2012-01-01

Assessing Cortical Electrophysiologic and Behavioral Activity in Individuals with Aphasia and Participants with No Brain Damage Responding to Spoken Sentence Length Messages

Patricia Lara
University of Texas at El Paso, plara2@utep.edu

ASSESSING CORTICAL ELECTROPHYSIOLOGIC AND BEHAVIORAL ACTIVITY IN INDIVIDUALS WITH APHASIA AND PARTICIPANTS WITH NO BRAIN DAMAGE RESPONDING TO SPOKEN SENTENCE LENGTH MESSAGES

PATRICIA LARA

College of Health Sciences

APPROVED:

Anthony P. Salvatore, Ph.D., Chair

Samuel Riccillo, Ph.D.

Joseph Tomaka, Ph.D.

Benjamin C. Flores, Ph.D.
Interim Dean of the GraduateSchool

Dedication

This dissertation is dedicated to my parents, Ramon and Gloria Lara.

ASSESSING CORTICAL ELECTROPHYSIOLOGIC AND BEHAVIORAL ACTIVITY IN INDIVIDUALS WITH APHASIA AND PARTICIPANTS WITH NO BRAIN DAMAGE RESPONDING TO SPOKEN SENTENCE LENGTH MESSAGES

PATRICIA LARA, M.A. CCC-SLP

DISSERTATION

Presented to the Faculty of the GraduateSchool of
The University of Texas at El Paso
in Partial Fulfillment
of the Requirements
for the Degree of

DOCTOR OF PHILOSPHY

College of Health Sciences
THE UNIVERSITY OF TEXAS AT EL PASO
August 2012

Acknowledgements

Many people have assisted and supported me throughout the various stages of this dissertation experience. Dr. Anthony Salvatore, chair of my dissertation committee, challenged and guided me while being ever so patient through a multitude of questions, corrections and rewrites. For that, I wish to express my sincere appreciation. He provided insightful feedback and set me on the road to becoming a better researcher and writer.

Dr. Samuel Riccillo, the next best thing to my own father, encouraged me and made me laugh through this process. He provided me with support, guidance and friendship while instilling in me an understanding and love for ERP. I will be forever grateful to him for giving of his time and knowledge. The expertise, suggestions and encouragement of Dr. Joe Tomaka strengthened this study and contributed to its completion.

I want to express my deepest appreciation to my patients, their families and the members of the Stroke Support Group who not only inspired this dissertation but also agreed to help me complete my work by participating in this study. In addition, I want to thank all of my family and friends who supported and believed in me during the completion of this dissertation.

Finally, I want to say a special thank-you to Thomas for his patience during this stressful and busy time. Without his love, support, and encouragement, this dissertation would not have become a reality.

Patricia Lara

Abstract

In this study, six individuals with aphasia and twelve participants with no brain damage responded to spoken sentence length messages using a modified version of the Revised Token Test (McNeil & Prescott, 1978) while cortical activation was recorded using event related potentials (ERP). ERP is a non-invasive imaging procedure that measures cortical activation reflected in the electrical activity that is produced at the level of the cortex in response to internal or external stimulus. The electrical activity is measured through the skull with electrodes that are attached to a skullcap.

Participants were presented with a visual display that provided eight different token arrangements per trial that were displayed on the computer screen. Spoken messages that increased in length and grammatical complexity were presented via speakers. Participants were instructed to listen to the spoken message and respond by touching the visual choice that matched the spoken message on a touch screen for a total of 90 trials.

The task of semantic processing may be reflected in the latency and peak amplitude of the N400 ERP component. This component has been reported to reflect the processing of single spoken words in participants with no brain damage and individuals with aphasia (Friederici et al., 1999; Handy, 2005; Luck, 2005; Rugg& Coles, 1997). The N400 ERP component has not been well investigated in the processing of spoken sentence length messages in individuals with aphasia.

In order to investigate the mechanisms involved in the auditory comprehension of spoken sentence length messages, we need to identify where in that temporal process the individual is processing the message. Since ERP can be time locked to a specific event, it seems reasonable to use ERP to investigate the temporal characteristics of auditory comprehension of sentence length messages.

The present study compared the performance of individuals with aphasia and participants with no brain damage responding to spoken sentence length messages. The results

show that the two groups performed differently in regards to correct response rate. The individuals with aphasia made more error responses than did the participants with no brain damage. The participants with no brain damage showed reduced accuracy as the complexity of the sentence increased while the individuals with aphasia maintained the same performance throughout the test. No statistically significant difference was found between the individuals with aphasia and the participants with no brain damage for the mean latency and amplitude of the P300 and N400 components. However, upon visual examination of the ERP waveforms, the individuals with aphasia exhibited longer ERP latencies of the N400 and the P300 than the participants with no brain damage. In addition, the individuals with aphasia displayed slightly increased amplitudes for the P300 ERP component. Furthermore, the cortical activation patterns between the two groups were different. The non-brain damaged individuals displayed some degree of activation in the left frontal electrodes for the N400 component while the individuals with aphasia exhibited activation in the frontal and right temporal electrodes with some degree of activation exhibited in the left temporal electrodes. Furthermore, the individuals with aphasia exhibit high areas of activation throughout the cortex for the P300 suggesting that the individuals with aphasia were accessing all cortical areas available to them to compensate for damage to localized areas. The participants with no brain damage displayed activation in the frontal, right and left temporal electrodes. Cortical activation was also exhibited in the parietal electrodes consistent with the literature that suggests that the P300 is elicited primarily in the parietal electrodes.

The nature of activation also differed across the duration of each sentence in each of the two groups of participants. The individuals with aphasia showed reduced attentional activity at the start of each trial and an increased latency and reduced amplitude when processing the spoken message. The participants with no brain damage showed quicker attentional response to the onset of each trial and a shorter latency and increased amplitude when processing the sentence.

The implication of this study is that the individuals with aphasia processed spoken sentence length messages different from participants with no brain damage. Their attentional response was delayed and they required added processing time, but obtained a moderate degree of correct response rate.

Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction

The recovery of aphasia continues to be worthy of study since the functional processes involved are still not fully understood. The purpose of this quantitative study is to compare cortical electrophysiologic and behavioral activity in individuals with aphasia and participants with no brain damage responding to spoken sentence length messages. It is anticipated that the knowledge gained from this study will increase the information currently available regarding auditory comprehension of spoken sentence length messages. While a review of the literature shows that individuals with aphasia process auditory messages slower than non-brain damaged individuals, the temporal mechanisms that underlie these processes are still not clearly understood. Since auditory comprehension is the primary language modality used for the treatment of aphasia, knowing the temporal characteristics involved in auditory comprehension of spoken messages may provide for the development of more effective therapeutic strategies as well as accurate prognosis for recovery. Accurate prognosis for recovery from aphasia is important in order to provide for allocation of funding sources and candidacy for treatment thus improving quality of life issues for individuals for aphasia.

This chapter will begin with an overview of the background and content framing the study. Following the overview are the problem statement, statement of purpose, research questions, assumptions, and significance of the study.

BACKGROUND AND CONTENT

Aphasia is language disorder that affects all language modalities including speech, reading, writing and auditory comprehension (Darley, 1982). Aphasia is caused by damage to the language areas of the brain. In most people, this is the left hemisphere. While aphasia is caused by a variety of different etiologies, the most common is stroke (Benson &Ardilla, 1996). It is estimated that approximately one million people in the United States suffer from aphasia and that 25 to 40% of individuals who survive a stroke will acquire the disorder. This means that

approximately 100,000 Americans will acquire aphasiaeach year (National Aphasia Association, 2007, National Institute of Neurological Disorders and Stroke, 2008).

Although aphasia affects all language modalities, auditory comprehension is present in all aphasia types to varying degrees. A patient's ability to understand spoken language accurately is essential to determining the behavioral interventions that will be used in treatment. As a result, research has focused on finding an effective means of assessing and treating the disorder. The difficulty lies in that recovery is mediated by several factors. Neuroplasticity, the brain's ability to reorganize its structures and functions to create new pathways is hypothesized to be one of these factors. In addition, factors such as initial severity of the disorder, gender, age, behavioral aphasia treatment provided, size and location of the lesion are thought to influence neuroplasticity.

In the past, behavioral methodology has been used to measure recovery of aphasia and to predict recovery patterns. However, these methods do not provide correlation between behavioral improvements, anatomical structures and the temporal processes involved in recovery. Additionally, many patients with aphasia suffer from physical disabilities that greatly limit the efficacy of behavioral assessment instruments (D'Arcy, et.al., 2003).

Advances in neuroimaging technology have provided new avenues to measure recovery in aphasia. The most common neuroimaging options used in aphasia research are Functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), and Event Related Potentials (ERP) (Handy, 2005).

fMRI examines functional brain activity by measuring the oxygen levels in the red blood cells that are produced as a result of increased neural brain activity. This is known as the BOLD (blood oxygen level dependent) signal (Buxton, 2002).

PET examines functional brain activity by using radioactive tracer isotopes that are injected directly into the bloodstream. The tracer isotopes are observed by the tissue or organ of interest such as the brain and are then reconstructed into a three-dimensional picture using a computer (Bailey, 2005; Handy, 2005; Ingham, 2007).

ERP measure the functional activity at the level of the cortex reflected in the electrical activity that is produced in the brain in response to internal or external stimulus. The electrical activity is measured through the skull with electrodes that are attached to a skullcap.

While fMRI and PET are excellent tools for localization of anatomical structures, they do not provide information regarding the temporal processes that occur in response to stimuli. ERP on the other hand, provide real-time information in the millisecond range since it can be time-locked to a specific stimulus event. Cost and availability make the use of fMRI and PET prohibitive in clinical and research settings. Furthermore, fMRI and PET are considerably more invasive than ERP since they use radiation directly injected into the blood stream or from scanners to measure functional cortical activity. Low invasiveness, low cost, higher availability and high temporal resolution make ERP an excellent tool for the study of aphasia.

