

MULTI-SENSORY PROCESSING IN ADULTS: AN EEG STUDY
OF LATENCY AND AMPLITUDE IN THE N1 AND P2 PEAKS

by

KERRY FAITH WILLIAMSON

B.A., University of Arizona, 2004

B.S., University of Arizona, 2009

A thesis submitted to the
Faculty of the Graduate School of the
University of Colorado in partial fulfillment
of the requirements for the degree of
Masters of Arts
Department of Speech, Language, and Hearing Sciences
2011

This thesis entitled:
Multi-sensory processing in adults: An EEG study of latency and amplitude in the N1 and
P2 peaks
written by Kerry Faith Williamson
has been approved for the Department of Speech, Language, and Hearing Sciences

Phillip Gilley

Dr. Brenda Schick

Ms. Amy Thrasher

Date _____

The final copy of this thesis has been examined by the signatories, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

Williamson, Kerry Faith (M.A., Speech Language Pathology)

Multi-sensory processing in adults: An EEG study of latency and amplitude in the N1 and P2 peaks

Thesis directed by Prof. Phillip Gilley

Research has shown that processing of modality specific stimuli begins early on in cortical processing, affecting the peaks of event related potentials that occur earlier in EEG waveforms. Processing of combined sensory inputs has been shown to affect the latency and amplitude of later occurring peaks, the N2 and P300, suggesting that sensory stimuli are processed in a combined manner in a later stage of cortical processing. Two of the earlier peaks, the N1 and P2, of auditory and visual event related potentials (ERPs) can be used to study the effects of multiple sensory inputs on the human sensory system. Using EEG to detect and record ERPs during a randomized single sensory and combined sensory detection task, this study examined whether or not a multisensory stimulus would affect the latency and amplitude of the auditory and visual N1 and P2 peaks. While outcomes of the study did not yield significant results for effects on the latency of the N1, there were significant effects for the amplitude of the N1 peak and the latency of the P2 peak; with the most significant results seen in an increase in amplitude of the P2 peak for the combined stimulus condition.

Research Question: Are changes in the latency and amplitude of the N1 and P2 auditory and visual ERP peaks seen in multisensory processing, and if so, do these changes imply integration of multiple sensory inputs at earlier cortical stages of sensory processing?

DEDICATION

To Porter, who taught me to search and explore in order to understand his world, and to all my clients and their families, who have taught me the importance and value of good research.

ACKNOWLEDGEMENTS

I would like to thank Dr. Gilley, first, for allowing me the great opportunity to be involved in this research, and second, for teaching me so much along the way. Thank you for allowing me to ask questions and encouraging me to arrive at my own conclusions. Your help and support throughout this process have been most appreciated.

I would also like to thank my committee for all their support and encouragement for the entirety of the thesis process. Thank you for your great advice, your patience with me, and for challenging me to strive for excellence in my research.

Finally, I would be remiss if I did not acknowledge my wonderful husband, Jamie, for all his help and encouragement on this thesis. He was instrumental in helping me compile this project, providing critiques when necessary and solutions when possible. I am forever grateful for you!

CONTENTS

| | |
|---|-----------|
| CHAPTER | |
| I INTRODUCTION | 1 |
| II BACKGROUND | 3 |
| Sensory Processing | 3 |
| Multisensory processing | 4 |
| Sensory processing in disordered populations | 6 |
| Cortical mapping of multisensory processing | 10 |
| Use of electroencephalography in studying multisensory processing | 13 |
| Using EEG to detect and study event related potentials | 13 |
| Using the N1 and P2 to identify multi-sensory processing | 15 |
| Latency and amplitude of the N1 and P2 peaks | 17 |
| Hypothesis | 18 |
| III METHODS | 20 |
| Subjects | 20 |
| Stimulus conditions | 20 |
| Presentation of stimuli | 21 |
| Experimental task | 22 |
| Data collection | 22 |
| Analysis | 23 |

| | |
|--------------------------------|-----------|
| Peak analysis | 23 |
| Statistical analysis | 27 |
| IV RESULTS | 34 |
| N1 latency | 34 |
| P2 latency | 35 |
| N1 amplitude | 37 |
| P2 amplitude | 37 |
| V DISCUSSION | 40 |
| N1 latency | 40 |
| P2 latency | 43 |
| N1 amplitude | 43 |
| P2 amplitude | 44 |
| Future research | 45 |
| VI CONCLUSION | 47 |
| BIBLIOGRAPHY | 50 |

TABLES

Table

| | | |
|---|--------------------------|----|
| 1 | Grand Averages | 33 |
|---|--------------------------|----|

FIGURES

Figure

| | | |
|----|--|----|
| 1 | Scalp map of the 10-20 international system indicating the specific electrodes used for this study. Graphs at each electrode location show Grand Averages for each stimulus condition across the subject population. | 24 |
| 2 | Method shown for N1 peak identification. | 25 |
| 3 | Method shown for P2 peak identification. | 26 |
| 4 | Mean values across all subjects for N1 latency and amplitude and P2 latency and amplitude for all stimulus conditions. | 28 |
| 5 | N1 latency mean values for each subject across stimulus conditions. | 29 |
| 6 | N1 amplitude mean values for each subject across stimulus conditions. | 30 |
| 7 | P2 latency mean values for each subject across stimulus conditions. | 31 |
| 8 | P2 amplitude mean values for each subject across stimulus conditions. | 32 |
| 9 | N1 latency values at each electrode for each stimulus condition. | 36 |
| 10 | P2 latency values at each electrode for each stimulus condition. | 36 |
| 11 | N1 amplitude values at each electrode for each stimulus condition. | 38 |
| 12 | P2 amplitude values at each electrode for each stimulus condition. | 38 |

CHAPTER I

INTRODUCTION

Sensory processing is one of the most basic of human neurological functions, yet, it is an extraordinarily complex process. For every sensory stimulus encountered, afferent signals are sent to specialized brain regions for processing before nerve cell receptors in these regions can properly receive and decode these signals. Despite the complexity inherent in sensory processing, there are many aspects of an average persons life that require him/her to be able to perform this process with ease and efficiency. For example, a college student walking across a university campus uses visual, auditory, and vestibular cues to avoid running into people or objects as well as maintain their balance while walking. A person at dinner receives multiple visual, olfactory and/or tactile stimulus inputs that tell him/her whether the food they are ingesting is healthy and enjoyable. Two friends having a conversation are receiving and processing visual and auditory stimuli as well as linguistic signals while maintaining a smooth flow of conversation. These are all scenarios humans engage in on a regular basis without stopping to think about them, yet they require an immense amount of neural activity and accuracy in neural connections, and they are prime examples of how humans use sensory inputs to inform and influence their behaviors.

While processing a single sensory stimulus is complex, the process escalates in complexity when there are multiple sensory stimuli being presented simultaneously. Processing multiple sensory inputs at the same time helps us perceive and understand the world in which we live with greater accuracy and in some cases more efficiently. We use multisensory

processing to help us pay attention during a conversation in a crowded room where there are multiple conversations going on, localize objects in proximity to us, and increase accuracy in detecting sounds. We also use multisensory processing to learn in environments that require us to attend to and process multimodal sensory stimuli at the same time without becoming confused or disoriented as to what is being presented. The ability to perceive our environment accurately and efficiently aids in survival and improving the quality of our lives.

Given that multisensory processing is such an important part of the typical human experience it logically follows that pathologies affecting a person's sensory processing abilities will greatly affect their daily functioning and quality of life. Understanding the neurology of multisensory processing provides insight into such neurological pathologies. Therefore, studying multisensory processing is beneficial in understanding typical brain functions as well as neurological disorders.

CHAPTER II

BACKGROUND

Sensory Processing

The human brain can be functionally divided into primary sensory cortices, motor cortices, and association cortices. The primary sensory cortices include the primary auditory cortex, the primary visual cortex, and the primary somatosensory cortex (Purves, Augustine, Fitzpatrick, Hall, LaMantia, McNamara, & White, 2008). When discussing sensory processing, one area of interest is how these primary sensory cortices interact with the association cortices. Each of these cortices is responsible for the detection and analysis of sensory inputs from the environment specific to their specialization. For example, the primary visual cortex processes visual stimuli and the primary auditory cortex processes auditory stimuli. Once each of these cortices has processed their appropriate stimuli they then send that information to the association cortices where sensory inputs, motor inputs and cognitive inputs are all integrated. This is not necessarily always the case, and the primary sensory cortices may process some stimuli and send only higher order information to the association cortices to process, but this is still being studied in order to be more fully understood. While it is apparent the primary sensory cortices are responsible for processing the sensory inputs specific to them, what is not known is the role each of these cortices plays in the detection and integration of multiple sensory inputs and how they interact with each other during

multisensory processing.

Multisensory processing

Evidence from previous research on multisensory integration reveals three important features related to multisensory processing. First, multisensory processing is an integrative process in which sensory stimuli are combined and processed as one sensory input (Miller, 1982; Murray, Foxe, Higgins, Javitt, & Schroeder, 2001). Second, sensory integration abilities are affected by sensory deprivation, neural plasticity, and environmental factors (Gilley, Sharma, Mitchell, & Dorman, 2010; Royal, Krueger, Fister, & Wallace, 2010; Hanganu-Opatz, 2010). Third, anatomical evidence from animal studies suggests that this integrative process of multisensory processing happens in cortices that have been traditionally thought to be unimodal sensory cortices (Murray, Molholm, Michel, Heslenfeld, Ritter, Javitt, Schroeder, & Foxe, 2005; Schroeder, Smiley, Fu, McGinnis, O’Connell, & Hackett, 2003).

Traditional views of sensory processing suggest that sensory inputs are decoded separately in unimodal brain regions, and that integration of these inputs occurs in later processing stages, for example in association cortices. Early models of sensory processing showed separate activation of individual sensory cortices in response to sensory stimuli. These models posited that the separate responses would then race against each other, with the faster of the two responses winning. Thus, these models are referred to as “race models” (Raab, 1962). However, studies from the last twenty years of multisensory processing research have found evidence that refutes this idea of multisensory processing and new models, called co-activation models, have been introduced. Co-activation models of multisensory processing show independent sensory cortices being activated in a combined manner, which allows the criteria for initiation of a response to be met faster (Miller, 1982). These models are based

on the idea that a certain amount of activation is needed before a response can be elicited, and when there are two or more sensory stimuli being detected at a time, activation can be achieved at a faster rate.

In his 1982 study, Jeff Miller designed an experiment to test behavioral responses to redundant and multimodal sensory stimuli. Miller appraised response times to multiple redundant signals as well as response times to multiple independent signals to determine whether the different response times would support the single-activation models or the co-activation models. According to single activation models, response times to multiple inputs cannot be faster than the response times to a single input because the response is based on the speed capabilities of individual sensory receptors. According to the co-activation models, response times to multiple inputs can be faster than response time to single inputs because the response is based on cumulative activation power in order to elicit a response. The more sensory receptors are being activated, the greater the activation power and the faster the response. The experiment performed by Miller (1982) demonstrated that response times to multiple sensory inputs are statistically faster than a single-activation model would allow, thus, Miller concluded that multisensory processing must adhere to a co-activation model. While this experiment did not prove that multisensory processing is an integrative process in which sensory inputs are combined, it did help rule out the idea that multiple sensory inputs are processed in a separate, independent fashion and only integrated at higher cortical levels. Further research has replicated Miller's results and supported his initial findings, providing further evidence of the integrative nature of multisensory processing (Ulrich, Miller, & Schröter, 2007).

A combined processing effect was also found in a study by Murray et al., 2001. In that study, visual stimuli were presented to two separate visual fields of adult subjects and response times for detection of the stimuli were recorded. Participants were presented with stimuli to each visual quadrant independently and simultaneously. The reaction times for the simultaneous presentation of stimuli were faster than the reaction times for the independent

stimulus presentation. These findings provided evidence that not only was the combined sensory stimulus processed in an integrated manner, but the stimulus was combined across visual quadrants. These results indicated a non-linear process in sensory processing, one in which sensory inputs are integrated and processed simultaneously rather than sequentially.

Sensory processing in disordered populations

Just as the study of the non-disordered neurological system can provide insight into its function and processing abilities, study of these same functions and processes in disordered populations can also provide insight. For example, Gilley, Sharma, Mitchell, and Dorman (2010) examined how the auditory-visual integration skills of children were affected by early auditory sensory deprivation. Gilley et al. compared auditory-visual detection in children implanted with cochlear implants before 3.5 years of age, children implanted with cochlear implants after 7 years of age, and a group of normally hearing children aged 7 to 12 years. Using Miller's "test of race model inequality" (Miller, 1982) as a measure of sensory integration, the authors presented auditory, visual and auditory-visual stimuli to each group of subjects and then measured the reaction times for each group. According to the test of race model inequality, faster reactions to combined sensory stimuli than to unimodal stimuli would suggest that early co-activation mechanisms modulate integration processes. Results from Gilley et al. revealed that, similar to the normally hearing children, the children who were implanted early had faster reaction times to combined sensory stimuli. Those children who were implanted later had response times that were slower than the early implanted and normally hearing subjects for both the auditory stimulus and the auditory-visual stimulus. These slower response times in the auditory-visual condition suggested a delay in the subject's sensory integration abilities. The overall results of the study imply that auditory sensory deprivation causes delays in auditory-visual integration abilities. The results

also imply that if the sensory deficit is treated early enough, integration abilities can be re-established.

Bergeson, Houston, and Miyamoto (2010) investigated the effects of hearing loss and intervention on audiovisual perception abilities of children. Bergeson et al. compared the abilities of normal hearing infants with those of infants who had received cochlear implants and those who used hearing aids. Their study sought to understand the extent to which audiovisual speech perceptual abilities were affected by hearing loss and whether or not the type of amplification device used in intervention affected the infant's multisensory processing abilities. That study compared three groups of children: normally hearing infants aged 11.5-39.5 months, hearing impaired infants aged 8-28 months who had been fitted with hearing aids between the ages of 2 and 19 months, and hearing impaired infants aged 16-39 months who had received a cochlear implant between the ages of 10 and 24 months. A visual of a woman saying the word "judge" and a visual of the same woman saying the word "back" were presented simultaneously to the infants. In addition to these visual stimuli, an auditory stimulus of either the word "judge" or "back" was presented at the same time. Bergeson et al. then observed the infant's behavior to determine which visual stimulus they paid most attention to. The normally hearing infants immediately matched the visual stimulus with the correct auditory stimulus. The infants with the hearing aids did not match the visual stimulus with the auditory stimulus, and the infants with cochlear implants did not at first correctly match the audiovisual stimuli but they improved with practice. The results from the Bergeson et al. study provide further evidence that early sensory specific deprivation affects an infant's sensory integration abilities. Also, there is strong evidence from the study that the type of early intervention employed can impact how much of these abilities can be recovered.

