help people further understand how the brain processes auditory information and about differences in these processes between children and adults.

Confidentiality:

Participation in this study is voluntary and I have a right to refuse to participate or withdraw at any time, without penalty. All information obtained from testing is strictly confidential and is protected under the laws governing privacy. No information specifically pertaining to me, other than reporting of test results without identifying information may be released without my signature. All identifying references will be removed and replaced by control numbers which will identify any disclosed or published data. Data collected in this study will be stored in a secured area accessible only to personnel associated with the study. I give permission to be videotaped, tape recorded, photographed, or other media impressions made for educational and research purposes.

Other Considerations:

There are no charges incurred by me for participation in this study. There is no treatment or intervention involved in this study although I may be counseled to seek such treatment or intervention. I understand that for any reason I may withdraw from the study at any time without penalty.

The procedures listed above have been explained to me by: _____
in a satisfactory manner and my questions relating to such risks and procedures have been
answered. If I have any questions about the research I may ask any of the investigators or
contact Dr. David L. McPherson, Audiology and Speech-Language Pathology, 129
TLRB, Provo, Utah 84602-8633; email: david_mcperson@byu.edu; phone: 801-422-
6458. If I have any questions as to my rights as a participant in this research project I may
contact Renea Beckstrand, Chair of Institutional Review Board, email:
renea_beckstrand@byu.edu; phone: 801-422-3873.

I consent to participate in the above explained study.

Signature of Participant

Date

Appendix B

Parental Informed Consent for Child to Act as a Human Research Participant
 Scalp Distribution of Sensory Gating
 David L. McPherson, Ph.D.
 Department of Audiology and Speech Language Pathology
 Brigham Young University
 (801) 422-6458

Name of Participant: _____ Date of Birth: _____
 Name of Parent or Guardian: _____

Purpose of Study:

This investigation is designed to study how the brain reacts when presented with pairs of auditory stimuli. Participation in this study will help scientists better understand how the brain processes auditory information in children and adults.

Procedures:

My child has been asked to participate in a research study being conducted by Dr. David L. McPherson and/or such assistants as may be selected by him. I may also be asked questions about my child, for research purposes, during this study.

The study will be conducted in rooms 109 and 111 of the John Taylor Building on the Brigham Young University campus. My child and/or I will be asked questions but do not have to answer any questions that we do not want to answer.

Participation in this study, including orientation and testing, requires two sessions, equaling about 2 hours, total. These two sessions will be scheduled on separate days, but it is possible for the two sessions to be combined into one visit. The first session will be for a hearing screening and a question and answer session, which should last approximately 20 minutes. The second session will be for the actual data collection, which should last about 1 ½ hours. I have been advised that these times are an estimate, and it may take more or less time, depending on how compliant my child is and how easy it is to set up the equipment. My child may ask for a break at any time during testing.

My child will be given a hearing screening. My child's ears will be examined with a lighted scope, and then soft probes, attached to a computer, will be inserted into my child's ears. A standard hearing test will then be administered which consists of my child listening to sounds of different volumes and pitches through a set of headphones. The researcher will give my child a button to signal that they have heard a sound through the headphones. If needed, my child may sit on a parent's lap during the hearing screening. Depending on my child's age and understanding of the task, the above hearing test procedures can be modified.

Surface electrodes (metal discs about the size of a dime) will be used to record electrical activity of my child's brain. These discs will be applied to the surface of the skin with a gel and are easily removed with water. Blunt needles will be used as a part of this study to help apply the electrode gel. They will *never* be used to puncture the skin.

Electrical activity of my child's brain will be recorded, but no electrical impulses/shocks will be applied to my child's brain.

Parent Initial _____, Page 1 of 3

This is similar to what is known as an “EEG” or brain wave test. These measurements are actual measurements of normal, continuous electrical activity in the brain. While my child is wearing the electrode cap, he/she will hear some sounds presented through earphones. He/she will be asked to push a button, similar to the hearing test, when he/she hears a sound through the earphones.

The procedures used to record the electrophysiological responses of the brain are standardized and have been used without incident in many previous investigations. While the recording procedures are standardized, some of the analysis techniques may be experimental.

Risks:

There are very few potential risks from this procedure, and these risks are minimal. There may be some local skin irritation resulting from the electrode discs. This will be treated in the usual manner by removing the discs and exposing the area to air, which results in alleviation of the irritation. While allergic reactions to the gel or electrodes are extremely rare (identified by a rash), they would be treated in the same manner as noted above, and the testing procedures would be discontinued. Another possible, but unlikely, discomfort would be if the scalp received an abrasion when the blunt needle is used to place the electrode gel. The electrodes would be removed immediately, and any gel on the injured site would be removed. If this were to occur my child and/or I would be given the option to discontinue their participation in the study. There are no other known risks with this procedure.

Benefits:

Possible benefits from participating in this study will be the assessment of my child’s hearing. I will be notified of any irregularities in my child’s ears (structures) and/or hearing abilities found during the routine hearing tests. If irregularities are discovered, I may be advised to have a professional examine my child’s ears/hearing, or I may be advised of possible treatments (if any). These procedures will benefit my child by providing (possible) early treatment. I also understand there may be no direct benefit to my child. However, the information obtained will help people further understand how the brain processes auditory information and about differences in these processes between children and adults.