The ability to use and comprehend language is one of the most valuable tools that human beings possess. Auditory comprehension is the foundation for development of language and is the primary language modality used in the treatment of aphasia. As a result, it plays an important role in communication. An injury to the brain may result in loss or disruption of this ability since auditory comprehension problems are present in a large percentage of individuals suffering from aphasia regardless of the type (Brookshire, 1978; Liles & Brookshire, 1975). The impact that such a communication loss has on the patient and family is huge since lifestyle changes are sure to follow (Parr, 2007). In addition, the financial burdens that are placed on an already overtaxed medical system are severe.

The literature suggests that auditory comprehension problems in aphasia are related to delayed access to the meaning of words. Several ERP studies have reported increased latencies in individuals with aphasia in tasks of auditory comprehension. These increased latencies have been reported both in tasks of auditory comprehension of single words and sentences that use explicit semantic judgment responses (D'Arcy et. al., 2003; Friederici&Kilborn, 1989; TerKeurs et. al., 1999).

The aim of the present study is to compare the performance of individuals with aphasia and participants with no brain damage responding to spoken sentence length messages using ERP. In order to do this we needed to determine at what time point the individual is processing the stimulus. Since ERP can be tightly linked in time to a specific event, it is possible to make inferences about the temporal characteristics involved in the auditory comprehension of spoken sentence length messages. Therefore, it is the most useful imaging procedure for use in this study.

STATEMENT OF THE PROBLEM

Auditory comprehension is impaired to varying degrees in all aphasia types. Since auditory comprehension is the foundation for the development of language and is the primary language modality used in the treatment of aphasia, it is important to understand the mechanisms that underlie its recovery. Deficits in auditory comprehension in individuals with aphasia may be due to the delayed access to the meaning of words. Imaging study findings have shown that individuals with aphasia perform different from participants with no brain damage in tasks of auditory comprehension (Musso et. al., 1999; Warburton et. al., 1999). In addition, ERP studies have reported that individuals with aphasia exhibit delayed latencies of the N400 ERP component in tasks that examine auditory comprehension (Swaab, Brown &Hagoort, 1997). Furthermore, behavioral studies have shown that individuals with aphasia improve their performance on tasks of auditory comprehension when pauses are inserted within the message (Liles & Brookshire, 1975).

Auditory comprehension of spoken single words has been well studied. However, auditory comprehension of spoken sentence length messages have not been well examined. The temporal characteristics of spoken sentence length messages are still not clear. The purpose of the current study was to compare the cortical electrophysiologic latencies in individuals with aphasia and participants with no brain damage responding to spoken sentence length messages.

In order to investigate the temporal characteristics of auditory comprehension of spoken sentence length messages, we need to determine at what time point the individual processes the spoken message. This can be achieved by measuring ERPs. The task of semantic processing may be reflected in the N400 ERP component. The N400 ERP component has been associated with intact semantic ability. Since ERP can be time locked to a specific event, the N400 was used to determine the time point at which auditory comprehension of spoken sentence length messages occurred in individuals with aphasia and participants with no brain damage.

PURPOSE OF THE STUDY

The purpose of this study was to compare performance on behavioral reaction time (measured from the onset of the spoken message to the time when the participant touches their choice on the touch screen monitor) and correct response rate in individuals with aphasia and participants with no brain damage. Differences in peak latencies and amplitudes of the N400 and P300 ERP components in individuals with aphasia and participants with no brain damage were also compared. Differences in cortical activation patterns between the two groups were also investigated.

RESEARCH QUESTIONS

Several research questions are addressed: 1) What are the correct response rate differences between the individuals with aphasia and the participants with no brain damage? 2) What are the behavioral reaction time (deduced by the onset of the spoken message to the time the participant touched their visual choice) differences between the individuals with aphasia and the participants with no brain damage? 3) What are the cortical electrophysiologic differences (deduced from ERP wave patterns) between the individuals with aphasia and the participants with no brain damage? 4) What are the cortical activation differences (deduced from topographic maps) between the individuals with aphasia and the participants with no brain damage?

ASSUMPTIONS

1) Individuals with aphasia exhibit auditory comprehension deficits. 2) Semantic processing may be reflected in the N400 ERP component. 3) Individuals with aphasia perform differently than participants with no brain damage. 4) Individuals with aphasia exhibit delayed latencies in tasks of auditory comprehension.

SIGNIFICANCE OF THE STUDY

Aphasia is a language disorder that affects all language modalities. In addition to a reduction in speech, a reduction in auditory comprehension is also present in all aphasia types to varying degrees. This reduction in auditory comprehension is such a significant impairment that it can lead to quality of life issues such as social isolation, depression, financial hardships, loss of personal relationships and stigma on the victim and their families (Parr, 2007). Furthermore, it is estimated that 25 to 40% of individuals who survive a stroke will acquire aphasia. This means that approximately 100,000 Americans will acquire aphasia each year (National Aphasia Association, 2007, National Institute of Neurological Disorders and Stroke, 2008). As a result, the quality of life issues as well as the financial implications resulting from treating this population are huge. Better understanding of the processes that mediate the recovery of auditory comprehension may result in development of improved prognostic and treatment strategies.

A review of the literature suggests that ERP can be useful in the study of aphasia and the physiological changes that occur during the recovery process because it can be tightly linked to time. Auditory comprehension of single spoken words has been well examined in individuals with aphasia and non-brain damaged individuals using ERP. However, auditory comprehension of spoken sentence length messages has not received a lot of attention.

This study seeks to broaden the understanding of sentence processing using ERP. In order to understand the mechanisms involved in the semantic processing of spoken sentence length messages, we need to determine the nature of this process across time. Since ERP can be time locked to a specific event and monitors electrical activity at the cortical level over time, it is

reasonable to use this methodology to investigate the temporal changes in auditory comprehension of spoken messages. Furthermore, correct response rate and reaction time can also be measured.

Chapter 2: Review of Literature

The purpose of this quantitative study is to compare cortical electrophysiologic and behavioral activity in individuals with aphasia and participants with no brain damage responding to spoken sentence length messages. This chapter presents a review of literature as it pertains to; A) the nature of aphasia, B) aphasia classification, C) factors thought to affect the recovery of aphasia, D) neuroplasticity, E) imaging technology used in the study of aphasia, F) advantages and disadvantages of ERP, G) basics of ERP techniques, H) comprehension of spoken language, I) auditory comprehension in non-brain damaged individuals, J) auditory comprehension in aphasia, K) ERP studies of single word auditory comprehension, L) ERP studies of spoken sentence auditory comprehension.

APHASIA

Aphasia is a language disorder that affects all language modalities including speech, reading, writing and auditory comprehension. Individuals with aphasia may demonstrate decreased performance in tasks of intellectual functioning when compared to individuals with no brain damage suggesting that cognitive ability may also be affected (Darley, 1982). In addition, aphasia may result in quality of life issues including social isolation, depression, stigma, financial hardship and changes in personal relationships (Parr, 2007).

It is estimated that approximately one million people in the United States suffer from aphasia and that 100,000 Americans will acquire the disorder each year (National Aphasia Association, 2007, NIH, 2008). Furthermore, the National Aphasia Association (2007) report that 25 to 40% of individuals that survive a stroke will acquire aphasia.

While aphasia is caused by different etiologies, the most common is stroke (Benson &Ardilla, 1996). According to Haynes and Pindzola (2004), there are two types of strokes. The two types of strokes are ischemic and hemorrhagic. An ischemic stroke is caused by interruption of blood flow to the brain, such as a blood clot. A hemorrhagic stroke is caused by a rupture of a blood vessel.

Stroke is the leading cause of disability in adults and the third leading cause of death in the United States. Strokes occur due to several causes including high blood pressure, inactivity, diabetes and coronary artery disease. Strokes do not discriminate against gender, race or age. However, some evidence exists that the risk of stroke increases with age and that some ethnic groups have a higher incidence (Torbey&Selim, 2007). While data on gender differences is limited, some evidence exists to suggest that more women than men suffer from strokes (American Heart Association Statistical Fact Sheets, 2010; Petrea, Beiser, Seshadri, Keyy-Hayes, Kase& Wolf, 2009).

Aphasia Classification

Currently aphasia is classified based on site of lesion, extent of lesion and language characteristics. There are two subtypes of aphasia under the global disorder of aphasia. The two subtypes are non-fluent and fluent aphasias. The non-fluent aphasias are characterized by anterior brain lesions, disrupted speech and relatively intact auditory comprehension. These aphasias are Broca's, transcortical motor, and global.

Broca's aphasia is characterized by agrammatic speech, restricted vocabulary and difficulty initiating speech. Transcortical motor aphasia is characterized by intact repetition but with limited speech. Phonemic and global paraphasias as well as syntactical errors are present. Patients exhibit perseveration and have difficulty imitating and organizing responses in conversation. In transcortical motor aphasia, auditory comprehension is impaired but confrontation naming is not. The third non-fluent aphasia is global aphasia. Global aphasia is characterized by impaired comprehension and expression. Patients with global aphasia demonstrate little to no ability to communicate, have difficulty following simple commands and responding to simple questions (Chapey, 2008).

The fluent aphasias are characterized by posterior brain lesions and fluent speech. The fluent aphasias include conduction, transcortical sensory, anomic and Wernicke's. Conduction aphasia is characterized by fluent speech with severe difficulty repeating words and

sentences. Auditory comprehension is usually mildly impaired in conduction aphasia. Transcortical sensory aphasia is characterized by fluent speech with frequent paraphasias and neologisms. Repetition is usually preserved but patients demonstrate difficulty with confrontation naming and auditory comprehension. The third fluent aphasia is anomic aphasia. It is characterized by fluent speech and word retrieval problems. The last fluent aphasia is Wernicke's aphasia. It is characterized by deficits in auditory comprehension. Fluent speech is marked with paraphasias. Paraphasias are in the form of word substitutions and sound transpositions (Chapey, 2008). The types of aphasia with the associated language characteristics and primary site of lesion are summarized in Table 2.1.