To examine the effects of visual deprivation on sensory integration, Amedi, Raz, Azulay, Malach, and Zohary (2010) compared brain activity using fMRI during tactile object recognition in sighted and blind subjects. Visually impaired participants served as the control

group for determining whether visual imagery is a result of multi-sensory inputs or strictly related to visual stimulation. The authors determined that activation in the visual cortex during a tactile object identification task occurred for both blind and sighted participants. The activation was negligible during hand movements and occurred bilaterally regardless of the hand being used. The Amedi et al. results indicate two things: visual imagery is not something that is linked only to visual stimuli but also tactile stimulation, and tactile stimulation is at least partially processed in the visual cortex.

Other studies have also addressed how sensory deprivation affects sensory integration and sensory processing (Putzar, Hötting, & Röder, 2010; Tremblay, Champoux, Lepore, & Thoret, 2010; Bottari, Nava, Ley, & Pavani, 2010). Each of these studies has addressed a different aspect of multisensory processing in a wide range of populations, from infants with cataracts to adults with cochlear implants and adults who were profoundly deaf who did not use amplification. The findings of each of these studies shed light on the integration of multimodal sensory stimuli and how multi-sensory processing can be affected by sensory deprivation. These studies also provided the opportunity to examine how neural plasticity and the environment play a part in determining multisensory processing abilities in an individual.

In exploring how neural plasticity and environment affect multisensory processing abilities, Royal, Krueger, Fister, and Wallace (2010) studied the effects of blindness on multi-sensory integration and whether or not reestablishing sight would in any way restore sensory integration abilities. Royal et al. took cats that had been born and raised in complete darkness for 6 months and placed them in a normally lit environment for the same approximate amount of time and compared them with cats that had been raised in a normally lit environment for both time periods. At the end of the second time period both groups of cats were implanted with electrodes to measure unimodal and multimodal sensory neuronal responses. Once implanted, the cats were presented with auditory, visual, and auditory-visual stimuli and their responses were recorded.

Findings from the Royal et al. study included differences in the neural make-up and the reaction times of the dark-reared cats from the normally-reared cats. While the dark-reared cats were able to develop some multi-sensory integration abilities, there were deficits and differences in the neurology and functioning of the dark-reared cats that caused these abilities to be incomparable to the abilities developed in a normal environment. These findings suggest there is an effect on multi-sensory processing and integration abilities from sensory deprivation. The findings also suggest that while there is some plasticity within the system that allows for reorganization and adjustment of the system, this plasticity is limited and deficits remain, despite a noticeable improvement once sensory input has been reestablished.

The Royal, Krueger, Fister, and Wallace (2010) research also suggests that there seems to be a sensitive period for sensory development and integration along with the plasticity in the neural sensory systems that allows them to respond to intervention and environmental factors. Results from other studies have verified these findings (Fiehler & Rösler, 2010; Röder & Wallace, 2010) and reveal converging evidence for a sensitive period for sensory development and integration.

Additionally, there appears to be a large amount of plasticity in the developing sensory system that facilitates intervention in young children with sensory impairments or processing deficits. Depending on the sensory disorder, amount of deprivation seen, and how early corrections are made; the plasticity of the system can vary. This indicates that the sensory system is affected by both nature and nurture. Ileana Hanganu-Opatz (2010) reviewed the current knowledge base on functional cortical maps of sensory processing with attention to their functional development. Evidence from that review suggests that development of sensory pathways in the cortex is affected by both environmental and molecular inputs. The author concludes that functional cortical systems for sensory processing, including multi-sensory processing, are innate, and environmental inputs help to refine and further develop these systems during the maturation process.

Cortical mapping of multisensory processing

The research discussed thus far has shown how the typical sensory system works and has provided insight into how sensory disorders affect the system. Taken together, the previously cited research suggests that multisensory processing is an integrative process. It is reasonable to assume, then, that the cortex contains functional connections that support such an integrative process. For example, studies examining detection and subsequent encoding of a sensory stimulus provide insight into how the functional brain connections can support both unimodal and multimodal sensory processing. Interactions between cortical structures and sensory systems are referred to as functional brain connectivity, a term which implies cortical regions are not only anatomically connected but also connected through the functions they provide, such as sensory processing. Finding anatomical or physical evidence that suggests a functional connection between these cortical structures is an important piece of understanding multisensory processing.

In their 2006 study Budinger, Heil, Hess and Scheich probed whether primary sensory cortices are strictly unimodal or can be activated by other sensory modalities. Using a bi-directional neuronal tracer inserted into the primary auditory cortex of Mongolian gerbils, the authors found the gerbils' primary auditory cortices (A1) contained neural cells that were specifically equipped to receive direct signals from somatosensory, visual, and multi-sensory cortices as well as the visual and multisensory thalamic and brainstem structures. They also found several axonal projections that originated in the A1 and terminated in other sensory cortical areas not usually affiliated with auditory perception. This finding of neural cells that directly projected from various sensory cortices to the auditory cortex and the projections from the A1 to other sensory areas both cortically and subcortically suggest that sensory cortices previously thought to be unimodal are in fact equipped to process multiple sensory inputs.

A similar study by Schroeder, Lindsley, Specht, Marcovici, Smiley and Javitt (2001) used electroencephalography to examine the primary auditory cortex of macaque monkeys. The authors attached electrodes to the auditory cortex and the cortical regions posteriorly adjacent to it. They then presented both auditory stimuli to the monkeys and labeled the responses accordingly. During the second phase of the study, the monkeys were presented with a somatosensory stimulus and the responses were recorded and then compared with the previous results. The results indicated that the somatosensory stimulus caused somatosensory responses in the monkey's auditory cortex. In their 2002 study, Schroeder and Foxe expanded their research to determine if visual inputs could also be found in the primary auditory cortex of macaque monkeys, further solidifying the idea that the primary auditory cortex is indeed a center for multisensory processing. The results of their study included findings of visual inputs in the A1 region of the monkeys. These results, combined with the results of their earlier study, shed light on how multiple sensory inputs are simultaneously processed at subcortical levels. There have been multiple studies that have corroborated these findings and have found similar results in humans (Molholm, Ritter, Murray, Javitt, Schroeder, & Foxe, 2002; Schroeder, Smiley, Fu, McGinnis, O'Connell, & Hackett, 2003).

Wallace, Ramachandran and Stein (2004) recorded local field potentials from rat occipital, temporal, and parietal cortices to determine the presence of multi-sensory neurons in sensory specific cortical regions. Electrodes were placed in each sensory cortex and recordings were taken during visual, auditory and somatosensory stimulation. Responses from each cortex were analyzed to determine the presence of sensory specific neurons based on responses to each stimulus modality. Wallace et al. (2004) found that the presence of multi-sensory neurons in sensory specific cortices was low and placement was concentrated along the borders of each cortex. In other words, the auditory cortex would include visual and somatosensory neurons along its borders, the visual cortex would include auditory and somatosensory neurons along its borders, and the somatosensory cortex would include visual and auditory neurons surrounding its borders. Wallace et al. concluded that such a low

presence of multi-sensory neurons in sensory specific regions corroborated the view that sensory processing is independent in the subcortical regions with integration taking place at higher cortical levels. However, the authors also concluded that the presence of multisensory neurons in the sensory cortices suggested that at least some sensory integration or combined sensory processing is occurring at lower cortical levels.

This study and others like it (Martin, Huxlin, & Kavcic, 2010; Kreitewolf, Lewald, & Getzmann, 2011; Murray et al. 2005) provide further insight into the mapping and structures of the brain that contribute to sensory processing and the process by which they receive and send sensory information. The overall finding of these studies and the recent research in cortical mapping of sensory processing is that multisensory processing and multisensory integration is not confined to upper cortical structures. In fact, the sensory specific areas of the brain appear to have the capability to receive and integrate sensory inputs not specific to them.

Hanganu-Opatz (2010) reviewed evidence that both single and multisensory processing begin at subcortical levels, with multiple sensory inputs being integrated at least to some extent before they reach the cortical levels. This evidence includes cellular patterns in the primary sensory cortices of both animal and human subjects that support multisensory processing at subcortical and cortical levels very early in development, well before environmental factors have begun to have an impact on the sensory systems. Hanganu-Opatz concluded that both the molecular and environmental evidence supported the view that multisensory processing is an integrative process that can be affected by environmental factors and occurs at subcortical levels.

Use of electroencephalography in studying multisensory processing

Many of the studies discussed thus far used electroencephalography (EEG) to study sensory processing. EEG is used to study electrical activity originating in the brain. An electroencephalogram uses multiple electrodes placed on the scalp to measure the electrical potentials generated by neuronal activity. Neurons are grouped together into neural circuits. When a neuron in the circuit is activated the action potential created by that neuron triggers other neighboring neurons in the circuit, producing a wave of propagated activity. This wave of activity grows larger as it passes from neuron to neuron, circuit to circuit, creating potentials large enough to be detected with EEG (Purves et al., 2008). In this way, the original neural signal is conducted from the region of the brain where it first started to the outer regions of the skull and scalp. Once this neural activity has reached the scalp, EEG electrodes detect, amplify, and digitize the signals, recording them for further study. Researchers can then process the data and create images to study. EEG has long been used to study activity in the brain including sensory processing. The results of EEG studies are reliable and replicable making EEG a good choice for further research in the area of multi-sensory processing (Burgess & Gruzelier, 1993; McEvoy, Smith, & Gevins, 2000; Gudmundsson, Runarsson, Sigurdsson, Eiriksdottir, & Johnsen, 2007).

Using EEG to detect and study event related potentials

When using EEG to detect and measure neuronal activity, the scalp recording contains multiple overlapping signals. Synchronous activity that originates from the axons of the brainstem pathways produces very fast, high-frequency potentials in the EEG recording. Conversely, post-synaptic dendritic currents in the cerebral cortex produce slow, low-frequency signals in the scalp EEG. These different types of activity can be separated by amplifying the electrical signal in pre-defined frequency ranges, or bands. For the purposes of studying the primary sensory cortices, which produce low-frequency cortical potentials,

the signals in the lower frequency bands are amplified.

In addition to differences in origins and frequency of EEG signals, there is also a difference in the rhythm of EEG signals. Spontaneous or random activity in the brain is constant, resulting in spontaneous activity in the EEG. This spontaneous brain activity has a temporal pattern to it that is randomly distributed, which allows extraction of non-random activity by averaging multiple time-locked trials. Additionally, properties of the time-locked voltage distributions allow the use of higher-order statistical analyses to extract the signals of interest.

There are various types of signals that are considered signals of interest in research. Two of these are evoked potentials (EPs) and event related potentials (ERPs). EPs are potentials that result from passive sensory processing and are simply responses to the sensory stimuli in our environments. Because they occur in response to stimuli outside our bodies and do not require any action in response, these potentials are also referred to as exogenous potentials. ERPs are electrical potentials that result from a subject attending and responding to the presentation of a stimulus. These potentials are also referred to as endogenous potentials because they are thought to originate within the person. ERPs can be studied by analyzing the series of peaks the potentials create in an EEG recording and classifying these peaks by several characteristics, including latency and amplitude (Crowley & Colrain, 2004). ERPs are thought to be related to the various stages of processing sensory and cognitive events and the peaks can provide information about the timing of information processing.

The series of peaks in an ERP will change based on the modality of the stimulus. In typical adults the auditory evoked potentials, or AEPs, are represented by a series of well-defined peaks. The first positive peak, the P1, occurs at about 50 *ms* after stimulation. The first negative peak, the N1 occurs in the 90 to 150 *ms* range after stimulus onset, and the second positive peak, the P2, occurs in the 160 to 200 *ms* range after stimulus onset. The second negative peak, the N2, occurs at about 275 *ms*, and in many subjects it can be

absent. The third positive peak, the P300, occurs between 275 and 300 *ms* (Hall, 2006). In adults the N1 peak is the peak that indicates the most robust response in the auditory cortex.

In visual evoked potentials (VEPs), the first negative peak, the N1, occurs at approximately 75ms after stimulus onset. The first positive peak, the P1, occurs at approximately 125ms after stimulus onset, and the second negative peak, the P2, occurs at about 135ms after stimulus onset. The second positive peak, the N2, occurs at about 200 *ms* and the third positive peak, the P300, happens between 250 and 300 *ms*. For all sensory modalities, the earlier ERP peaks are those with shorter latencies, such as the P1, N1, and P2. Later peaks are those that have longer latencies, such as the N2 and the P300.

In cognitive sensory processing shorter latency peaks occur earlier in the sensory processing stage than the longer latency peaks that follow them. Shorter latency peaks, such as the P1, N1, and P2 are thought to be linked to modality specific perceptions, while longer latency peaks, such as the N2 and the P300 are thought to not be specific to a particular sensory modality (Michalewski, Prasher, & Starr, 1986; Spence & Driver, 2004). For both AEPs and VEPs the N2 and P300 are considered to be cognitive potentials and represent identification and classification of a stimulus as well as activation of responses and are sensory integrative.

Using the N1 and P2 to identify multi-sensory processing

Because the N1 and P2 peaks are considered earlier peaks that reflect sensory modality specific inputs, it follows that they can be considered valid representations of sensory processing in the brain. The N1 peak has long been investigated and implicated in multi-sensory processing (Näätänen & Picton, 1987; Crowley & Colrain, 2004; Pérez, Meyer, & Harrison, 2008). Although the N1 has a strong presence in visual processing, it can also be

clearly seen in somatosensory and auditory processing, making it an easy peak to identify and study in any type of sensory processing research. The P2 has not historically been explored as intensely as the N1 peak. For many years researchers thought the N1 and P2 peaks were connected to each other, creating a wave complex, rather than resulting from different anatomical regions. Research performed on the P2 peak was done in conjunction with study of the N1 peak. Recent research has shown the P2 peak to be independent from the N1 peak, varying in response to changes in stimuli characteristics, and having a different maturational pattern of development (Crowley & Colrain, 2004; Hall, 2006).