Confidentiality:

Participation in this study is voluntary and my child or I has/have a right to refuse to participate or withdraw at any time, without penalty. All information obtained from testing is strictly confidential and is protected under the laws governing privacy. No information specifically pertaining to me or my child, other than reporting of test results without identifying information may be released without my signature. All identifying references will be removed and replaced by control numbers which will identify any disclosed or published data. Data collected in this study will be stored in a secured area accessible only to personnel associated with the study. I give permission for my child to be videotaped, tape recorded, photographed, or other media impressions made for educational and research purposes.

Other Considerations:

There are no charges incurred by me for my child's participation in this study. There is no treatment or intervention involved in this study although I may be counseled to seek such treatment or intervention.

The procedures listed above have been explained to me by: _____ in a satisfactory manner and my questions relating to such risks and procedures have been answered. If I have any questions about the research I may ask any of the investigators or contact Dr. David L. McPherson, Audiology and Speech-Language Pathology, 129 TLRB, Provo, Utah 84602-8633; email: david_mcpherson@byu.edu; phone: 801-422-6458. If I have any questions as to my child's rights as a participant in this research project I may contact Renea Beckstrand, Chair of Institutional Review Board, email: renea_beckstrand@byu.edu; phone: 801-422-3873.

I voluntarily consent to participate and allow my child to participate in the above explained study.

Signature of Parent

Date

Appendix C

Child Assent Form

This study is to look at how our ears listen to sounds and how our brain thinks about those sounds. Being a part of this study will help teachers and scientists better understand how the brain thinks about sounds we hear. What we learn from this study will be useful to people who study how we hear and how our brains work. My parents have agreed that I can help with this study.

I will visit BYU for this study. I will be asked questions, but I do not have to answer them if I do not want to. If I get tired, I can ask for a break. I will have my hearing checked. Also I will wear a hat that has connections attached to a computer. While I am wearing the hat I will hear some sounds through headphones. I will press a button to show that I have heard the sound. If I get tired, I can ask for a break.

I understand that I do not have to do any part of this study. If I change my mind and do not want to do any more of the study, I can quit at any time.

I would like to be a part of this study.

Signature of Participant

Date

Appendix D

Youth Assent Form

This study is to look at how the brain processes sounds that we hear. Being a part of this study will help researchers better understand how the brain reacts to sounds. What we learn will be useful to people who study how we hear and how the brain works. My parents have agreed that I can help with this research.

I will visit BYU for this study. I will be asked questions, but I do not have to answer a question if I do not want to. If I get tired, I can ask for a break. I will have my hearing checked. Then I will wear an electrode cap (as explained/shown to me), that has connections attached to a computer. While I am wearing the cap, I will hear some sounds through headphones. I will press a button to tell the researcher that I have heard the sound. If I get tired, I can ask for a break.

I understand that I do not have to do any part of this study. If I change my mind, I can quit the study at any time.

I would like to be a part of this study.

Signature of Participant

Date

Appendix E

Participant Checklist

First Session

- Participant voluntarily signs consent form and agrees to be a participant
- Parent (of participants under age 18) voluntarily signs consent form and agrees to allow his/her child to be a participant
- Participant (if under the age of 18) voluntarily signs the child or youth assent form and agrees to be a participant
- Perform otoscopic examination
- Collect tympanometry measures
- Audiogram
- File the forms, and screening information

*Second Session**Pre-test set-up*

- Turn on all three red power switches on the Tucker-Davis equipment
- Wait for SynAmps to show SN1/SN2
- Turn on Neuro Scan computer
- Turn on Audiometer
- Open up Neuro Scan software
 - Open calibration screen on NeuroScan
- On Stim computer, open Stim program
- Biological check on audiometer
- Record participant information in lab book
- Set out supplies
 - Syringe
 - Syringe tip (blunt 16 gauge needle)
 - Alcohol wipe
 - Cotton swabs
 - NuPrep skin prepping gel
 - Electrode gel
 - Surgical tape strips (6) about two inches long
 - Thin wooden dowel
 - Clean cloth towels (2)
 - Electrode cap
 - Facial and ear electrodes
- Put surgical tape strips on facial electrodes and poke holes through the tape with the dowel
- Fill syringe with gel

Once participant arrives

- Give an explanation of the study and instruct patient
- Place cap on the head of the participant
- Fill the cap electrodes with electrode gel

- Take participant into sound-attenuating booth and ask him/her to sit down
- Instruct participant to look straight ahead with eyes open and remain motionless, or to keep eyes closed, during the presentations
- Put insert earphones in participant's ear canals for presentation of stimulus
- Plug the electrode cap into SynAmps 150 gain adapter
- Explain the usage of the response pad that will indicate psychophysical response
- Check electrode cap impedance and adjust electrodes with impedance above 10,000 ohms
- Run sets of stimuli
- Computer records the electrophysiological results emitted by the electrode cap
- Once stimuli are complete, remove electrode cap adapter on SynAmps 150 gain amplifier
- Outside the booth, remove electrode cap and facial electrodes
- Give the participant a wet warm cloth to remove the excess gel from the face

Once participant departs

- Wash free electrodes with soap and water
- Soak electrode cap in soapy water for 30 minutes
- Clean out electrode cap electrodes
- Set electrodes and electrode cap out to dry
- Turn off computers and equipment
- Turn off lights in booth
- Record any additional information in lab notebook as needed
- Record raw data into data analysis spreadsheet