Classification of aphasia can be done using behavioral assessment instruments such as the Western Aphasia Battery (WAB) (Kertesz, 1982) and the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass& Kaplan, 1972). However, these behavioral assessment instruments may not always agree with each other, other classification systems or clinicians. This disagreement is reported in studies that compare classification systems (Swindell, Holland & Fromm 2009; Wertz, Deal & Robinson, 1984; Wertz, 2009).

Table 2.1 Aphasia Classification (Brookshire, 2003)

Category	Type of Aphasia	Site of Lesion	Language Characteristics
Non-fluent	Broca's	Inferior frontal gyrus known as Broca's area	Agrammatic speech Relatively preserved comprehension Abnormal prosody
Non-Fluent	Transcortical Motor	Prefrontal cortex superior and anterior to Broca's area	Similar to Broca's aphasia Preserved repetition
Non-fluent	Global	Perisylvanian region	Compromised expressive and receptive language Reading and writing are affected
Fluent	Conduction	Arcuate Fasciculus	Impaired repetition Relatively preserved expressive and receptive language
Fluent	Transcortical Sensory	Posterior areas of the brain close to the boundary of the temporal and occipital lobes	Similar to Wernicke's aphasia Preserved repetition
Fluent	Anomic	Posterior temporoparietal boundary Posterior superior temporal gyrus	Word naming difficulties Relatively normal expressive and receptive language
Fluent	Wernicke's	Superior temporal gyrus known as Wernicke's area	Impaired comprehension Use of jargon Preserved articulation, prosody, grammar and syntax

Wertz (2009) compared agreement of aphasia classification between two clinicians using the WAB and the BDAE. The findings from this study show that agreement between the clinicians was obtained on only 62% of the patients evaluated. Other studies have found similar results. Swindell, Holland and Fromm (2009) examined aphasia classification by the WAB to clinical impression. The results of this study revealed that aphasia classification between the WAB and clinical impression matched on only 54% of the patients examined. In another study on comparison of aphasia classification on the WAB and the BDAE of forty-five individuals

with aphasia, the overall agreement between the two behavioral assessment instruments was 27% (Wertz, Deal & Robinson 1984).

Other classification systems have been developed since classification of aphasia based on site of lesion and language characteristics is thought to be unreliable. Schuell developed a classification system for aphasia that is based on the severity of the language impairment, presence or absence of related sensory or motor deficits and prognosis. According to Schuell's classification system, aphasia is classified as follows; simple aphasia, aphasia with visual involvement, aphasia with persisting dysfluency, aphasia with scattered findings, aphasia with sensorimotor involvement, aphasia with intermittent auditory imperception and irreversible aphasia syndrome (Chapey, 2008).

According to Schuell's system, simple aphasia is described as a mild multimodality language impairment with excellent prognosis for recovery. Aphasia with visual involvement is a mild aphasia with difficulties in visual discrimination, recognition and recall. Prognosis is excellent except for reading and writing. Aphasia with persisting dysfluency is a mild aphasia with dysfluent speech and excellent prognosis. Aphasia with scattered findings is a moderate aphasia characterized with dysarthria, emotional lability and/or visual involvement. Prognosis is limited.

Aphasia with sensorimotor involvement is severe language disorder with impaired perception and production of phonemic patterns. Prognosis is limited to functional recovery of language. Aphasia with intermittent auditory imperceptions is described as a severe language disorder with severe difficulties involving auditory processing. Prognosis for recovery is limited. Irreversible aphasia syndrome is described as almost complete loss of all language modalities with poor prognosis for recovery (Chapey, 2008). Schuell's aphasia classification system is summarized in Table 2.2.

Table 2.2 Schuell's Aphasia Classification

Aphasia Type	Severity	Related Sensory/Motor Deficits	Prognosis
Simple Aphasia	Mild	Multi modality language impairments	Excellent
Aphasia with Visual Involvement	Mild	Visual discrimination, recognition, recall	Excellent
Aphasia with Persisting Dysfluency	Mild	Dysfluent Speech	Excellent
Aphasia with Scattered Findings	Moderate	Dysarthria, emotional lability, visual Involvement	Limited
Aphasia with Sensorimotor Involvement	Severe	Impaired Perception and Production of Phonemic Patterns	Limited to Recovery of Functional Language
Aphasia with Intermittent Auditory Imperception	Severe	Auditory Processing	Limited
Irreversible Aphasia	Severe	Complete Loss of All Language Modalities	Poor

Since Schuell's classification system of aphasia includes a description of the severity, complexity and prognosis for recovery, studies have shown its utility for non-clinicians. For example, in a study on incidence of aphasia type in acute stroke patients, Brust, Shafer, Richter, and Bruun (1976) demonstrated that descriptions of language characteristics by non-speech pathologists were more useful in the emergency room and could be used to determine whether the patient was improving or not. This study examined eight hundred and fifty patients and was conducted in a hospital emergency room. As a result, it differs from other studies on aphasia classification because of the large number of patients that were included and the setting where the study was conducted.

The utility of describing language characteristics found in a particular aphasia is evidenced by the fact that as aphasia recovers; it evolves into a different type. For example, in a study on evolution of aphasia type, Holland, Swindell and Forbes (2009), observed fifteen patients with global aphasia for 15 minutes every day during the course of their hospitalization.

Observations consisted of conversation with a speech pathologist. Conversations were recorded and transcribed by a trained speech language pathologist. At discharge, patients were evaluated using the WAB. Patients were re-evaluated at 4-6 weeks, 10-12 weeks, 6 months and at one year. The criterion for re-evaluation was a score below the WAB's normal aphasia quotient of 93.8. Results show that two of the male patients evolved from global aphasia to Broca's aphasia and two female patients evolved into Wernicke's aphasia suggesting that their language characteristics had changed over the course of recovery. In his study on agreement of aphasia classification, Wertz (2009) reported that over 50% of the patients examined evolved into a different aphasia type within one-year post onset.

Swindell, Holland and Fromm (2007) compared aphasia type based on the WAB classification and clinical impression and reported similar results. Results show that in a group of sixty-nine participants, aphasia classification changed based on clinical impression. One patient's aphasia evolved from conduction to anomic aphasia while two patients with Wernicke's aphasia evolved to conduction aphasia.

Studies on the usefulness of describing language characteristics in the individual with aphasia show that as the individual with aphasia recovers language characteristics change and are therefore difficulty to classify. In addition, a description of language characteristics is beneficial for several reasons. First, aphasia classification does not necessarily describe the current language characteristics exhibited by a patient at the time of treatment. Secondly, a description of the language characteristics is necessary since change in aphasia type requires changes in treatment strategies, prognosis, counseling and education options provided to the patient and family. Evolution of aphasia type may be related to several factors thought to influence recovery from aphasia.

FACTORS ASSOCIATED WITH RECOVERY FROM APHASIA

Initial severity of the disorder, behavioral intervention provided, age, gender and lesion size and location are factors thought to influence recovery form aphasia.

Initial Severity and Recovery from Aphasia

Recovery of aphasia continues to be a problem since it is difficult to determine how much recovery will be achieved. According to Kertesz and McCabe, (1977) initial severity of the disorder appears to be the most important factor in determining level of recovery. Findings from a prospective study that looked at 330 patients with aphasia provide evidence to support Kertesz and McCabe (1977) findings. Severity ratings for 330 patients were determined based on the Aphasia Scale of the Scandinavian Stroke Scale. This scale rates aphasia based on the following levels; severe, moderate, mild and no aphasia. According to this aphasia scale, language restricted to only "yes", "no" or less is rated as severe aphasia. In aphasia rated as moderate, language consists of more than "yes", "no" and a few other things. In moderate aphasia, long sentences are not produced. In mild aphasia, a patient demonstrates limited vocabulary or incoherent speech.

At admission to the hospital, 48% of the 330 patients were classified as severe aphasia, 18% as moderate aphasia and 10% as mild aphasia. By discharge from the hospital, only 12 % of the patients with severe aphasia had recovered. However, 41 % of the patients with moderate aphasia and 56 % of the patients with mild aphasia had recovered. The findings reported in this study suggest that patients with initial severity levels in the mild to moderate range have a better probability of recovery than those with initial severity levels in the severe range. The authors concluded that for individuals with aphasia with severity levels in the severe range, reaching pre-morbid levels was highly unlikely (Pedersen, Jorgensen, Nakayama, Raaschow& Olsen, 1995).

However, Demeurisse, Demol, Derouck, De Beucklaer, Coekaerts and Capon (1980) reported results that were different from those reported by Pedersen et. al., (1980). This study examined seventy-five individuals with aphasia and left hemisphere lesions. Thirty-four males with a mean age of 66 years and forty-one females with a mean age of 68 years were included in this study. Participants were divided into groups based on aphasia classification; Broca's,

Wernicke's or global. Participants were evaluated upon admission to the hospital and monthly thereafter for a period of six months.

Assessment consisted of ten tests for comprehension and nine tests for expression. Comprehension tests included tasks such as following simple, semi-complex and complex commands using body parts, and material aids. In addition, participants were asked to identify objects in a set of pictures. Expressive language assessment tasks included naming familiar objects, reciting automatic sequences such as digits and names of the week. Additional tasks used for the expressive language assessment were repetition of words and sentences as well as naming of category members. While behavioral results show that individuals classified as Wernicke's or Broca's aphasia improved more than the globally aphasic patients, no statistically significant effect was found between the initial severity of aphasia and final assessment score at the sixth month assessment.