One interesting viewpoint on the origin of the N1 and P2 peaks comes from Crowley and Colrain (2004). Those two authors suggest that due to the similarity of response in the auditory the N1 and P2 peaks and the somatosensory N1 and P2 peaks, these two peaks should be considered partially representative of exogenous responses, which do not require the subject to attend to the stimulus. Past research would suggest that the N1 peak is directly affected, however, by attending to a stimulus, but the P2 peak is not (Näätänen & Picton, 1987; Näätänen, Gaillard, & Mäntysalo, 1978; Pérez, Meyer, & Harrison, 2008). There is still much debate about whether or not there is truly an attentional effect on both the N1 and P2, with much research having been done to compare latency and amplitude of the peaks in both sleep conditions and during attention tasks. The results of the research vary along with the interpretations of the results and there is still no consensus as to what the changes in the auditory N1 and P2 mean for each condition (Crowley & Colrain, 2004).

Several research studies have looked at the functional significance of the P2 auditory peak. These studies suggest the P2 peak is related not only to the detection of a stimulus but the perception that a stimulus is being detected and is thus, related, at least somewhat, to stimulus classification and discrimination (Crowley & Colrain, 2004; Čeponienė, Torki, Alku, Koyama, & Townsend, 2008). Perception involves more attention and cognition than just sensory detection and implies that attention and cognition can affect the latency and amplitude values of the P2. However, there have been some studies done that demonstrate

changes in the latency and amplitude of the P2 peaks can be elicited while the subject is sleeping or during non-attentional tasks, thus raising the question that perhaps the changes seen in the P2 peak are more related to sensory processing rather than cognitive or attentional activity (Crowley & Colrain, 2004).

There is research that indicates both the auditory and visual N1 peaks result from the initial detection and processing of sensory stimuli, and although it is affected by attention, the N1 peak is not reflective of the higher cortical stage analysis of stimuli that the N2 and P300 are thought to reflect. The P2 peak, on the other hand, appears to be relatively similar topographically and temporally across sensory stimulation with only slight attentional effects, suggesting it is reflective of both unimodal and multimodal sensory detection (Crowley & Colrain, 2004). Taken together, the above statements indicate that the auditory and visual N1 and P2 peaks are results of early neural activation in sensory specific cortices, yet they are capable of integrating multiple sensory inputs (Näätänen & Picton, 1987; Crowley & Colrain, 2004). Thus, changes in the N1 and P2 peaks during multisensory stimulation can indicate integration of sensory stimuli, at least in part, at lower cortical levels.

Latency and amplitude of the N1 and P2 peaks

As mentioned earlier, comparison and analysis of ERPs can be done by comparing and contrasting peak characteristics. Latency and amplitude of the peaks generated and/or source localization of specific ERPs are peak characteristics that are commonly used to study ERPs. For this study we are comparing the latency and amplitude of the N1 and P2 peaks for ERPs elicited during an experimental task with three stimulus types—an auditory stimulus, a visual stimulus, and a combined auditory-visual stimulus.

Assuming that sensory specific areas are capable of detecting and integrating other sensory inputs, there should be an effect seen in the ERPs recorded during multi-sensory

tasks. This effect should be seen in the peaks elicited during sensory specific tasks such as the N1 and P2. This study will compare the latency of ERPs elicited during single sensory tasks with those elicited during multi-sensory tasks. If during a dual sensory processing task, latency increases in an earlier occurring peak, such as the N1, it would indicate that multiple sensory inputs are affecting the elicitation of the peak, and integrative processing has begun at that stage rather than at a later stage.

Similar to latency, the ERP amplitude is related to sensory modality specific processing. The amplitudes for the visual N1 peak are higher for visually specific stimuli and the amplitudes for the auditory P2 are higher for auditory specific stimuli. If the amplitude for either of these two peaks is increased it is assumed that the sensory input that is specific for each of those peaks is the one that is most salient in the experiment condition. However, because the amplitudes of these peaks are also related to attention and cognition, the attentional or cognitive demands of an experiment task can affect the ERPs of the N1 and P2 peaks. For example, if an experimental condition has multiple sensory stimuli present but the subject is instructed to attend to only the visual stimulus then the amplitude of the N1 is increased more than the amplitude of the P2 because the subject is focusing their attention on the visual stimulus. Similarly, if the subject was instructed to focus on the auditory stimulus then the increase in amplitude of the P2 would be greater than an increase in amplitude for the N1 because the subject is focusing their attention on the auditory stimulus (Spence & Driver, 2004).

Hypothesis

This study will examine differences in sensory processing from sensory specific and multisensory stimulation. The goal of this study is to identify and describe changes in EP/ERP components that reflect multisensory processes. Because the N1 peak is clearly

present for both auditory and visual conditions and is strongly linked to unimodal sensory processing, it is an important peak to study in determining differences in unimodal and multimodal sensory processing. Because the P2 peak is so closely associated with the N1 peak, and because it is the last identified peak to occur in what is considered to be the early sensory processing stage before later cortical processing stages are identified, it is an ideal peak to use in studying multisensory processing.

The author hypothesizes that the findings of this study will reveal multisensory processing earlier in the cortical process than previously thought, occurring at the same stages as unimodal sensory processing, and this will be evident by changes produced in the N1 and P2 peaks, namely there will be a decrease in latency resulting in an earlier onset time for each peak and an increase in amplitude resulting in larger peaks for both the N1 and P2.

CHAPTER III

METHODS

Subjects

Participants for this study were 9 normally hearing female adults and three normally hearing male adults aged 23.7 to 26.16 years [$mean = 24.46$, $SD = 0.89$]. The participants were screened using a questionnaire that assessed hearing, speech, language, visual, and neurological development. Criteria for participation in the study were hearing thresholds at or below 20 *dB* HL, normal speech language and neurological development, and normal or corrected-to-normal vision. A portion of the participants in this study were the same as those reported in Gilley et al. (2010). All participants were college students recruited for the study from undergraduate classes. A small financial compensation was provided in exchange for participation.

Stimulus conditions

There were three experimental conditions for this project. The first was an auditory alone condition in which only an auditory stimulus was presented. The auditory stimulus used for this condition was a 1000 *Hz* pure tone 60 *ms* in duration (5 *ms* rise/fall times).

This stimulus was presented at 70 dB SPL from a loud speaker situated on top of a video monitor at 0 degrees azimuth. The video monitor was used for the presentation of the visual stimulus described below.

The second stimulus condition was a visual alone condition in which only a visual stimulus was presented. The visual stimulus consisted of a white disk 1.2 degrees in diameter on a black background, presented at a viewing distance of 100 cm with a duration time of 60 ms on a flat panel LCD computer monitor. The disk was presented on the center of the monitor screen ($x = 0, y = 0$). The third condition was a combined auditory-visual condition in which both an auditory and visual stimulus were presented. For this combined condition, the same auditory and visual stimuli used in the auditory alone and visual alone conditions were presented simultaneously with stimulus onset asynchronies of 0 ms .

Presentation of stimuli

All stimuli were presented using Presentation software (Neurobehavioral Systems, Inc., Albany, CA) on a desktop computer with a 23 bit/192 kHz , stereo sound card that was routed to a GSI-61 audiometer (Grason-Stadler, Inc., Madison, WI), as well as a GeForce FX 5700 Ultra video card (NVIDIA Corporation, Santa Clara, CA) routed to a Samsung SyncMaster 710MP TFT flat screen video monitor (Samsung Electronics America, Inc., Ridgefield Park, NJ). Each type of stimuli—auditory alone (A), visual alone (V), and auditory-visual combined (AV)—was presented randomly with equal probability and was randomly interleaved with interstimulus intervals (ISIs) that ranged from 1000 to 3000 ms . Auditory evoked potentials (AEPs) and visual evoked potentials (VEPs) were recorded in response to the presentation of stimuli in all three conditions.

Experimental task

Testing procedures took place in a sound treated booth with the lights turned off. Participants were seated in comfortable chairs in front of the LCD video monitor and were asked to maintain focus on a crossbar in the center of the monitor. They were then instructed to press a button on a video game controller as quickly as possible after they detected either an auditory or visual stimulus. Before testing began, each task condition (A, V, or AV) was described in detail to ensure the participant understood what the task was. Stimuli were presented in blocked sequences of 175-200 trials (each block lasting approximately 5 minutes) with each subject completing 10 blocks. Between blocks, participants were encouraged to take breaks in order to maintain concentration during the research task.

Data collection

Electroencephalography (EEG) data was collected using an electrode cap (Neuroscan Quickcap) with 64 sintered *Ag/AgCl* electrodes placed on the subject's head. Electrode placement was based on the Extended International 10-20 system for electrode placement. Two additional bi-polar channels were included separate from the cap, to monitor eye movement. These electrodes were placed at the right and left lateral-canthi and the superior and inferior orbits of the left eye. EEG activity was recorded using a Synamps2 68-channel acquisition unit (Compumedics/Neuroscan, El Paso, TX) and was digitally stored on a PC computer. Incoming EEG was filtered from DC to 200 *Hz* at 1000 *Hz* sampling rate. The stimulus onset times were digitally encoded in the EEG by sending a time-locked, low voltage TTL signal from the stimulus computer to the Synamps unit.

Analysis

All analysis was completed off-line after completion of the recording session. The EEG signal was passed through a high pass filter (0.1 *Hz* cutoff, zero-phase shift, 12 *dB/octave*) in order to remove slow DC drift in the recordings. Continuous EEG was examined for contaminating artifact, and all EEG blocks containing excessive noise were rejected from further analysis. Eye blink contaminating artifacts were removed by applying a spatial filter using a linear derivation computed from the spatio-temporal singular value decomposition (SVD) of the average eye blink across the 64 recording channels and the two eye channels. Once contamination artifacts were removed, the corrected EEG was then divided into epochs around the stimuli using a 100 *ms* pre-stimulus interval and 600 *ms* post-stimulus interval. Each epoch was baseline corrected to the average amplitude of the entire waveform.

Peak analysis

EEG epochs were averaged for each subject in order to obtain the event related potential (ERP) for the N1 and P2 for all conditions across the testing period. The ERPs for each peak from the auditory and visual alone stimuli conditions and the auditory-visual condition were combined and compared for each subject. Once the epochs for each subject averaged, these files were analyzed to identify the peaks. Data from eleven electrodes—FZ, FC5, FCZ, FC5, C5, CZ, C6, P5, PZ, P6, and OZ—was used to identify peaks (See Figure 1). Peaks for each component were identified visually. The N1 peak was identified for the auditory evoked potential (AEP) as the first negative robust peak occurring in the range of 50-200 *ms* after stimulation, and the for the visual evoked potential (VEP), as the first robust peak occurring in the range of 20-150 *ms* (See Figure 2). The P2 was identified as the second positive robust peak for the VEP and the AEP occurring right after the occurrence of the

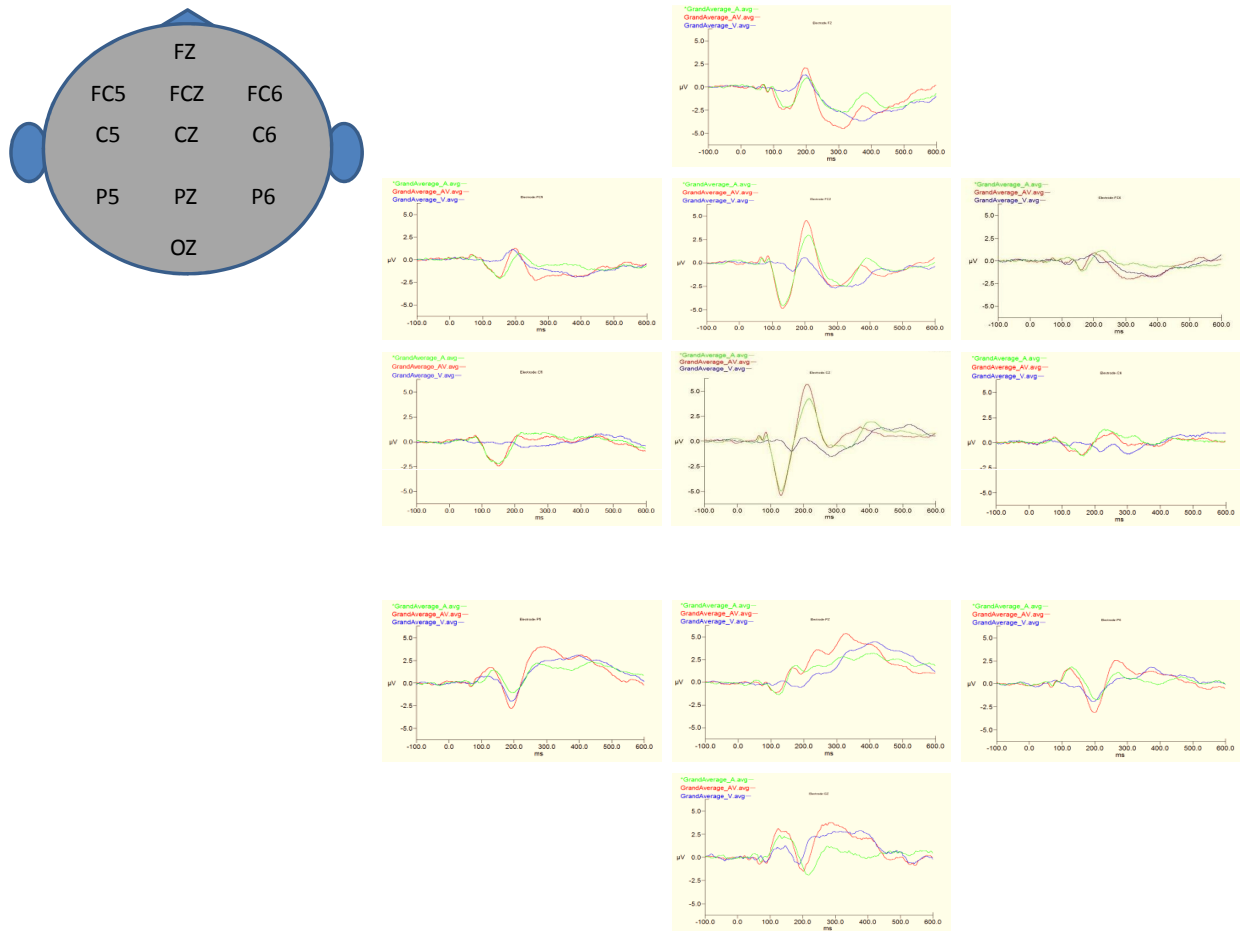


Figure 1: Scalp map of the 10-20 international system indicating the specific electrodes used for this study. Graphs at each electrode location show Grand Averages for each stimulus condition across the subject population.

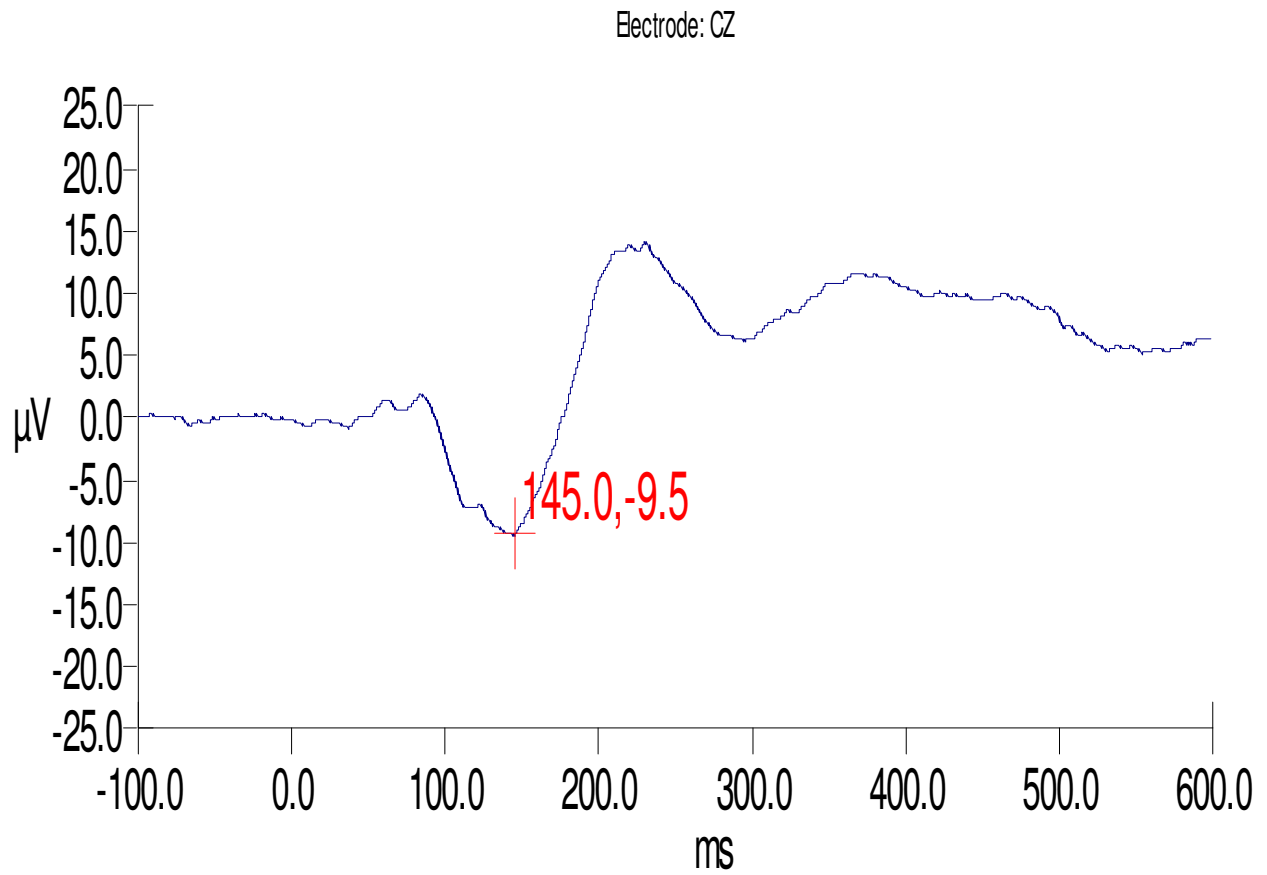


Figure 2: Method shown for N1 peak identification.

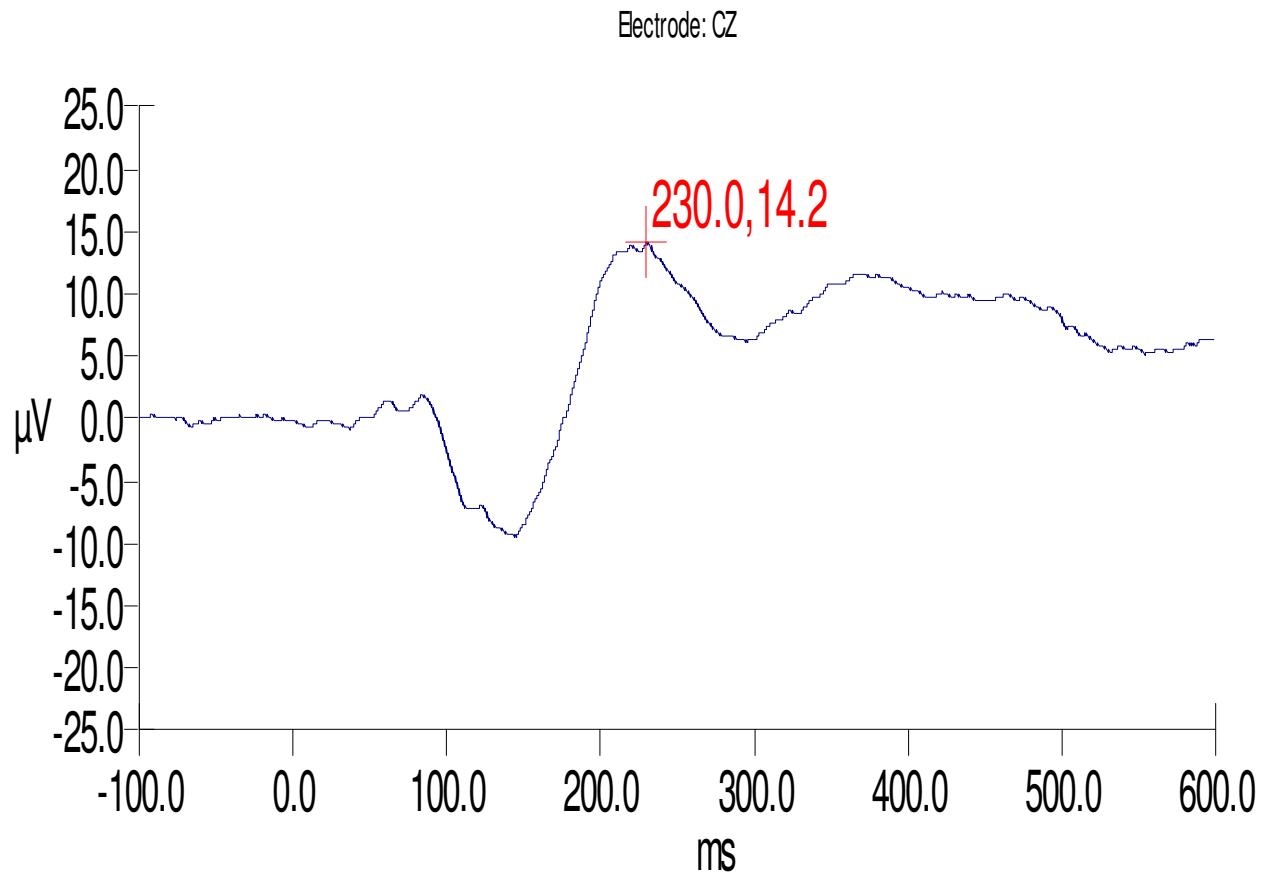


Figure 3: Method shown for P2 peak identification.

N1 waveform (See Figure 3). Latency and amplitude values were determined for the N1 and P2 peaks equally across all conditions and subjects. Once the latency and amplitude values for each subject had been found, these values were then combined to form a grand average for each peak.

Statistical analysis

Using the Grand Averages from all three conditions, the N1 and P2 peaks were again identified, adhering to the same criteria as for the individual subject peak identification. The Grand Averages were then compared to determine differences in the latency and amplitude for both the N1 and P2 peaks across electrodes (See Table 1). In addition to using the Grand Averages, data from eleven channels for each subject was examined and recorded for each condition. The latency and amplitude of the N1 and P2 peaks acted as the dependent variables in within subject separate fully repeated measures analysis of variance (ANOVA) corrected for multiple comparisons (Tukey-Kramer HSD). See Figure 4 for the mean results for each peak by stimulus condition and Figures 5-8 for mean results for each peak by electrode.

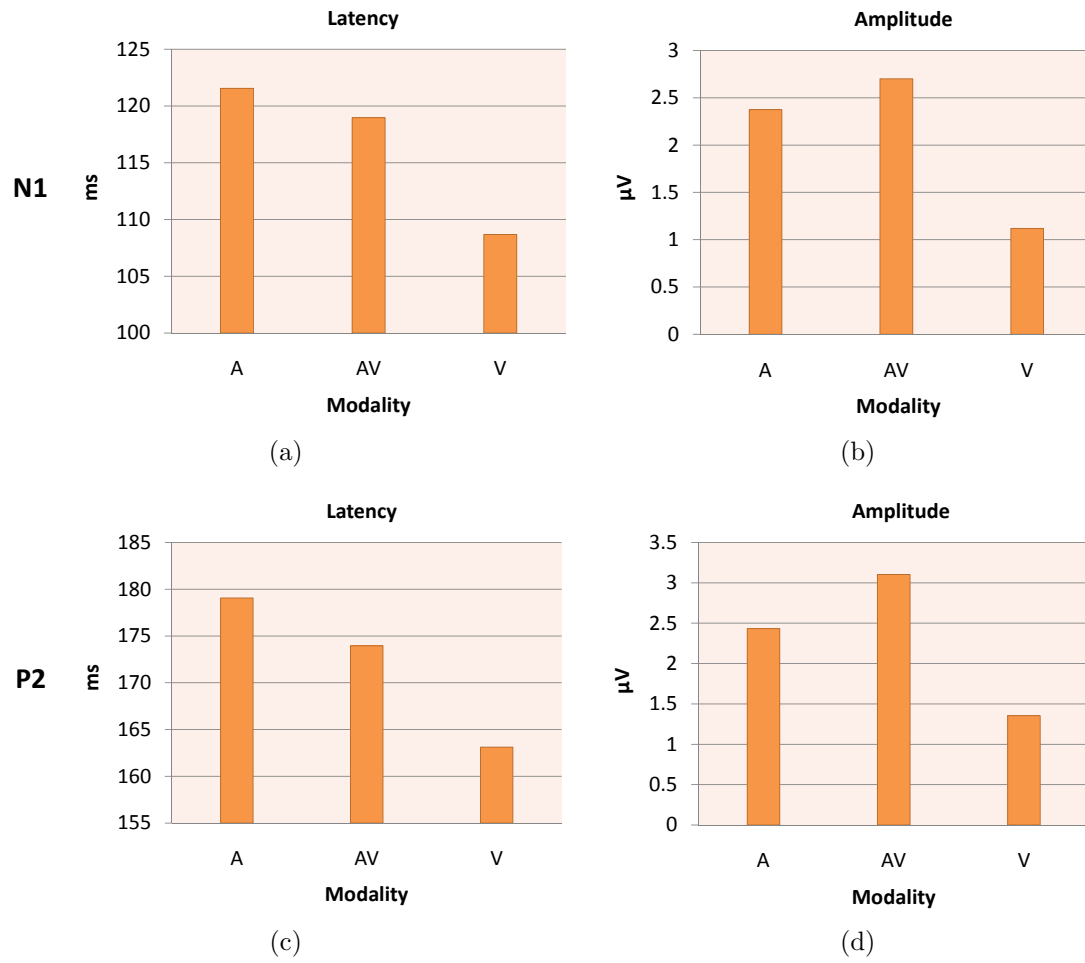


Figure 4: Mean values across all subjects for N1 latency and amplitude and P2 latency and amplitude for all stimulus conditions.

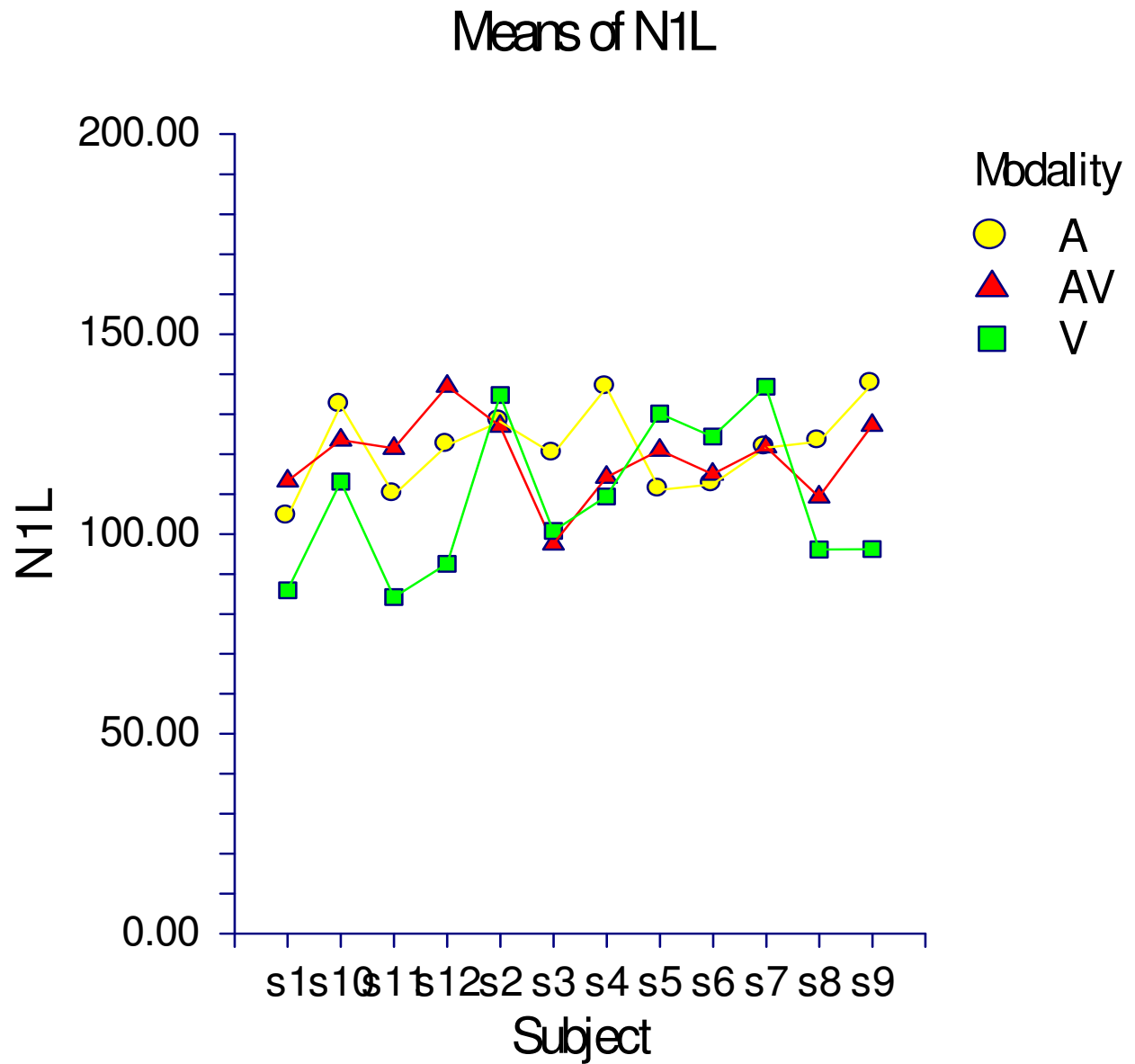


Figure 5: N1 latency mean values for each subject across stimulus conditions.