Behavioral Intervention

According to some researchers behavioral intervention appears to benefit some patients if it is provided months post onset while others suggest that recovery can be seen even two years post onset (Aten, Caliguiri& Holland, 1980; Hanson, 1989; Shewan&Kertesz, 1984). Aten, Caliguiri and Holland (2008) examined seven patients with aphasia that were treated using functional communication therapy in a group setting. The goal of functional communication therapy is to improve communication in functional situations. These situations include activities such as shopping in a grocery store, social greetings and exchanges, providing personal information, giving and following directions, reading signs and directories and responding with gestures to express ideas. Treatment was provided in hour-long sessions twice a week for twelve weeks.

Following the twelve-week therapy period, patients were post-tested using the Porch Index of Communicative Ability (PICA) (Porch, 1967) and the Communicative Abilities in Daily Living (CADL) (Holland, Frattli& Fromm, 1999). At post-testing, the patients did not

show any improvement in their PICA scores however, the patients showed improvement in their performance scores on the CADL. Evidence to support the finding of improved functional behavior reported by Aten, Caliguiri and Holland (2008) is found in the results reported by Doyle, Goldstein, Bourgeois, and Nakles (1989). In this study, four Broca's patients were examined. The number of self-initiated requests with familiar and unfamiliar conversation partners was recorded at baseline. Participants were trained to use verbal requests to initiate conversations about various topics such as personal information, leisure and health using feedback and prompting. Feedback consisted of praise and rewards. Rewards were in the form of response from the conversation partners. Prompting consisted of cues through eye contact, modeling or use of cue cards. All of the participants demonstrated an increase in their self-initiated requests from baseline to treatment on the topics used for training but generalization did not occur for untrained topics. In addition, the results show that following the functional communication training all four of the participants generalized to conversations with familiar conversation partners. Furthermore, all four participants increased their requests for information with unfamiliar conversation partners but the effect was not as strong as that demonstrated with familiar conversation partners.

Studies on the effects of speech therapy intervention were reviewed. In this study, thirty-two males and twenty females were randomly assigned either to a group receiving speech therapy or to a group that did not receive speech therapy. Participants that were in the speech therapy intervention group were treated twice a week for twenty-four weeks. All participants were assessed at four, ten, twenty-two and thirty-four weeks after the onset of the stroke using the PICA, Functional Communication Profile (FCP) (Sarno, 1969) and the Speech Questionnaire. Results show that a statistically significant difference was found at the four-week assessment on the PICA for all participants. However, no significant difference was found between the 10th and 22nd week assessments. In addition, participants were found to deteriorate in their overall PICA score over time. Results also show that while some recovery was seen in all participants between the 10th and 22nd week assessment on the FCP, it was not statistically

significant. Furthermore, no statistically significant difference was found between the 10th, 22nd, and 34th week assessment on the FCP for all participants. These findings suggest that speech therapy intervention may not improve scores on measures of language ability in individuals with aphasia (Lendrum& Lincoln, 1985).

Findings from some imaging studies have shown that speech therapy intervention did not change cortical activity in individuals with aphasia. Richter, Miltner and Straube (2008) used fMRI to compare brain activation patterns of individuals with aphasia that received speech therapy intervention to non-brain damaged individuals. Twelve males and four females with aphasia that were at least twelve months post onset participated in the study. Seven participants were classified as Broca's, seven anomic and two globally aphasic. The participants with no brain damage included four males and four females. All participants were scanned at baseline. The individuals with aphasia were scanned at baseline and after speech therapy concluded. Individuals with aphasia participated in constraint induced aphasia therapy. Constraint induced aphasia therapy practices language tasks that the patient has difficulties with but without the use of compensatory strategies such as gestures and/or phonemic cues. Constrained induced aphasia therapy was provided for three hours a day for ten days.

During the fMRI scanning, all participants were instructed to read words that appeared on a screen and to complete word stems to one meaningful word silently. At the baseline scan, the participants with no brain damage exhibited brain activation in the left inferior frontal gyrus, insula, right superior frontal gyrus, middle and inferior occipital gyri. The individuals with aphasia exhibited activation in the right inferior frontal gyrus, insula, left inferior frontal gyrus and insula and frontal perilesional tissue.

The fMRI results show no statistically significant difference in cortical activation between the left and right hemisphere from scan one to scan two for the individuals with aphasia. The results also show that individuals with aphasia exhibited activation similar to participants with no brain damage but with stronger activation in the right inferior frontal gyrus and insula

during the reading task. In individuals with aphasia, activation was seen in the right post central gyrus during the word-stem completion task.

Behavioral results from the study described above show that the individuals with aphasia improved from the first assessment to the second assessment on spontaneous speech, auditory comprehensibility and semantic comprehensibility. Furthermore, behavioral changes on measures of language ability correlated with stronger activation in the right hemisphere suggesting some degree of reorganization in the right hemisphere.

Age

Many have suggested that younger individuals with aphasia have better recovery rates as measured by language performance on behavioral assessment instruments (Holland, Swindell& Forbes, 2009; Kertesz& McCabe, 1977: Landrum & Lincoln, 1985). Holland, Swindell and Forbes (2009) observed a group of patients with lesions in the left hemisphere and global aphasia aged 35-84 years. The patients were evaluated using the WAB at hospital discharge, 6-8 weeks, 10-12 weeks, six months and a year post onset. Results show that two patients in their thirties recovered normal language function while those in their eighties remained globally aphasic at one year post-onset. Similarly, Landrum and Lincoln (1985) reported that patients in the 60-69 age group had lower performance scores on the PICA than the younger patients examined in their study on effects of speech therapy on recovery from aphasia.

According to Ferro and Crespo (1988), younger individuals with aphasia tend to have better recovery. The lack of recovery in older individuals with aphasia may be associated with age-related mental decline, anatomic and physiologic changes. The results of this retrospective study show that younger patients rarely suffer from temporal infarcts. Thus, their aphasia severity level is less than that found in older patients. The authors concluded that recovery from aphasia in this group of younger patients was related to location of lesion. Similarly, Kertesz and McCabe (1977) reported that younger patients demonstrated better recovery patterns but the recovery was dependent on other factors such as initial severity of the disorder.

Gender

Gender is another variable that has been examined in the recovery from aphasia. According to Basso, Capitani and Moraschini (1982) females recover better than males in oral expression but not in auditory verbal comprehension. On the other hand, several others have reported that gender has no influence on recovery from aphasia (Demeurisse et. al., 1980; Kertesz, 2009). For example, Lendrum and Lincoln (1985) investigated the effects of speech therapy on a group of individuals with aphasia. Participants were randomly assigned either to a group receiving speech therapy or to a group not receiving speech therapy. Participants were assessed at four, ten, twenty-two and thirty-four weeks using the PICA, FCP and the Speech Questionnaire. The results show that gender had no effect on the amount of change in performance as measured by the PICA and the FCP.

Lesion Size and Site of Lesion on Recovery

According to Kertesz (1988), lesion size and site of lesion are also important factors in recovery from aphasia. Kertesz (1988) used available CT scans from eighty-two individuals with aphasia to examine lesion size and recovery from aphasia during the 0-3 months post stroke. Results show that structures within the temporal and inferior parietal regions as well as in the insula of the homologous hemisphere appear to be responsible for compensation in auditory comprehension in individuals with aphasia. This suggests that if structures within the temporal and inferior parietal regions as well as the insula of the homologous hemisphere are damaged, then recovery and compensation is unlikely.

In a retrospective study that examined the relationship between aphasia severity and lesion location in Koreans, Kang, Sohn, Han, Kim, Han and Paik (2010) reviewed the medical records of ninety-seven patients with left hemisphere stroke and aphasia. The patient's aphasia type was classified according to the scores on the validated Korean version of the Western Aphasia Battery (Kim & Na, 2001). Findings from the medical record review show that the most

severe aphasia type in this group of patients was global aphasia and that the site of lesion was the insular cortex suggesting that aphasia type correlated with site of cortical lesion.

In a study that used fMRI to examine brain activation after speech therapy training, Richter, Miltner and Straube (2008) reported that patients with aphasia, exhibited activation in the insula of the right hemisphere pre and post speech therapy. These findings suggest that individuals with aphasia access the insula of the right hemisphere to compensate for the lesioned left hemisphere. These findings are consistent with the findings reported by Kertesz (1988) suggesting that when the lesion involves the insula of the homologous hemisphere, aphasia severity level is severe and compensation by the homologous hemisphere is highly unlikely.

Summary

Factors such as initial severity of the disorder, behavioral intervention provided, age, gender, lesion size and location are thought to influence recovery from aphasia. Review of the literature, shows that those with more severe aphasia type (i.e. global aphasia) improve less than those with less severe aphasia types (i.e. Wernicke's and Broca's.).

Several study results support the claim that behavioral intervention may not improve scores on measures of language ability in individuals with aphasia. However, evidence has also been presented to support the claim that behavioral intervention treatment results in increased performance in functional language such as social greetings and providing personal information.

Furthermore, findings from studies on the influence of speech therapy intervention and recovery from aphasia report that following speech therapy intervention, individuals with aphasia generalize to trained topics with familiar and unfamiliar conversation partners but they do not generalize to untrained topics.

Neuroimaging studies on the effects of speech therapy intervention on cortical activation show that patients with aphasia exhibit patterns of cortical activation similar to non-brain damaged individuals but with stronger activation in the right inferior frontal gyrus and insula post speech therapy treatment.

Younger individuals with aphasia demonstrate improved performance on measures of language ability but this improvement may be associated to other factors such as lesion size, location and initial severity of the disorder. According to the results from several studies on factors that influence recovery from aphasia, gender has been found to have no significant effect on the recovery process. Lesion size and location appear to be important to recovery from aphasia, particularly if the lesion size is so large that it engulfs the insula of the right hemisphere.

It is important to consider age, gender, behavioral intervention provided, lesion size, location and severity of the disorder when looking at recovery from aphasia.

NEUROPLASTICITY

Studies on recovery of aphasia suggest that individuals with aphasia do get better in spite of damage to the language centers. One of the explanations for recovery of function in aphasia is neuroplasticity. Neuroplasticity is the brain's ability to reorganize its structures and function to create new pathways. While there are several types of neuroplasticity, only the ones specific to recovery from aphasia will be discussed. The two types of neuroplasiticity specific to recovery from aphasia are homologous area adaptation and map expansion.

Homologous Area Adaptation

Homologous area adaptation refers to the shifting of functions from a damaged brain region to another that is not affected (Grafman, 2000). In the case of aphasia, the right hemisphere takes over functions that were previously executed by the left hemisphere. Imaging technology has been used to study the theory of homologous area adaptation in individuals with aphasia.

Musso, Weiller, Kiebel, Muller, Bulau and Rijntjes (1999) examined short-term changes in the cortical network of four individuals with aphasia brought about by short, intense periods of language comprehension training using PET.

Language comprehension training consisted of eleven, eight-minute sessions. Training was completed during the 12-minute intervals that it took the radioactive isotopes to be

absorbed into the blood stream between each PET scan. Training consisted of five different tasks that included following five commands using different objects, looking at pictures of different objects and indicating one of the pictures after an oral command was presented. The third task asked the participant to decide whether an orally presented sentence was correct or not. The fourth task required the participant to decide whether an orally presented sentence was phonologically accurate. The final task required that the participant match a picture to the corresponding sentence. The complexity and length of the tasks increased with each presentation. The tasks were presented in random order.

Results show that the all four of the participants exhibited increased activation in the posterior part of the right superior temporal gyrus suggesting that intense language comprehension training activates the right hemisphere.

Further evidence to support the theory of homologous area adaptation is reported by Heiss, Kessler, Thiel, Ghaemi and Karbe (1999). The twenty-three participants included in the study were grouped according to site of lesion. Seven participants were included in the frontal lesion group. Nine participants were included in the subcortical lesion group and seven in the left temporal lesion group. A baseline PET scan was done at two weeks post onset. The second scan was completed at eight weeks post onset. The task required participants to repeat single words that were presented orally. At the two-week scan, participants in the subcortical and frontal lesion group demonstrated activation in the inferior frontal gyrus and the right superior temporal gyrus during the word repetition task. The participants in the left temporal lesion group showed activation in Broca's area and the supplementary motor areas. Furthermore, at the eight-week scan the participants in the subcortical and frontal groups activated the left superior temporal gyrus during the same word generation task. In addition, the participants in the left temporal group activated the right superior temporal gyrus and the precentralgyrus bilaterally. The authors concluded that the right hemisphere assists in language function recovery when lesions are located in the left hemisphere.

In a study by Cao, Vidingstad, George, Johnston and Welch (1999) fMRI was used to examine the functional anatomy of individuals with aphasia after substantial recovery from aphasia. Seven individuals with aphasia including two males and five females between the ages of twenty to fifty-six years participated in the study. Two language related tasks were used. During the picture-naming task, the participant was shown thirty-six black and white line drawings of objects. The participant was instructed to name the line drawings silently. During the verb-generation task, thirty-six pictures of common concrete nouns were shown to the participant. The participant was instructed to silently generate a verb associated with that noun. The tasks were divided into four periods with nine items presented during each period.

Participants were scanned during the four periods with a control scan completed in between each experimental period. The purpose of the control scan was to remove activation from early visual processing.

Results show that the participants exhibited activation in the left inferior frontal lobe, left inferior frontal gyri, left inferior parietal/superior temporal lobes including the supramarginal angular and superior temporal gyri during the picture-naming task. Activation was also seen in the occipital lobe, insula, superior parietal lobule and anterior cingulated gyrus. In addition, activation was also exhibited in the homologous right hemisphere but to a lesser degree.

During the verb generation task, participants exhibited activation in the left inferior frontal lobe, inferior parietal and superior temporal lobes as well as regions adjacent to the intraparietal sulcus. These data suggests that recovery of aphasia may be influenced by reorganization of damaged language areas in the left hemisphere with compensation provided by areas in the right hemisphere.

Behavioral evidence presented by Basso, Gardelli, Grassi and Mariotti (1989) support the theory of homologous area adaptation. They describe two patients with aphasia following a left hemisphere stroke. After suffering a second stroke in the right hemisphere, the patients demonstrated worsening of their language abilities. These findings suggest that the right hemisphere may provide assistance in processing language when damage to the left hemisphere

occurs. This reorganization of function to the right hemisphere has been reported in the literature. For example, Vanlancker-Sidtis (2004) described a fifty-seven year old patient that underwent a hemispherectomy at the age of five and a half years due to seizure activity. The hemispherectomy involved surgical removal of the left cerebral cortex with sparing of the basal ganglia and some ependyma of the temporal horn. Behavioral testing was completed on the patient with hemispherectomy using the Revised Token Test. Results show normal performance in response to spoken messages.

Map Expansion

Others disagree with the hypothesis that homologous area adaptation plays a role in recovery of aphasia and suggest that map expansion is responsible for recovery of aphasia. Map expansion refers to the expansion of cortical brain regions around the damaged area. In the case of aphasia, the damaged area is the left hemisphere. This is achieved through practice or continual exposure to a stimulus (Grafman, 2000).

Warburton, Price, Swinburn and Wise (1999) used PET to compare brain activation during a word retrieval task in individuals with aphasia and participants with no brain damage. Six individuals with aphasia including three males and three females with a mean age of 48 years participated in the study. Nine individuals with no documented history of brain damage served as the control group. The participant was instructed to think of as many verbs that could be associated with an auditorially presented noun.

Results show that the individuals with aphasia activated the left dorsolateral frontal cortex and all but one of the individuals with aphasia exhibited activation of the left posterior inferolateral temporal cortex. Three of the individuals with aphasia showed activation in the right dorsolateral frontal cortex but to a lesser degree. Eight of the nine participants with no brain damage exhibited activation in the left thalamus. These findings are consistent with the findings from other studies that use PET to investigate the theory of map expansion and report reactivation of left hemisphere areas around the lesioned area. For instance, DeBoissezon,

Marie, Castel-LaCanal, Marque, Bezy, Gros, Lotterie, Cardebat, Puel, Demonet, (2009) used PET to examine whether reactivation of left hemisphere areas around the lesioned areas contribute to recovery from aphasia. Thirteen individuals with left hemisphere stroke and aphasia participated in the study. All individuals with aphasia had received speech therapy intervention. Based on the performance of the individuals with aphasia on the word generation task used during the second PET scan, participants were assigned to a good recovery or a poor recovery group. Participants were scanned twice at one-year intervals. Six PET scans were completed during each experimental session. Participants were asked to silently produce semantically related words belonging to the grammatical category of the target stimuli.

Behavioral results show that the participants in the good recovery group had improved significantly for fluency and close to significantly for word generation. Although no significant effect was found, the poor recovery group demonstrated improvement in comprehension and naming. Neuroimaging results show that on the word generation task, the individuals with aphasia in the good recovery group demonstrated activation of both posterior superior temporal gyri and the right inferior frontal gyrus and of the cerebellum during the first PET scan. At the follow up scan, a year later, the PET scan results show that the good recovery group exhibited activation in the left posterior superior temporal gyrus and that it was greater than on the first PET scan. Activation was also extended to a portion of the supramarginalgyrus.

In contrast, the poor recovery group exhibited activation to both posterior superior temporal gyrus during the first PET scan. At the follow up PET scan a year later, the same activation was still present but to a smaller degree.

In yet another study of aphasia, PET was used to investigate the theory of map expansion. Karbe, Thiel, Weber-Luxenbuger, Herholz, Kessler and Heiss (1998) examined twelve individuals with aphasia during a word repetition task. Participants were scanned two times within one week. During the first scan, participants were asked to rest with eyes closed. During the second scan, patients were asked to repeat nouns that were presented orally.

Activation of the left superior temporal cortex with some recruitment of the right hemisphere was observed.

A follow-up PET scan was completed one year later. Results of the follow-up scan showed activation of the left superior temporal cortex but with no activation of the right hemisphere suggesting that the damaged left hemisphere had reorganized the language networks.

fMRI has been used to study the theory of map expansion. Zahn, Drews, Specht, Kemeny, Reith, Willmes, Schwarz and Huber (2004) found that semantic processing activated the left hemisphere. However, no significant left to right hemisphere shift was observed. Similar findings were reported by Zahn, Huber, Drews, Specht, Kemeny, Reith, Willmes, and Schwarz (2002). In a task of explicit semantic judgment that required participants to listen to a list of congruous and incongruous words, activation of the left posterior superior middle and inferior temporal gyrus was seen.

Summary

Findings from studies using neuroimaging technology such as PET and fMRI as well as behavioral descriptions show that reorganization of the brain may be beneficial to individuals with aphasia. The results of studies on map expansion and homologous area adaptation and recovery from aphasia suggest that recovery may be related to a reorganization of a network of areas. Despite the findings that have been reported in the literature regarding map expansion and homologous area adaptation, the roles of the right and left hemisphere in the recovery of aphasia are still not clear.

NEUROIMAGING TECHNOLOGY USED IN APHASIA RESEARCH.

In the past, recovery of aphasia has been assessed using standardized assessment instruments that measure functional gains. While these behavioral assessment instruments provide standardized measures and baseline performance data, they are not reflective of one specific cognitive process but of many individual cognitive processes. In addition, behavioral assessment measures cannot determine the specific stage at which processing occurs so the

temporal characteristics of processing are unknown. Furthermore, behavioral assessment methods do not provide data about specific anatomical structures associated with recovery of aphasia. Finally, a participant's physical disabilities may limit the efficacy of the behavioral assessment measures.

The availability of neuroimaging technology makes the task of correlating recovery from aphasia with specific anatomical structures and temporal aspects of semantic processing easier to accomplish. Currently several imaging options exist. Those most commonly used in aphasia research include Functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), and Event Related Potentials (ERP).