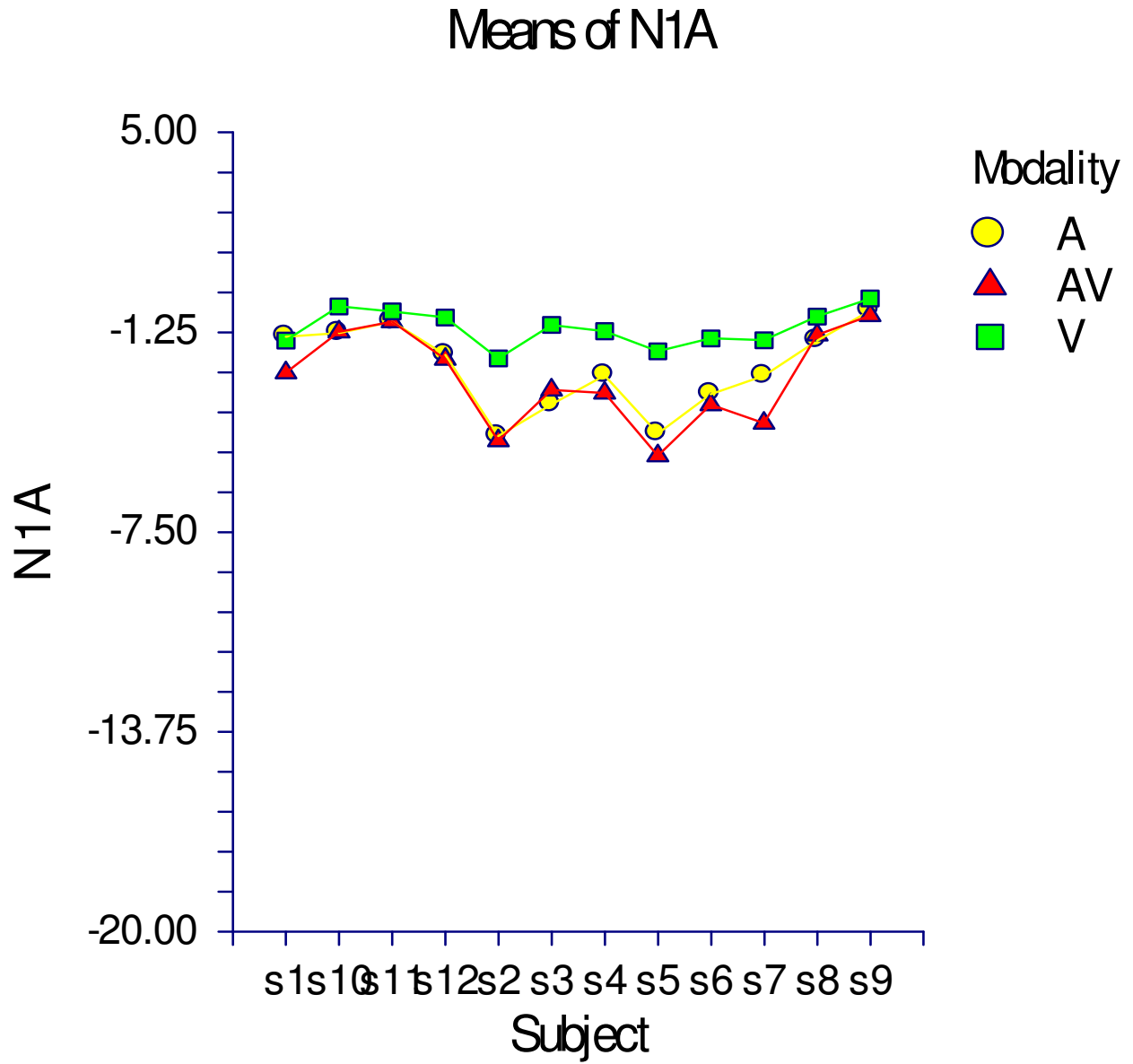


Figure 6: N1 amplitude mean values for each subject across stimulus conditions.

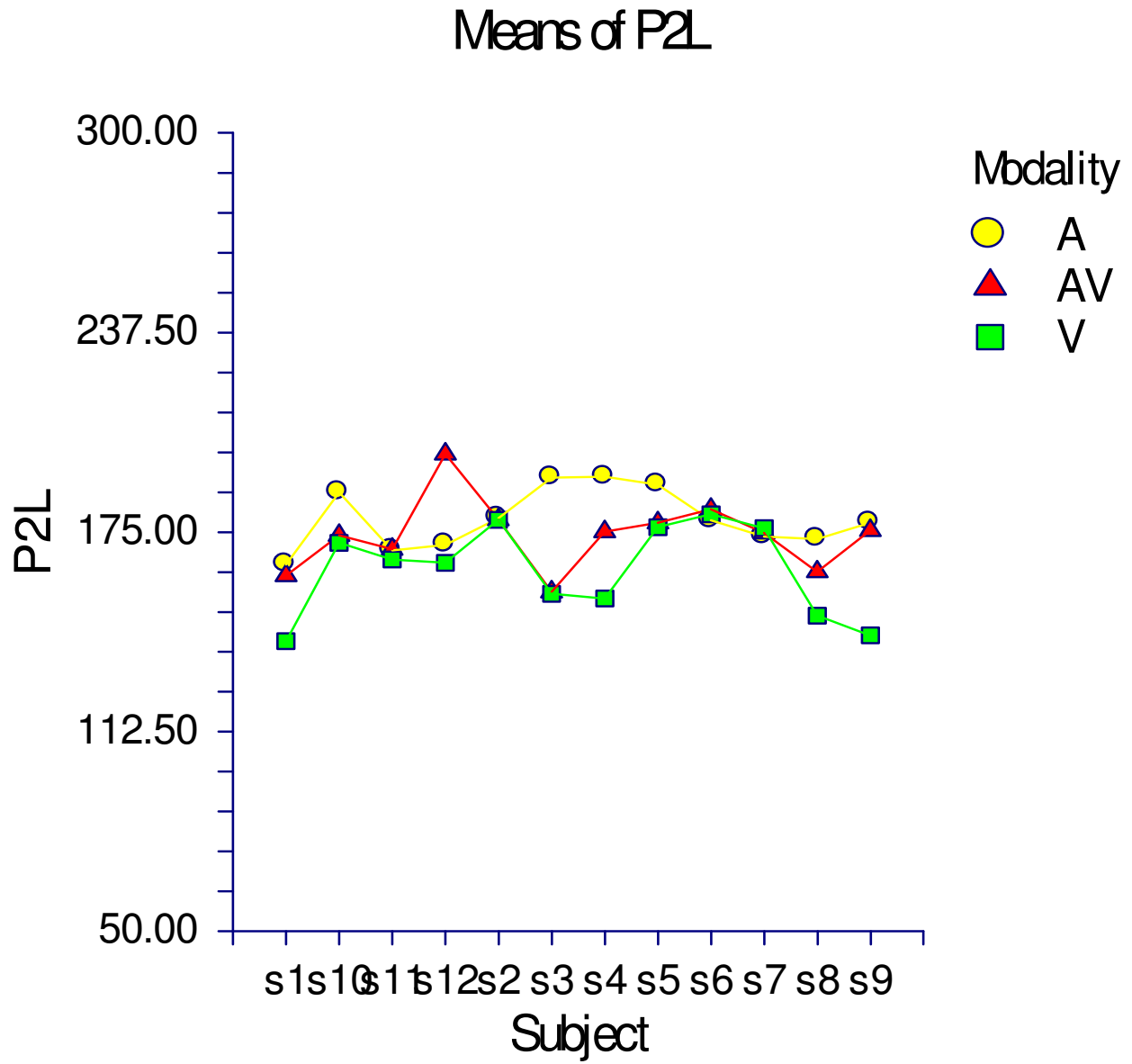


Figure 7: P2 latency mean values for each subject across stimulus conditions.

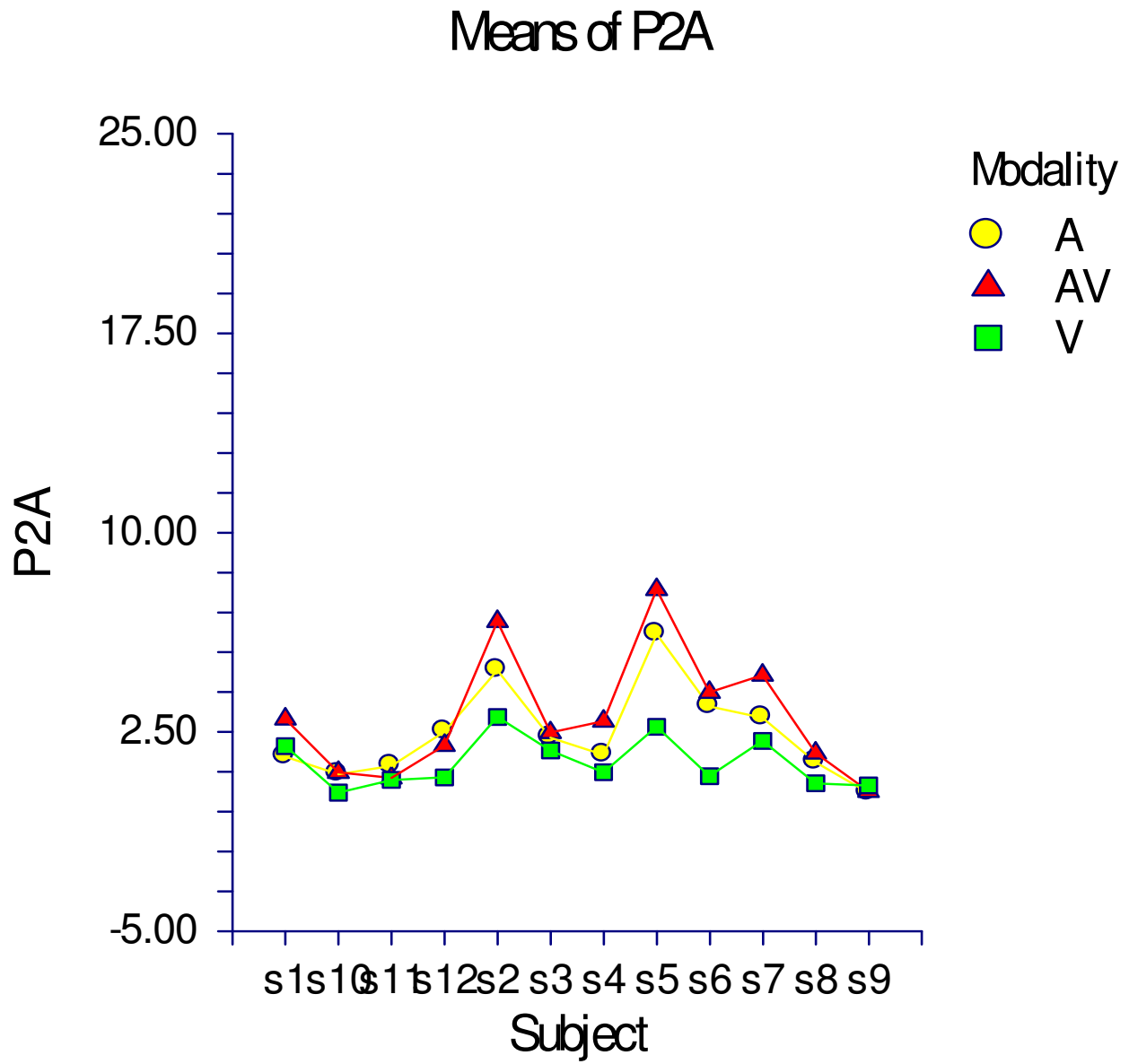


Figure 8: P2 amplitude mean values for each subject across stimulus conditions.