Functional Magnetic Resonance Imaging (fMRI)

fMRI examines functional activity in the brain by measuring oxygen content in the blood that results from an increase in neural activity in response to a stimulus. Since fMRI can be used with participants that are actively engaged in cognitive tasks, it is considered a valuable tool for examining the anatomical structures associated with recovery from aphasia (Buxton, 2002; Cao, Vidingstad, George, Johnston & Welch, 1999).

fMRI has been used to examine the functional anatomy of individuals with aphasia after substantial recovery from aphasia (Cao, et. al., 1999). In this study, the participants were instructed to silently name thirty-six black and white drawings of objects and to silently generate a verb associated with that noun.

fMRI scanning results showed that the seven participants exhibited activation in the left inferior frontal lobe, left inferior frontal gyri, left inferior parietal/superior temporal lobes including the supramarginal angular and superior temporal gyri during the object-naming task. Activation was also seen in the occipital lobe, insula, superior parietal lobule and anterior cingulated gyrus. In addition, activation was seen in the homologous right hemisphere but to a lesser degree.

Furthermore, participants exhibited activation of the left inferior frontal lobe, inferior parietal and superior temporal lobes as well as regions adjacent to the intraparietal sulcus during the verb generation task. Performance on behavioral responses was not monitored because the language task was done silently in order to avoid producing too many artifacts that could distort the BOLD signal (Cao, et. al., 1999).

Positron Emission Tomography (PET)

PET is another technique that is used in aphasia research because it has spatial resolution in the millimeter range. Radioactive tracer isotopes are injected directly into the blood stream and observed by the organ or tissue of interest. A three dimensional picture is constructed by computer analysis of the radioactive concentrations within the organ or tissue being studied (Bailey, 2005; Handy, 2005).

Evidence to support the utility of PET to localize the anatomical brain regions involved in the recovery of aphasia is presented in a study by Musso, Weiller, Muller, Bulau, Rijntjes (1999). The results of this study show that training designed to improve auditory comprehension activated the right hemisphere. Further support is provided by the findings reported by Heiss, Kessler, Thiel, Ghaemi and Karbe (1999). Regional cerebral blood flow (rCBF) patterns in individuals with aphasia were examined during the spontaneous recovery period. Twenty-three participants were grouped according to site of lesion. There were seven participants with frontal lesions, nine with subcortical lesions and seven with left temporal lesions. Baseline scan was done at two weeks post onset. The second scan was completed at eight weeks post onset. At the two-week scan, participants in the subcortical and frontal lesioned group activated the inferior frontal gyrus and the right superior temporal gyrus during a word repetition task. The participants in the left temporal lesioned group activated Broca's area and the supplementary motor areas. Results show that at the eight-week scan the participants in the subcortical and frontal groups activated the left superior temporal gyrus during the same word generation task.

At the eight-week scan, the participants in the left temporal group activated the right superior temporal gyrus and the precentralgyrus bilaterally during the word generation task.

The studies discussed above highlight the utility of fMRI and PET technology in localizing the anatomical structures associated with the recovery from aphasia. While they provide excellent spatial resolution that allows localization of anatomical areas associated with the recovery of aphasia, they lack temporal resolution required to determine at what time point in the process auditory comprehension of a spoken sentence length message occurs.

Event Related Potential (ERP)

ERP measures electrical activity at the level of the cortex in response to internal and external stimulus through the skull and scalp using an electrode cap with electrodes attached (Handy, 2005; Luck, 2005). While interest in measuring electrical activity in the brain started in the 1920's, it was not until the 1960's that ERP research gained popularity due to its non-invasive nature and cost effectiveness (Rugg& Coles, 1997). The electrical activity can be time-locked to a specific stimulus event allowing researchers to make functional inferences about cognitive activities such as memory, attention and language at the level of the cortex (Handy, 2005; Hagoort et al., 1997; Luck, 2005; Rugg& Coles, 1997).

ADVANTAGES AND DISADVANTAGES OF NEUROIMAGING TECHNOLOGY

Since ERP can be time locked to a specific stimulus, it measures electrical activity in the brain over time. ERPs have a temporal resolution in the millisecond range. fMRI, on the other hand measures hemodynamic responses that occur in response to a stimulus after a delay of approximately 1-5 seconds with peak occurring at 4-5 seconds after the onset of the stimulus (Buxton, 2002). Similarly, PET requires a waiting time of 10 minutes to an hour while the radioactive isotope binds to the glucose or water in the body before any measurements can be taken (Handy, 2005; Ingham, 2007). Since listening and interpreting spoken messages frequently takes place in milliseconds and given that ERP has excellent temporal resolution of one millisecond, ERP can be used to determine the time point at which auditory processing of

spoken messages occurs. Therefore, the advantage of ERP is that it will capture cortical activity immediately overtime and thus permit some confidence in the relationship between external stimulation and cortical activity.

Cost and availability are issues that are considered in research because of limited resources. ERP is cost effective and easily available in most university labs. PET on the other hand, is costly. The isotopes used in PET procedures have short life spans and manufacturing them requires special chemical synthesizing equipment Similarly, fMRI is costly due to the equipment and specialized rooms required to house it (Bailey, 2005; Buxton, 2002; (Handy, 2005; Ingham, 2007).

One of the most important benefits of ERP is its non-invasive nature. In its early use, the participant's scalp was scraped slightly to attach the electrodes. Currently, the electrodes can be attached to a skullcap. Furthermore, the non-invasive nature of the ERP procedure has the potential for increasing the number of participants willing to participate in experimental studies. Since ERP measures electrical activity at the level of the cortex over time, it can provide an online measure of processing without the need of a behavioral response. This characteristic makes ERP a good tool for use with individuals that have developmental delays and limited language abilities (Handy, 2005; Luck, 2005).

One of the limitations of ERP is its poor spatial resolution so localizing specific anatomical regions associated with the functional activity is problematic. Electrodes are placed along specific points on the scalp using the International 10-20 System (Jasper, 1958). The reason for the lack of spatial resolution is that the ERP signals are the result of the averages of many electrodes. Therefore, localization of brain activity is reported relative to the electrode placement (Handy, 2005).

To summarize, ERP has several advantages that make it a reasonable choice for use with brain-damaged individuals. While ERP has low spatial resolution that does not allow for localization of specific anatomical areas, it has excellent temporal resolution of approximately one millisecond. As such, it measures the brain's response to an internal and external stimulus

across time and because it can be time locked to a specific event, it provides continuous measurement of brain processing. Secondly, ERP is a relatively low invasive procedure. It measures electrical activity using a skullcap with electrodes attached. Finally, ERP is cost effective and readily available in most research and clinical settings.

THE BASICS OF ERP

Given the advantages that ERP has over fMRI and PET, including its relatively non-invasive nature, excellent temporal resolution and its ability to measure electrical activity at the level of the cortex across time it is a reasonable choice for use in the study of auditory comprehension.

Artifacts

ERP signals are the result of the averages of many electrodes. As such, the signal of interest is hidden within other brain activity called artifacts. It is during the averaging process, that artifacts are rejected. Artifacts are caused by muscle movement, eye blinks, galvanic responses and electrical activity from other sources. The ERP wave must be averaged and filtered in order to extract the signal (Handy, 2005). The process of filtering removes artifacts from the waveform so that the relevant signal can be seen. Filtering is usually carried out with electronic circuits built into the recording equipment. Filters remove high and low frequency components that are not expected to contain the relevant components. Since ERP can be time-locked to a specific event, averaging across time windows known as epochs will result in a clear ERP signal (Handy, 2005; Luck, 2005).

Event Related Potential (ERP) Components

ERP waveforms consist of peaks and troughs called components that are observed after the signal has been extracted. The components of a wave are the increased electrical activity that is observed after the onset of an internal or external stimulus. This increased electrical activity is assumed to reflect the underlying cognitive or communication process that is being examined.

Components are typically referred to by either an N (negative) or P (positive). This negativity or positivity indicates polarity. The N or P is followed by a number indicating the latency in milliseconds after the onset of a stimulus (Handy, 2005; Luck, 2005; Rugg& Coles, 1997). For example, N100 indicates that it is a negative wave occurring 100 milliseconds after the onset of a stimulus.

Some of the most common ERP components include the N100, N200, P300, N400 and P600. The N100 component is referred to as the readiness potential. It is associated with preparation of motor movements. The N200 component is seen following visual and auditory stimuli. The P300, N400 and P600 components are associated with cognitive and language processes. The P300 is associated with sensory stimuli and has been linked to cognitive tasks. A late occurring P300 component is associated with an attentional response. The N400 component is associated with intact semantic processing and is distributed in the centro-parietal electrodes. The P600 is indicative of syntactic processing and has been observed in studies of syntactic violations. Like the N400 component, it is distributed along the centro-parietal electrodes (Freiderici et al., 1999; Handy, 2005; Hohnsbein et al., Luck, 2005).

In summary, ERP provides real time information about the brain's response to internal and external stimulus on a millisecond-to-millisecond time range and because it can be time locked to a specific event it provides continuous measurement of processing. Since ERP can be obtained even when no behavioral response is required, it makes them appropriate for use in the study of language disorders. As such, it is a better measure of recovered brain function in individuals with aphasia.

COMPREHENSION OF SPOKEN LANGUAGE

Processing of auditory speech signals requires that the brain access prior knowledge such as words and meanings in the millisecond time range with the goal being to comprehend the meaning of the spoken message. In order to achieve this goal, the individual must access the

semantic representational system at a high rate (Friederici&Kilborn, 1989; Hagoort, Wassenaar& Brown, 2003).

Auditory Comprehension in Individuals with No Brain Damage

Studies of auditory comprehension in individuals with no brain damage are valuable because the information they provide can be used to recognize abnormal patterns in populations with brain damage. Auditory comprehension in individuals with no brain damage has been studied extensively using neuroimaging techniques such as PET, fMRI and ERP. One such study was completed by Demonet, Chollet, Ramsay, Cardebat, Nespoulous, Wise, Rascol and Frackowiak (1992). PET was use to investigate the functional anatomy of language comprehension. Nine participants with no brain damage were asked to complete three tasks. The first task required that the participants detect rising pitch within a series of pitch tones. The second task required the participants to judge the sequential phonemic organization of non-words. In the final task, participants were asked to judge concrete nouns according to semantic criteria. Results show that phonological processing activated the left superior temporal gyrus with some degree of activation in the right superior temporal gyrus. Of specific interest is the finding that lexical semantic processing activated the left middle and inferior temporal gyri, the left inferior parietal region and the left superior prefrontal region as well as the superior temporal areas.

In another study on individuals with no brain damage, Zatorre and Belin (2001) propose that the right hemisphere is specialized for pitch processing while the left hemisphere is specialized for processing of speech signals. rCBF (regional cerebral blood flow) patterns were examined in six males and six females with no brain damage using pure tone patterns that varied in frequency and duration. The pure-tone stimuli were presented via earphones inserted in the ears. The participants were instructed to listen to the pure-tone stimuli during two conditions. During the spectral condition, the number of frequencies was varied while in the temporal

condition, the rate of presentation was varied. Results show activation of the left and right Heschel'sgyri during the temporal condition.

Belin, Masure and Samson (1998) used synthesized sounds to demonstrate that temporal processing is localized in the left hemisphere in the brain of individuals with no brain damage. In this study, two groups of sounds were used. One group of sounds contained rapid formant transitions (40 ms) similar to those of speech sounds that were not pronounceable. The other group of sounds was identical except that the formant transitions were extended to 200 ms.rCBF patterns were examined during participant differentiation of sounds. Both sets of sounds activated the right and left hemisphere. However, the left hemisphere had larger areas of activation. Additionally, the sounds with the formant transition of 40 ms activated the left dorsolateral prefrontal region while the group of sounds with the formant transition of 200 ms activated both the right and left superior temporal gyri. The findings reported in this study suggest that temporal processing is localized in the left hemisphere.

In an fMRI study of individuals with no brain damage, Friederici, Ruschemeyer, Hahne and Fiebach (2003) used fMRI to identify the anatomical brain areas specific to processing of semantic and syntactic linguistic information. Fifteen males with no documented history of brain damage between 23-30 years participated in the study. A list of short sentences that were semantically and syntactically correct and incorrect was presented to the participants. The participants were asked to make judgments as to the correctness or incorrectness of the sentences. Correct response rate was high for all conditions including semantically correct, syntactically correct, semantically incorrect and syntactically incorrect for all participants. fMRI results show that semantically correct sentences resulted in increased activation along the superior temporal gyrus bilaterally, mid portion of the superior temporal gyrus and the insular cortices bilaterally. Syntactically correct sentences activated the superior temporal gyrus bilaterally. In the left hemisphere, activation was seen in the mid portion of the superior temporal gyrus, lateral to Heschel'sgyrus in response to syntactically correct sentences.

ERP studies have demonstrated that semantic processing in individuals with no brain damage may be reflected in the N400 ERP component. The N400 waveform was first observed by Kutas and Hillyard (1980). Participants were asked to read seven word sentences in which the last word was 75% congruous and 25% incongruous. The N400 waveform with a centroparietal distribution was elicited in response to the anomalous word.

Semantic processing using ERP was examined by Salmon and Pratt (2002). This study examined the N400 in response to sentences and stories that were semantically congruous and incongruous in eighteen participants with no brain damage. The task required that the participants judge whether spoken sentences and stories were logical and appropriate or not by pressing a button. The participants exhibited large N400 peaks for congruous sentences and stories than for the incongruous ones. These results are consistent with the results found by Kutas and Hillyard (1980).

Neville, Nicol, Barass, Forster, and Garret (1991) examined forty adults with no brain damage. The participants were instructed to read sentences that were semantically and grammatically congruous or incongruous. Result show that semantic violations elicited an N400 ERP component with cortical distribution over the temporal and parietal regions of the left hemisphere. The results of this study further support the findings of other studies that found that the N400 is elicited in response to semantic violations.

Summary

Evidence from neuroimaging studies of individuals with no brain damage show that semantic processing is localized primarily in the superior temporal gyrus in the left hemisphere. Findings from ERP studies indicate that the N400 component is elicited in tasks of semantic processing and that semantic violations elicit large N400s with a centroparietal distribution. ERP studies of individuals with no brain damage can help to distinguish normal from abnormal patterns.

Auditory Comprehension in Aphasia

It is well known that auditory comprehension is impaired in aphasia regardless of the type of aphasia. The extent to which it is impaired varies. Auditory comprehension deficit is usually associated with damage to the area in the posterior part of the superior temporal gyrus of the left hemisphere known as Wernicke's area (Darley, 1982).

Patients with auditory comprehension deficits demonstrate inability to recognize single spoken words, identification of objects and/or pictures and/or confusion of words that are closely related in sound or in meaning. In addition, comprehension of sentence length messages is difficult and details contained within the sentence may be lost. This is especially evident as the length and complexity of the sentence increases (Darley, 1982).

Several hypotheses have been put forth to account for the auditory comprehension deficits in aphasia. These deficits are hypothesized to be related to the difficulty that individuals with aphasia have in rapidly accessing the meaning of words, lost or delayed access to stored linguistic information, delayed analysis of the grammatical relationship between the words and/or difficulty integrating the two system (Swabb, Brown &Hagoort, 1997).

Behavioral methodology has been used to examine auditory comprehension deficits in aphasia. Pashek and Brookshire (1982) examined whether rate of speech and linguistic stress influenced the processing of paragraphs presented orally. Participants included eight individuals with no brain damage and twenty individuals with aphasia. Individuals with aphasia were assigned to either a low comprehension or high comprehension group based on their performance on a shortened version of the Token Test (De Renzi&Vignolo, 1962).

Participants were instructed to listen to twelve paragraphs that contained eight sentences with 10-15 facts (names, places, numbers and ideas). The participants were instructed to respond to sixteen yes/no questions that test eight facts presented in the paragraph. Four different paragraph conditions were tested including slow rate (120 words per minute), slow rate and normal linguistic stress, normal rate (150 words per minute) and exaggerated linguistic stress and normal rate and normal linguistic stress. Results show that rate of speech and linguistic

stress did not affect performance of the participants with no brain damage. The participants with no brain damage performed better than the high or low comprehenders. Additionally, slow rate and exaggerated stress improved comprehension of paragraphs in the high and low comprehenders. Furthermore, results show that number of correct responses was higher with exaggerated stress.

Nicholas and Brookshire (1986) examined whether rate of speech had an effect on comprehension, whether comprehension depended on the main idea or details of the information and whether the effects of speech rate depended on stated or implied details.

Three groups consisting of high auditory comprehension, low auditory comprehension and right hemisphere damage subjects were examined. Subjects were asked to listen to ten stories that contained approximately 13-14 sentences and answer yes/no questions about the stories. Subjects in the low comprehension group performed lower than the other two groups. However, this group was able to comprehend main ideas better when slow rate of speech was used to present the information. Results also show that individuals with aphasia improved performance in comprehension of stated details when slow rate of speech was utilized to present the stories and that performance was consistent across two testing sessions. However, the results of this study also indicate that while group results remained consistent across two testing sessions, variability in individual results were seen suggesting that all individuals with aphasia may not benefit from slow rate of speech. Nonetheless, these results show that slow rate of speech may improve auditory comprehension in some individuals with aphasia.

ERP has been used to examine auditory comprehension in individuals with aphasia due to its non-invasive nature and its ability to assess participants during active engagement. In addition, since ERP can be time locked to specific events, this methodology lends itself for the study of auditory comprehension in individuals with aphasia.

For example, Friederici and Kilborn (1989) used ERP to examine the temporal restrictions associated with language processing in Broca's aphasia. Two experiments were run in which cross modal syntactic priming was used. A syntactic prime consisting of an

auditoriallypresented sentence fragment and a visually presented word were used in both experiments. The experiments differed in the ISI (interstimulus interval). One of the experiments had an ISI (interstimulus interval) of zero ms while the second experiment had an ISI of 200 ms. The ISI was placed between the offset of the auditory prime and the visual target. Results show that when presented with auditory stimuli, the individuals with aphasia performed similar but much slower than participants with no brain damage. This delay was longer when the ISI of zero ms was placed between the auditory prime and the visual target. In addition, individuals with aphasia demonstrated decreased latencies and improved performance in the experiment that used a 200 ms ISI (interstimulus interval) placed between the auditory prime and the visual target. The implication of this study is that individuals with aphasia process auditory messages in a similar manner to the individuals with no brain damage but at a slower rate.

Swabb, Brown and Hagoort (1997) examined whether patients with aphasia have the capability of rapidly accessing lexical information in sentence context. Fourteen individuals with aphasia, six right-hemisphere lesion subjects without aphasia and twelve subjects without brain damage were asked to listen to sentences that had congruent and non-congruent endings with respect to the previous sentence context. The group with right hemisphere brain damage and no aphasia demonstrated a smaller N400 component with a centroparietal distribution similar to the participants with no brain damage. The group with aphasia demonstrated an N400 component that occurred at a latency of 300 – 700 ms indicating a delay in processing of the auditory message. The findings from this study are consistent with findings report by Friederici and Kilborn (1989).

Results from behavioral and ERP studies suggest that individuals with aphasia process auditory information similar to individuals with no brain damage but require extra processing time.