Table 1: Grand Averages

| Channel | N1 (Auditory) | | N1 (Aud.-Vis.) | | N1 (Visual) | | P2 (Auditory) | | P2 (Aud.-Vis.) | | P2 (Visual) | |
|---------|-----------------------|-----------------------|----------------|------|-------------|------|---------------|------|----------------|------|-------------|------|
| | Latency (<i>ms</i>) | Amplitude (μV) | Lat. | Amp. | Lat. | Amp. | Lat. | Amp. | Lat. | Amp. | Lat. | Amp. |
| FZ | 142 | -2.2 | 201 | 1.0 | 129 | -2.4 | 196 | 2.1 | 127 | -0.5 | 198 | 1.3 |
| FC5 | 151 | -2.1 | 152 | 1.9 | 122 | -0.2 | 211 | 0.8 | 199 | 1.3 | 191 | 1.2 |
| FC6 | 162 | -1.5 | 162 | -1.0 | 123 | -0.4 | 229 | 1.2 | 204 | 0.9 | 194 | 0.7 |
| FCZ | 132 | -4.5 | 211 | 3.0 | 132 | -4.9 | 205 | 4.6 | 162 | -0.9 | 199 | 0.6 |
| C5 | 145 | -2.2 | 219 | 1.0 | 123 | -0.9 | 207 | 0.7 | 160 | -0.2 | 182 | 0.1 |
| C6 | 166 | -1.4 | 229 | 2.3 | 162 | -1.3 | 131 | -0.9 | 117 | -0.4 | 141 | 0.0 |
| CZ | 132 | -5.0 | 217 | 4.3 | 131 | -5.4 | 211 | 5.7 | 163 | -1.0 | 206 | 0.4 |
| P5 | 70 | -0.4 | 67 | -0.5 | 72 | -0.1 | 137 | 1.5 | 129 | 1.8 | 122 | 0.8 |
| P6 | 71 | -0.3 | 66 | -0.4 | 27 | -0.4 | 130 | 1.9 | 124 | 1.7 | 79 | 0.4 |
| PZ | 124 | -1.4 | 173 | 1.9 | 115 | -1.5 | 238 | 3.6 | 189 | -0.6 | 245 | 1.0 |
| OZ | 70 | -0.5 | 67 | -0.7 | 84 | -0.6 | 129 | 2.4 | 124 | 3.1 | 147 | 1.3 |

CHAPTER IV

RESULTS

The N1 and P2 peaks were detected for each of the subjects in all conditions. The latency of the N1 peak had a mean value of 121.55 *ms* in the auditory alone condition. For the visual alone condition it had a mean value of 108.67 *ms*, and for the combined auditory-visual condition it had a mean value of 118.97 *ms*. Statistical analysis revealed there was no main effect for the modality of experimental condition [$F(2, 395) = 3.35, p = 0.054$]. This is consistent with other research findings that have shown little to no effect in the latency of the auditory and visual N1 peaks across experiment conditions (Michalewski, Prasher, & Starr, 1986; Pérez, Meyer, & Harrison, 2008; Gilley, Sharma, Dorman, & Martin, 2005).

N1 latency

For the N1 latency ANOVA there was a main effect for electrode [$F(10, 395) = 15.69, p < 0.000$], and a main effect for the electrode-modality interaction [$F(20, 395) = 1.89, p = 0.015$]. The main effect for electrode is most likely due to the large number of electrodes being analyzed. An earlier ANOVA was run using only 6 electrodes and this effect was not seen for the N1 latency. With a greater amount of electrodes analyzed the possibility of seeing a latency effect increases due to variability of latency across hemispheres and between anterior and posterior channels. Thus, this main effect must be considered with caution.

The main effect for electrode-modality interaction may be due to the significant electrode main effect and also must be interpreted with caution.

Trends for N1 latency showed latency onsets of 114 *ms* or more for all channels except the OZ, P5, and P6 channels in the auditory condition, which had latency times of 90 *ms*, 93.5 *ms*, and 87.9 *ms* respectively. Latency onsets were in the 80-120 *ms* range for the visual condition with the P5 channel having the earliest onset time of 80 *ms*. There was a noticeable difference between onset times for the P5 (80 *ms*) and P6 (106.67 *ms*) channels in the V condition, suggesting an hemispheric interaction of the stimulus. For the auditory-visual condition (AV) latency onset times were longest for the medial and frontal channels with the posterior channels—OZ, P5, and P6—having the shortest onset time. The latency for the AV condition in the OZ, P6, and PZ channels was earlier than for the A or V conditions on those same channels. There was a lateral effect for all bi-hemisphere channels. Notably, there was an increase in latency from the C5 and C6 channels for both the A and V conditions, but a decrease in latency for the AV condition. It was also noted that in the FC5 and FC6 channels the A and AV conditions show large differences in latency times across the 2 channels, but the V condition does not have this same effect. Please refer to Figure 9 for a graph of the results for each electrode.

P2 latency

Latencies for the P2 peak did show some statistically significant results. There was a main effect for modality [$F(2, 395) = 7.46, p = 0.003$] as well as for electrode [$F(10, 395) = 11.66, p < 0.000$] and the modality-electrode interaction [$F(20, 395) = 1.65, p = 0.043$]. Trends for P2 latency in the AV condition showed an earlier onset than for the A condition for all channels except the C5, FC6 and FZ channels. The AV condition had an earlier onset time from the V condition for the OZ, P5, P6 and PZ channels, making these 4 channels the

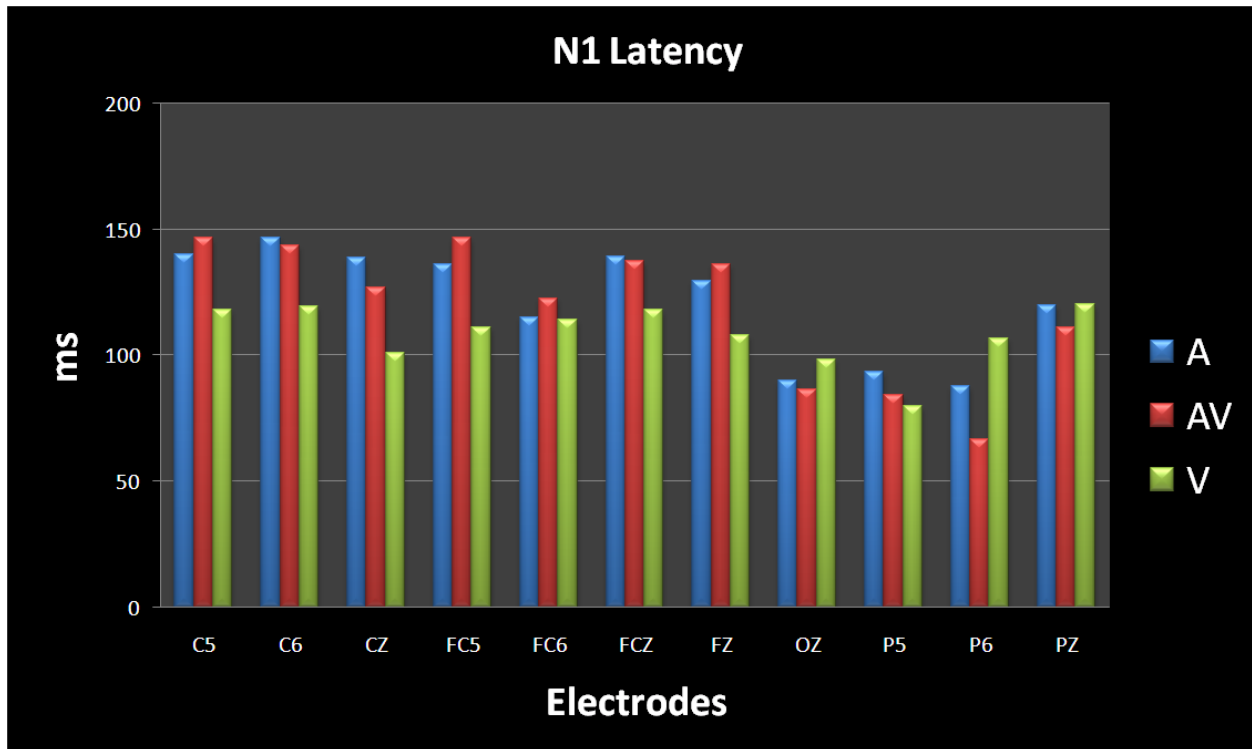


Figure 9: N1 latency values at each electrode for each stimulus condition.

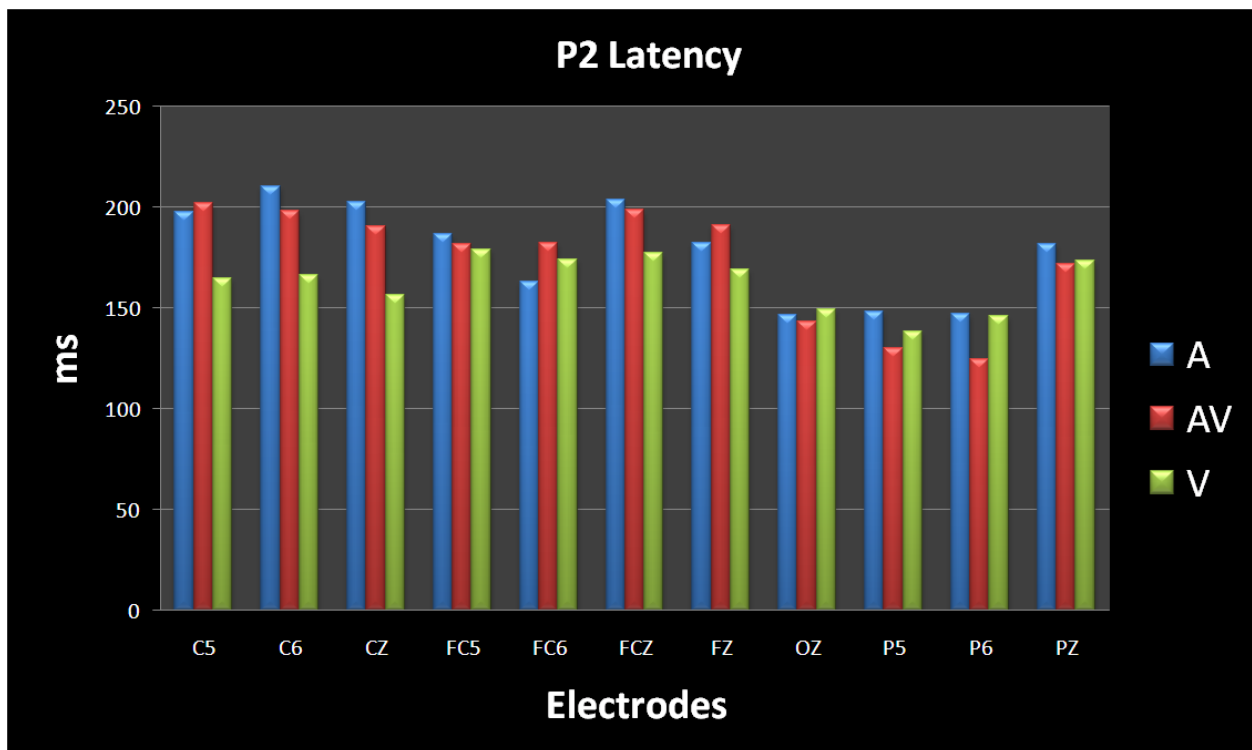


Figure 10: P2 latency values at each electrode for each stimulus condition.

only ones to demonstrate a modality effect in the latency of the P2 peak for this experiment. Again, as with the N1 latencies, the posterior channels showed the greatest effect for the AV condition. The FC5 and FC6 channel interactions noted in the N1 latency data was also noted for the P2 latencies. There was also a difference in the latency times between the C5 and C6 channels for the A condition that was lightly seen in the AV condition and not seen in the V condition. Please refer to Figure 10 for a graph of the results for each electrode.

N1 amplitude

Analysis of the amplitude of the N1 peaks also showed a main effect for modality [$F(2, 395) = 24.75, p < 0.000002$], for electrode [$F(10, 395) = 10.06, p < 0.000$], and for the electrode-modality interaction [$F(20, 395) = 8.10, p < 0.000$]. Amplitudes for the AV condition in the C5, C6, CZ, FC6, FCZ, FZ, OZ, and P6 channels was higher than for the A condition. Amplitudes for the AV condition were higher for all channels except the P6 channel when compared to the V condition. The anterior channels appeared to show the strongest effect for increase in amplitude for the AV condition. There was a notable difference between the C5 and C6 channels and the FC5 and FC6 channels for both the A and AV conditions with the AV condition have a higher amplitude as compared to the A condition in the right hemisphere channels. There was also a much greater amplitude shift across hemispheres in the C5 and C6 channels than there was in FC5 and FC6 channels. Please refer to Figure 11 for a graph of the results for each electrode.

P2 amplitude

The largest significant results of analysis were seen in the results for the amplitude of the P2 peaks. Main effects for the P2 amplitude were seen in modality [$F(2, 395) = 12.40,$

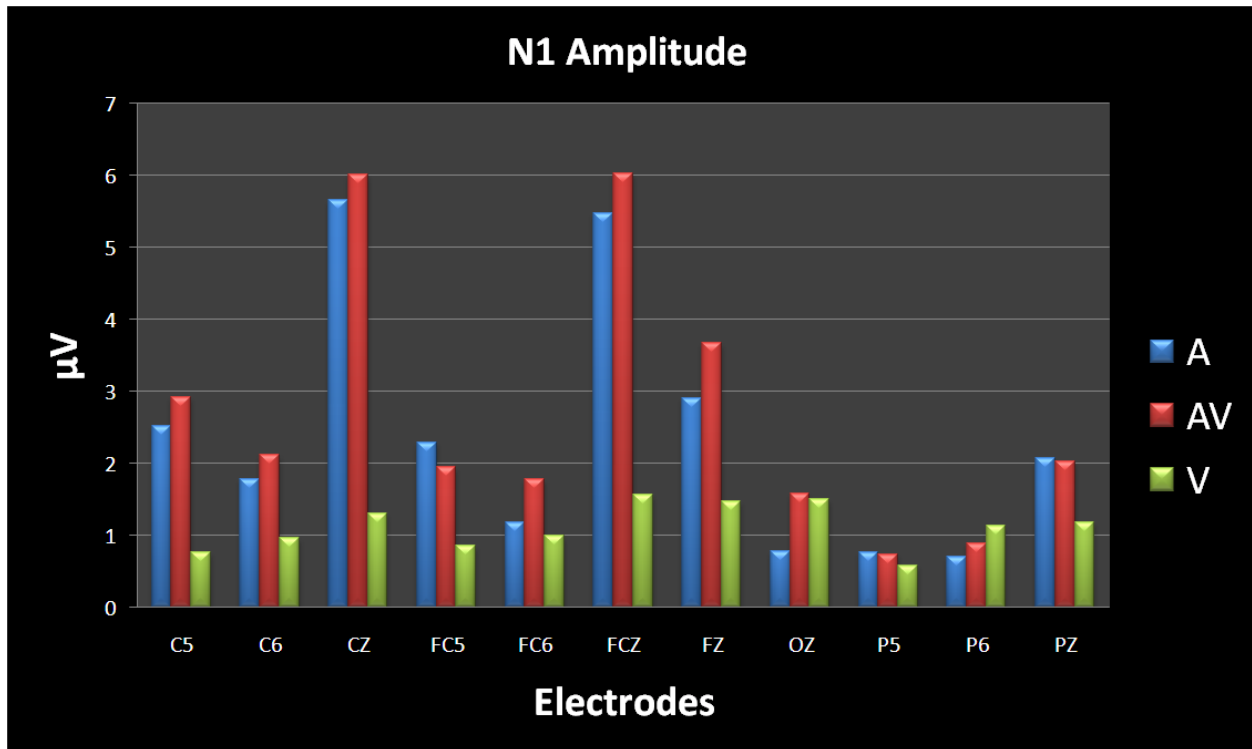


Figure 11: N1 amplitude values at each electrode for each stimulus condition.

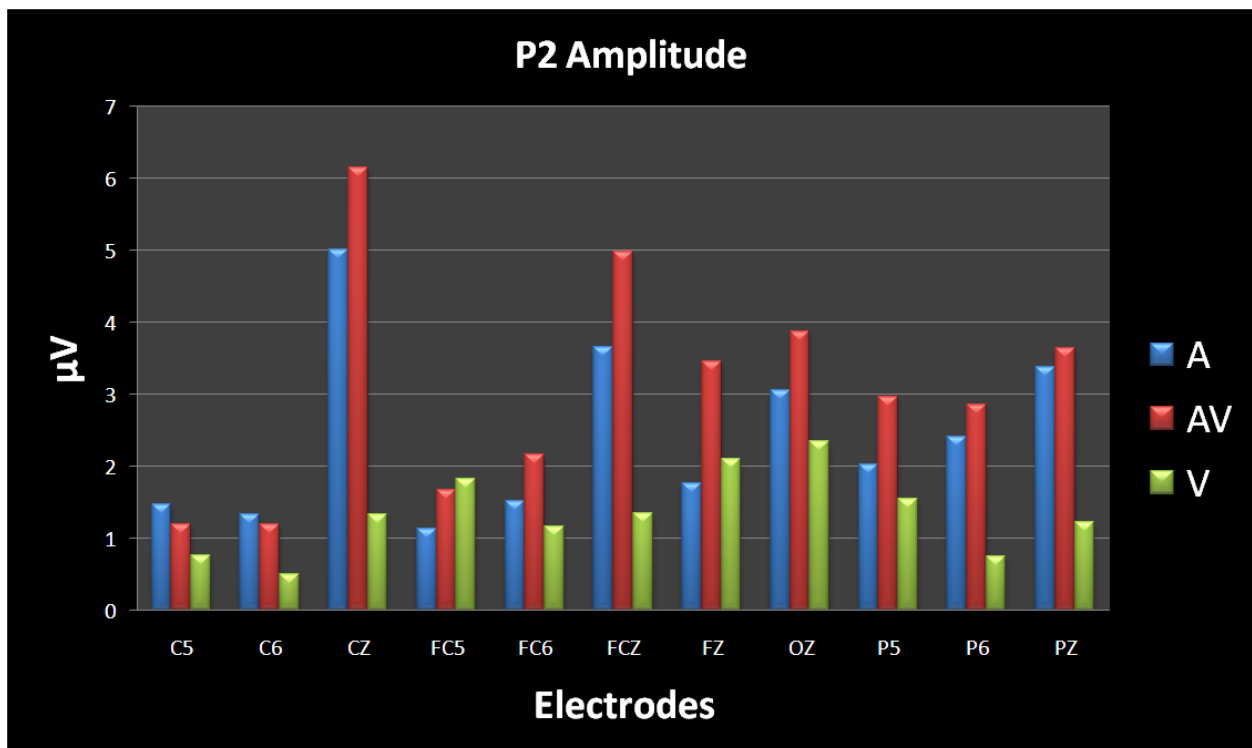


Figure 12: P2 amplitude values at each electrode for each stimulus condition.

$p = 0.000248$], electrode [$F(10, 395) = 3.98$, $p = 0.000119$], and for electrode-modality interaction [$F(20, 395) = 3.34$, $p < 0.000006$]. The amplitude effect was greatest across all midline channels from anterior to posterior for the AV conditions. The CZ, FCZ, and FZ channels showed the largest increase in amplitude for the AV condition. There was an effect for the posterior hemispheric channels—the P5 and P6, and for the FC6 channel. Amplitude for the AV condition was higher for all channels except the C5 and C6 channels than for the A condition and for all channels except FC5 for the V condition. The difference in amplitude across the medial channels for the AV condition was between $1.2 \mu V$ and $6.1 \mu V$. That is significant considering the difference in amplitude between the medial channels for the A and V conditions was between $1.1 \mu V$ and $5 \mu V$ and $0.5 \mu V$ and $2.3 \mu V$ respectively. Please refer to Figure 12 for a graph of the results for each electrode.

CHAPTER V

DISCUSSION

Results from this study imply that there is indeed a modality effect on the amplitude of both the auditory and visual P2 peaks. There is also an effect on the amplitude of the N1 peak and the latency of the P2 peak, but these effects are not as robust as that for the amplitude of the P2 peak. There were interesting results seen across the anterior and posterior channels and between hemispheric channels for all stimulus conditions. While some of these results can be explained by electrode location on the scalp, there are several that cannot. Explanations for these differences must include at least a consideration of the effect of multiple sensory inputs on the entire system and not just unimodal sensory areas. These combined effects indicate that multiple sensory stimuli are in some way impacting sensory processing.

N1 latency

While there were no significant results for changes in latency of the N1 peak due to stimulus modality, there were some effects seen that are worth discussing. First, there was a latency effect seen in the P6 and PZ channels for the AV condition. This fact is particularly interesting when one considers the regions over which these channels are placed include the parietal lobe. The parietal lobe sits next to the occipital lobe, which is responsible

for processing visual stimuli. Thus, a visual stimulus should have a greater impact on these channels than on channels closer to the medial or anterior portions of the scalp, such as the C5, C6 or the FC5, FC6. It is interesting that the combined stimulus condition demonstrated earlier latencies than the visual alone condition did in areas considered to be visually dominant.

Two other interesting electrode effects was seen in the OZ channel. The OZ channel is placed directly over the occipital lobe and best records the visual evoked potentials. Because the OZ channel is most responsive to visual stimuli, it would be expected that the latency times for this channel would be earlier than for almost all other channels, but this is not the case. The P5 channel, in fact, has an earlier latency time than the OZ channel for both the V and AV conditions (See Figure 9). The P5 channel having an earlier latency than the OZ channel in the visual alone and combined audio-visual condition is unexpected and lends itself to further questioning.

The second effect seen is the AV condition having an earlier latency time than either the auditory or visual conditions. In addition, responses in the OZ channel (See Figure 9) reveal that the auditory stimulus has an earlier latency onset time than the visual stimulus. In part the earlier auditory latency may be due to the fact that the auditory N1 occurs slightly earlier than the visual N1, and this may be influencing the AV effect seen in the OZ channel. Earlier research has shown an auditory dominance in multisensory stimuli presentation (Budinger, Heil, Hess, & Scheich, 2006). Still, the extent to which the auditory stimulus is affecting the latency of the combined auditory-visual condition is not known, and it is interesting that the combined AV effect is seen clearly in a visually dominant channel. Given the design and execution of this particular experiment, there are limitations to assessing why latencies in visually dominant channels were so greatly influenced by a multimodal sensory stimulus. However, it is safe to suggest that the N1 latency results of the PZ, P6, and OZ channels in the AV experiment condition are indicative of a multisensory processing effect.

A third effect seen in the N1 latency data is from the FC5 and FC6 channels. The data

for these two electrodes shows a decrease in latency for the A and AV conditions from the left hemisphere, the FC5 channel, to the right hemisphere, the FC6 channel. Interestingly, though, there is an increase in latency for the V condition from FC5 to FC6. Overall, the latency times for the AV condition are greater than both the A or V conditions in both channels. It is also interesting to note that there is a large difference between stimulus conditions in the FC5 channel, but the latency times in the FC6 channel are grouped much closer together. Within the parameters of this experiment it is not readily apparent any of the effects occur. It is possible that simultaneous stimulation is causing responses to occur slower across hemispheres or affecting one hemisphere more than another. It is also possible that attention and pattern recognition responses are affecting the latency onset times and causing the differences between hemispheres. Obviously there is some effect due to the multisensory condition, but the underlying mechanisms that drive such an effect remain unclear.

Although there was no significant effect seen for the N1 latency results as a whole, effects seen in certain individual channels for the N1 latency data appear to be significant and provide questions for future research. These questions of how electrode placement affects N1 latency results, why auditory stimulation affects latency more so than visual stimulation in a visual channels, why N1 latency differs across the FC5 and FC6 channels, and why the greatest latency effect is seen in the P6 channels would be interesting to answer in the pursuit of better knowledge of sensory processing and cortical interactions. For this project, N1 latencies do not clearly indicate a sensory integration effect, but they do indicate that something is occurring in the multimodal sensory condition that warrants further inquiry.

P2 latency

The results from the P2 latency data were significant as well, and there did appear to be a trend of earlier onset of the P2 peak in the AV condition. However, the latency onset was not largely affected across all channels, suggesting that the effect seen may be partially due to the significant increase in amplitude of the P2 peak for the AV condition. The largest change in latency for the AV condition was seen in the P5, P6, PZ, and OZ channels, just as it was in the N1 latency. This finding for the P2 latency is just as interesting as the P6, PZ and OZ findings for the N1 latency, and raises the same questions “why is there such a large decrease in latency for these channels, particularly when they are linked more closely to the visual cortex and the P2 peak is more robust in auditory stimuli?” Clearly, the auditory part of the combined stimulus is affecting the outcomes for the OZ channel, but the earlier latency times seen in all these channels are much earlier than even the auditory channels, suggesting a cumulative effect from the multimodal stimulus. One reason for this trend in the latency of the P2 peak may be that the P5 channel sits closely to the angular gyrus, which is a well-known area for multisensory processing (Matsushashi, Ikeda, Ohara, Matsumoto, Yamamoto, Takayama, Satow, Begum, Usui, Nagamine, Mikuni, Takahashi, Miyamoto, Fukuyama, & Shibasaki, 2004). However, the effect is seen strongest in the P6 channel, which is not necessarily near the angular gyrus. This latency effect is indeed interesting and should be further explored to determine what it means for processing multiple sensory inputs.

N1 amplitude

The effects for the N1 amplitude were significant and do indicate that the author’s hypothesis is at least partially valid. The amplitude effects for the AV condition were seen in 7 out the 11 electrodes sampled, indicating it was a consistent, valid effect. It is also interesting to note that the amplitude effect was greatest for the anterior and medial channels and was much smaller or not seen at all in posterior channels. The OZ channel

data for the N1 amplitude is perhaps the strongest indication of multisensory processing in the N1 amplitude. As mentioned earlier, the OZ channel is a visual channel and the visual N1 is stronger than the auditory N1. Yet, the amplitude for the auditory-visual condition in the OZ channel is higher than either the auditory or visual alone modalities. This is good evidence that the multisensory stimulus was combined and processed as an integrated stimulus and increased the amplitude of the N1 peak.

When considering the implications of the N1 amplitude effects, one must also consider the effects of attention. As mentioned above, there have been multiple studies that have shown an increase in the N1 amplitude in response to an attentional task. It is possible that the attention demands of the task affected the amplitude of the N1 peak. Consider, though, that the task was a detection task and the stimuli were randomized. The subjects would have no way of knowing which type of stimulus was going to come next, therefore, such attention related mechanisms should affect the peaks similarly across all three stimulus modalities. As it were, the main effect is for the AV modality and is not seen in the auditory or visual modalities, suggesting that the increase in amplitude is more likely due to presence of multiple sensory stimuli than to an attentional effect.

P2 amplitude

The results from the P2 amplitude data are the single greatest validation of the author's hypotheses. The multimodal stimulus condition revealed increased amplitude of the P2 peak more than either the auditory or visual stimuli in 8 out of 11 channels. For the C5 and C6 channels the AV condition produced amplitudes greater than the visual condition and in the FC5 channel the AV condition had higher amplitude than the auditory condition. The effects seen are large and robust and are clearly indicative of a multimodal sensory effect on sensory processing. Given the P2 peak ERPs are consistent across sensory modalities,

varying little in peak characteristics, the findings of this study are even more important as an indication that multisensory processing is occurring at earlier cortical levels and they are being processed as an integrated signal, rather than individual signals.

Most interestingly, the findings from this project raise the question of whether the auditory and visual P2 peak should be considered exogenous or endogenous peaks. While the experimental task for this project included a behavioral response, which by its nature makes these peaks endogenous, the difference in amplitude seen across the sensory modalities would suggest that the sensory stimulus, more so than the behavioral response, is affecting peak production. This would point to the P2 peak being more exogenous in nature. This leads to questions of whether or not the P2 peak is perhaps both exogenous and endogenous in nature, detecting stimuli in the environment and using it to formulate responses to stimuli. These questions are beyond the scope of this study, but the results of this study provide valuable information as to how to pursue these questions.

Future research

Several questions for future research have already been outlined in the above sections. Looking more closely at the interaction between electrodes during sensory activation could provide information about how the sensory system detects and encodes multiple sensory inputs. It is also worth exploring the audiovisual effects seen in the more visually dominant channels to determine why those channels detected activity differently than other channels. Of course, research to answer these questions would advance our knowledge of the sensory system as a whole while also providing knowledge of how the various sensory cortices interact with each other and the association cortices. In that pursuit, it would also be valuable to perform this study again in a population with sensory deficits in order to compare the results of those who are typical with those who are not. That information could render

invaluable insight into which cortical regions or functional connections are affected when sensory deprivation and deficits occur. This would help in understanding why we see the symptoms and behaviors seen in those with sensory deficits and processing disorder, which would, in turn, help in knowing what to target when developing intervention strategies and implementing them. Interesting research has been done in animal models studying the effects of environmental and internal conditions on the ERPs of the sensory system. That research has led to the discovery of possible answers to disorders in the sensory system. With results from studies like the present one, researchers can begin to look at completing similar studies in different populations.

CHAPTER VI

CONCLUSION

Much research has been done to determine how the human sensory system works and how the various components of the sensory system interact with each other. Over the past several decades many valuable lessons have been learned and insights have been gained. Still, there is much to learn in order to more completely understand the complex system we use to perceive and understand our world. One of the areas we still know relatively little about is multisensory processing. When considering how much of our everyday lives are affected and dependent upon our ability to detect and process multiple sensory inputs efficiently and correctly, it is clear how important research in this area is. The benefits of understanding our sensory system better, and understanding how to address disorders in the system, are numerous. There are benefits to quality of life, our knowledge of the world, and clinical implications for interventions.

One way to pursue the unanswered questions in multisensory processing is to examine the neurology of the sensory system while it is in use, presenting stimuli to subjects and seeing how they respond. Using EEG to study the event related potentials that stimulus presentation and reactions elicit, can provide information about the sensory system and how it responds to the environment. This information can help researchers design better questions to answer, thus improving the results of their research.

Research using EEG to audit the effects of multiple sensory stimuli has focused largely on the auditory, visual, and somatosensory peaks that occur prior to the P2. For this

study, one of the more well-studied peaks in sensory processing, the N1, as well as a less-studied peak, the P2, were used. This allowed for the study to be well grounded in prior research while exploring new hypotheses. Because this study focused on both the N1 and P2 peaks independently, it adds unique information to the body of knowledge on multisensory processing.

This study proposed that the latency and amplitude of the auditory and visual N1 and P2 peaks would be affected by the processing of combined multiple sensory inputs. It was further proposed that effects on the latency and amplitude of the N1 and P2 peaks would suggest that multimodal sensory inputs are integrated to some extent in unimodal sensory cortices. This hypothesis is significant because the N1 and P2 peaks are considered early cortical peaks, generated in primary sensory cortices, seen in all sensory modalities when a single sensory stimulus is presented. Given the N1 and P2 peaks are considered earlier, sensory specific, event related potentials, findings of effects on the characteristics of the two peaks that are directly related to combined sensory inputs would suggest integration and processing of multimodal sensory stimuli at early cortical stages.

For this study, EEG was employed to gather information about the neurological activity produced when subjects were asked to respond to either a single sensory stimulus or a combined sensory stimulus. The event related potentials generated by the subjects in response to the sensory stimulation they received provided information as to whether or not a combined sensory stimulus affects the early stages of sensory processing, previously thought to be affected only by unimodal sensory stimulation. Results from this study demonstrated that a combined sensory stimulus impacted the amplitude of the auditory and visual P2 peak, with smaller effects seen on the latency of the P2 peak and the amplitude of the auditory and visual N1 peak. While there was no significant effect seen in the overall latency results of the N1 peak, there were significant results seen in the latency results for posterior electrodes that indicated a clear effect of a combined stimulus on sensory processing in a unimodal sensory cortex. These significant results should be further studied in order to gain

more insight into the effects of multiple sensory inputs on the human sensory system.

BIBLIOGRAPHY

- Amedi, A., Raz, N., Azulay, H., Malach, R., & Zohary, E. (2010). Cortical activity during tactile exploration of objects in blind and sighted humans. *Restorative Neurology and Neuroscience*, *28*(2), 143 – 156.
- Anderer, P., Semlitsch, H. V., & Saletu, B. (1996). Multichannel auditory event-related brain potentials: effects of normal aging on the scalp distribution of n1, p2, n2 and p300 latencies and amplitudes. *Electroencephalography and Clinical Neurophysiology*, *99*(5), 458 – 472.
- Anokhin, A. P., Vedeniapin, A. B., Heath, A. C., Korzyukov, O., & Boutros, N. N. (2007). Genetic and environmental influences on sensory gating of mid-latency auditory evoked responses: A twin study. *Schizophrenia Research*, *89*(1-3), 312 – 319.
- Bergeson, T. R., Houston, D. M., & Miyamoto, R. T. (2010). Effects of congenital hearing loss and cochlear implantation on audiovisual speech perception in infants and children. *Restorative Neurology and Neuroscience*, *28*(2), 157 – 165.
- Bottari, D., Nava, E., Ley, P., & Pavani, F. (2010). Enhanced reactivity to visual stimuli in deaf individuals. *Restorative Neurology and Neuroscience*, *28*(2), 167 – 179.
- Budinger, E., Heil, P., Hess, A., & Scheich, H. (2006). Multisensory processing via early cortical stages: Connections of the primary auditory cortical field with other sensory systems. *Neuroscience*, *143*(4), 1065 – 1083.
- Burgess, A. & Gruzelier, J. (1993). Individual reliability of amplitude distribution in topographical mapping of eeg. *Electroencephalography and Clinical Neurophysiology*, *86*(4), 219 – 223.
- Čeponienė, R., Torki, M., Alku, P., Koyama, A., & Townsend, J. (2008). Event-related potentials reflect spectral differences in speech and non-speech stimuli in children and adults. *Clinical Neurophysiology*, *119*(7), 1560 – 1577.
- Crowley, K. E. & Colrain, I. M. (2004). A review of the evidence for p2 being an independent component process: age, sleep and modality. *Clinical Neurophysiology*, *115*(4), 732 – 744.

- Curran, E. A. & Stokes, M. J. (2003). Learning to control brain activity: A review of the production and control of eeg components for driving brain-computer interface (bci) systems. *Brain and Cognition*, 51(3), 326 – 336.
- Dye, M. W. G. & Bavelier, D. (2010). Attentional enhancements and deficits in deaf populations: an integrative review. *Restorative Neurology and Neuroscience*, 28(2), 181 – 192.
- Fiehler, K. & Rösler, F. (2010). Plasticity of multisensory dorsal stream functions: Evidence from congenitally blind and sighted adults. *Restorative Neurology and Neuroscience*, 28(2), 193 – 205.
- Fingelkurts, A. A., Fingelkurts, A. A., & Kähkönen, S. (2005). Functional connectivity in the brain—is it an elusive concept? *Neuroscience & Biobehavioral Reviews*, 28(8), 827 – 836.
- Gilley, P. M., Sharma, A., Dorman, M., Finley, C. C., Panch, A. S., & Martin, K. (2006). Minimization of cochlear implant stimulus artifact in cortical auditory evoked potentials. *Clinical Neurophysiology*, 117(8), 1772 – 1782.
- Gilley, P. M., Sharma, A., Dorman, M., & Martin, K. (2005). Developmental changes in refractoriness of the cortical auditory evoked potential. *Clinical Neurophysiology*, 116(3), 648 – 657.
- Gilley, P. M., Sharma, A., & Dorman, M. F. (2008). Cortical reorganization in children with cochlear implants. *Brain Research*, 1239, 56 – 65.
- Gilley, P. M., Sharma, A., Mitchell, T. V., & Dorman, M. F. (2010). The influence of a sensitive period for auditory-visual integration in children with cochlear implants. *Restorative Neurology and Neuroscience*, 28(2), 207 – 218.
- Grossmann, T. (2010). The development of emotion perception in face and voice during infancy. *Restorative Neurology and Neuroscience*, 28(2), 219 – 236.
- Gudmundsson, S., Runarsson, T. P., Sigurdsson, S., Eiriksdottir, G., & Johnsen, K. (2007). Reliability of quantitative eeg features. *Clinical Neurophysiology*, 118(10), 2162 – 2171.
- Hall, J. W. (2006). *New Handbook for Auditory Evoked Responses*. Upper Saddle River: Allyn & Bacon, 1st edition.
- Hanganu-Opatz, I. L. (2010). Between molecules and experience: Role of early patterns of coordinated activity for the development of cortical maps and sensory abilities. *Brain Research Reviews*, 64(1), 160 – 176.
- Kreitewolf, J., Lewald, J., & Getzmann, S. (2011). Effect of attention on cortical processing of sound motion: An eeg study. *NeuroImage*, 54(3), 2340 – 2349.

- Martin, T., Huxlin, K. R., & Kavcic, V. (2010). Motion-onset visual evoked potentials predict performance during a global direction discrimination task. *Neuropsychologia*, *48*(12), 3563 – 3572.
- Matsushashi, M., Ikeda, A., Ohara, S., Matsumoto, R., Yamamoto, J., Takayama, M., Satow, T., Begum, T., Usui, K., Nagamine, T., Mikuni, N., Takahashi, J., Miyamoto, S., Fukuyama, H., & Shibasaki, H. (2004). Multisensory convergence at human temporo-parietal junction - epicortical recording of evoked responses. *Clinical Neurophysiology*, *115*(5), 1145–1160.
- McEvoy, L. K., Smith, M. E., & Gevins, A. (2000). Test-retest reliability of cognitive eeg. *Clinical Neurophysiology*, *111*(3), 457 – 463.
- Michalewski, H. J., Prasher, D. K., & Starr, A. (1986). Latency variability and temporal interrelationships of the auditory event-related potentials (n1, p2, n2, and p3) in normal subjects. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, *65*(1), 59 – 71.
- Miller, J. (1982). Divided attention: Evidence for coactivation with redundant signals. *Cognitive Psychology*, *14*(2), 247 – 279.
- Molholm, S., Ritter, W., Murray, M. M., Javitt, D. C., Schroeder, C. E., & Foxe, J. J. (2002). Multisensory auditory-visual interactions during early sensory processing in humans: a high-density electrical mapping study. *Cognitive Brain Research*, *14*(1), 115 – 128.
- Murray, M. M., Foxe, J. J., Higgins, B. A., Javitt, D. C., & Schroeder, C. E. (2001). Visuo-spatial neural response interactions in early cortical processing during a simple reaction time task: a high-density electrical mapping study. *Neuropsychologia*, *39*(8), 828 – 844.
- Murray, M. M., Molholm, S., Michel, C. M., Heslenfeld, D. J., Ritter, W., Javitt, D. C., Schroeder, C. E., & Foxe, J. J. (2005). Grabbing your ear: Rapid auditory somatosensory multisensory interactions in low-level sensory cortices are not constrained by stimulus alignment. *Cerebral Cortex*, *15*(7), 963 – 974.
- Näätänen, R., Gaillard, A. W. K., & Mäntysalo, S. (1978). Early selective-attention effect on evoked potential reinterpreted. *Acta Psychologica*, *42*(4), 313 – 329.
- Näätänen, R. & Picton, T. (1987). The n1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology*, *24*(4), 375 – 425.
- Odom, J. V., Bach, M., Brigell, M., Holder, G. E., McCulloch, D. L., Tormene, A. P., & Vaegan (2010). Iscev standard for clinical visual evoked potentials (2009 update). *Doc Ophthalmol*, *120*(1), 111 – 9.
- Pérez, E., Meyer, G., & Harrison, N. (2008). Neural correlates of attending speech and non-speech: Erps associated with duplex perception. *Journal of Neurolinguistics*, *21*(5), 452 – 471.

- Peterson, N. R., Pisoni, D. B., & Miyamoto, R. T. (2010). Cochlear implants and spoken language processing abilities: Review and assessment of the literature. *Restorative Neurology and Neuroscience*, *28*(2), 237 – 250.
- Purves, D., Augustine, G. J., Fitzpatrick, D., Hall, W. C., LaMantia, A.-S., McNamara, J. O., & White, L. E. (2008). *Neuroscience*. Sunderland: Sinauer Associates, Inc., 4th edition.
- Putzar, L., Hötting, K., & Röder, B. (2010). Early visual deprivation affects the development of face recognition and of audio-visual speech perception. *Restorative Neurology and Neuroscience*, *28*(2), 251 – 257.
- Raab, D. H. (1962). Statistical facilitation of simple reaction times. *Transactions of the New York Academy of Sciences*, *24*(5), 574 – 590.
- Röder, B. & Wallace, M. (2010). Development and plasticity of multisensory functions. *Restorative Neurology and Neuroscience*, *28*(2), 141 – 142.
- Royal, D. W., Krueger, J., Fister, M. C., & Wallace, M. T. (2010). Adult plasticity of spatiotemporal receptive fields of multisensory superior colliculus neurons following early visual deprivation. *Restorative Neurology and Neuroscience*, *28*(2), 259 – 270.
- Schroeder, C. E. & Foxe, J. J. (2002). The timing and laminar profile of converging inputs to multisensory areas of the macaque neocortex. *Cognitive Brain Research*, *14*(1), 187 – 198.
- Schroeder, C. E., Smiley, J., Fu, K. G., McGinnis, T., O'Connell, M. N., & Hackett, T. A. (2003). Anatomical mechanisms and functional implications of multisensory convergence in early cortical processing. *International Journal of Psychophysiology*, *50*(1-2), 5 – 17. Current findings in multisensory research.
- Spence, C. & Driver, J. (2004). *Crossmodal Space and Crossmodal Attention*. New York: Oxford: Oxford University Press, 1st edition.
- Tremblay, C., Champoux, F., Lepore, F., & Thoret, H. (2010). Audiovisual fusion and cochlear implant proficiency. *Restorative Neurology and Neuroscience*, *28*(2), 283 – 291.
- Ulrich, R., Miller, J., & Schröter, H. (2007). Testing the race model inequality: An algorithm and computer programs. *Behavior Research Methods*, *39*, 291 – 302.
- Valeriani, M., Pera, D. L., Restuccia, D., de Armas, L., Miliucci, R., Betti, V., Vigeveno, F., & Tonali, P. (2007). Parallel spinal pathways generate the middle-latency n1 and the late p2 components of the laser evoked potentials. *Clinical Neurophysiology*, *118*(5), 1097 – 1104.
- Wallace, M. (June 2004). The development of multisensory processes. *Cognitive Processing*, *5*(2), 69 – 83.

- Wallace, M. T., Carriere, B. N., Perrault, T. J., Vaughan, J. W., & Stein, B. E. (2006). The development of cortical multisensory integration. *The Journal of Neuroscience*, *26*(46), 11844 – 11849.
- Wallace, M. T., Ramachandran, R., & Stein, B. E. (2004). A revised view of sensory cortical parcellation. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(7), 2167 – 2172.