Auditory Comprehension of Single Words

Comprehension of single words has been well studied in individuals with no brain damage and individuals with brain damage using ERP, fMRI and PET. For example, Warburton, Price, Swinburn and Wise (1999) used PET to assess word retrieval and comprehension in six individuals with aphasia that had already demonstrated some degree of improvement in their aphasia syndrome. The task required that the individual with aphasia think of as many verbs as possible that were appropriate to the noun that was presented auditorially. The task was repeated six times with the experimental task scan and the resting scan being alternated.

Word retrieval results from individuals with aphasia were compared to the results of the individuals with no brain damage. The individuals with no brain damage produced an average of 18.4 words per minute while the individuals with aphasia produced from 12.3 to 17.6 words per minute. In addition, variable degrees of activation to the right hemisphere were observed but only in three of the six patients examined. Instead, activation of the left inferolateral temporal lobe was observed in all participants suggesting this area is specialized for semantic processing.

Zahn, Drews, Specht, Kemeny, Reith, Willmes, Schwarz and Huber (2004) used fMRI to examine auditory comprehension in seven patients with global aphasia. Three tasks were used. The first task required that the participant discriminate phonetically reversed words presented orally from signal correlated complex sounds. The second task was an auditory lexical decision task that required the participant to classify spoken words and non-words, in this case, the phonetically reversed words. The third task required the participant to discriminate animal names presented orally from names of other natural kind. Examination of the non-brain damaged individuals' fMRI scans revealed activation in the left prefrontal and posterior parietal areas. Visual examination of the scans completed on the patients with global aphasia showed no activation in areas associated with semantic processing; i.e. left prefrontal and posterior parietal areas. Furthermore, one of the individuals with aphasia exhibited activation in the right inferior frontal and perilesional left posterior middle superior temporal area while another demonstrated

activation of the right hemisphere (Zahn,et.al., 2004). These results suggest that semantic processing of single words may be attributed to the left prefrontal and posterior parietal areas.

Similar results were found by Zahn, Huber, Drews, Specht, Kemeny, Reith, Willmes, and Schwarz (2002). In this study, the left posterior superior middle and inferior temporal gyrus were activated during processing of congruous and incongruous words.

Single word comprehension has been studied using ERP. Nigam, Hoffman, and Simons (1992) compared pictures and words in order to elicit an N400 component. The participants were presented with sentences that were either all word sentences or sentences in which the last word of the sentence was a picture representing the same concept. Results show that an N400 with increased amplitude was exhibited for sentences that were all word sentences rather than for sentences that contained the picture.

In another study, ERP was used to assess comprehension of spoken words in individuals with aphasia. D'Arcy, Marchand, Eskes, Harrison, Phillips, Major and Connolly (2003) examined ten individuals with aphasia. Participants were presented with a picture that appeared on a computer screen. The picture was followed by a word that was either semantically congruent or incongruent to the picture presented. The words were presented auditorially via headphones. The visual and auditory stimuli used were similar to those used in the Peabody Picture Vocabulary Test-Revised (Dunn & Dunn, 1981). Participants demonstrated an N400 component in response to incongruent words. Examination of ERP waveforms revealed an N400 peak latency at 507 ms with a centroparietal distribution. A significant correlation between the spoken words from the PPVT-R (Dunn, 1981) and the N400 was found suggesting that the N400 is reflective of intact semantic ability.

Friederici and Kilborn (1989) found similar results as those presented by Hagoort, Wassenaar and Brown (2003). This study investigated the temporal constraints required for processing of linguistic information in participants with Broca's aphasia. Two experiments were conducted using sentence fragments that were presented auditorially and a target word that was presented visually. The two experiments were the same except that they differed in the length of

the ISI (interstimulus interval) that was inserted between the offset of the auditory stimulus and the onset of the visual stimulus. The first experiment used an ISI of zero ms while the second experiment had an ISI of 200 ms. Participants with Broca's aphasia performed better in the second experiment that had an ISI of 200 ms. Improved performance was seen with visually presented single words. These results suggest that individuals with Broca's aphasia demonstrate improved performance in lexical decision tasks when increased processing time is provided.

In a study on agrammatic processing of closed-class words in individuals with Broca's aphasia, results show a delayed onset of negativity. In addition, the wave pattern exhibited by the individuals with Broca's aphasia was different from the individuals with no brain damage and from the individuals with right hemisphere lesions (TerKeurs, Brown, Hagoort&Stegemen, 1999).

Evidence provided by neuroimaging studies on auditory comprehension show that activation is seen in the left hemisphere in response to single words. Additionally, ERP studies show that semantic violations elicit an N400 ERP component.

Auditory Comprehension of Sentences

While comprehension of single spoken words has been well studied, comprehension of spoken sentence length messages has not. However, some studies have examined comprehension of sentences using ERP. For example, Connolly, Mate-Kole and Joyce (1999) used ERP to examine whether a 27-year old male with global aphasia subsequent to a brain injury was able to comprehend sentences. The patient was presented with sentences that were either semantically appropriate or semantically incorrect. Sentences were presented visually and auditorially. Examination of the ERP waveform revealed a negative deflection occurring at 350-500 msec indicating that an N400 component was present. The presence of an N400 component suggests that the patient understood the semantic differences between the semantically appropriate and semantically inappropriate sentences.

Similarly, Hagoort, Wassenaar and Brown (2003), report that ERP is a good tool to assess sentence processing in individuals with agrammaticBroca's aphasia. Ten individuals with lesions in the left perysylvian area and diagnosed with aphasia and twelve individuals with no brain damage were presented with sentences that contained one or two syntactic violations. ERPs were recorded from 13 electrodes. Syntactic violations are expected to elicit a P600 ERP component but in this case, the P600 was not observed. Instead, the ERP waveform of the individuals with agrammatic aphasia showed an N400 component but with decreased amplitude when compared to the participants with no brain damage. Hagoort et. al., (2003) concluded that individuals with agrammatic aphasia may access the semantic representational system to improve performance in processing lexical information.

DeLong, Urback and Kutas (2005) examined whether individuals predict the upcoming word based on the preceding word using the articles (an) and (a) to process sentences. In this study, thirty-two participants were asked to read sentences at a rate of two words per minute. The task required that the participant read 160 sentences that contained 160 articles and 160 nouns.

The results show that as the noun became more predictable, the amplitude of the N400 decreased though the latency was longer than 500 ms after the onset of the stimulus. Similar results were obtained for articles though the amplitude of the N400 was much less than that of the N400 observed with nouns. The decrease in the amplitude of the waveform for articles was attributed to nouns being more semantically rich. Nevertheless, this data suggests that prediction may play a role in the comprehension of sentences.

Friederici, Yves von Cramon, and Kotz (1999) looked at sentence comprehension in a group of seven individuals with aphasia. The aphasic group was separated into two subgroups based on lesion sites. One group included patients with cortical lesions and the second group included patients with lesions in the basal ganglia. The task required patients to listen to sentences and press a button to indicate whether the sentence was correct or not.

Examination of the ERP waveform indicates that an N400 component with a mean latency of 300-600 ms was observed in both subgroups. The results of both aphasic groups were compared to results of participants with no brain damage. The individuals with the cortical lesions performed almost as well as the individuals with no brain damage while those in the basal ganglia group demonstrated an N400 component with decreased amplitude.

Swaab, Brown, &Hagoort (1997) used ERP to determine whether deficits in auditory comprehension of sentences were due to a delay in the integration of lexical information. Participants included seven individuals with Broca's aphasia, seven individuals with Wernicke's aphasia, twelve individuals with no brain damage and six individuals with right hemisphere damage. Participants were instructed to listen to congruous and incongruous sentences that were presented auditorially. ERP recordings were completed during the presentation of the sentences. The N400 ERP component was observed in all four groups. The individuals with Wernicke's aphasia exhibited negativity in the 500-700 ms time window after the onset of the stimulus while the individuals with Broca's aphasia exhibited the negativity between 300-1850 ms after the onset of the stimulus. The authors concluded that N400 was present in all groups but with delayed latency for individuals with aphasia suggesting that increased processing time is required to access lexical information.

Summary

A central question in studies of semantic processing in aphasia is whether individuals with aphasia exhibit difficulties with comprehension because they loose stored linguistic information or because they lack rapid access to the semantic representational system. A review of the literature presented here, suggests that individuals with aphasia are able to comprehend an auditory message in much the same way that individuals with no brain damage do but require additional time to process the spoken message.

The current study uses a modified version of the Revised Token Test (RTT) (1978) to assess auditory comprehension of spoken sentence length messages using ERP. Stimuli similar

to that of the RTT were chosen because each sentence contains semantic information that cannot be predicted. ERP can be time locked to a specific event thus monitoring electrical activity over time. Since ERP has a temporal resolution of approximately one millisecond it allows us to determine the point in time where processing occurs. The characteristics described above make ERP a reasonable choice for use in this study. ERP studies of sentence comprehension have demonstrated delayed latency of the N400 in tasks that use explicit semantic judgments. It is anticipated that ERP will elicit similar findings in the current study. However, this study uses a novel experimental procedure that requires an overt touching response in response to the spoken message. Review of the literature did not find any studies using an experimental procedure similar to the one used in the current study.

PILOT STUDY

A pilot study was carried out to assess equipment and test integrity. Five right-handed males with no documented history of brain damage were included in the control group. The mean age for the participants with no brain damage was 33.4 years. The individuals with aphasia included two right-handed females with diagnosis of left hemisphere stroke. Both participants were diagnosed with aphasia by a physician and assessed as having aphasia by a certified speech language pathologist during their inpatient rehabilitation stay. The mean age for the individuals with aphasia was 54 years. The individuals with aphasia in this study were 72 and 96 months post onset with a mean of 84 months. Pilot study participant characteristics are summarized in Table 2.3.

Table 2.3 Pilot Study Participant Characteristics

	Individuals with Aphasia	Participants with no Brain Damage
Sample size (N)	2	5
Left hemisphere stroke	2	-
Diagnosis of Aphasia	2	-
Auditory comprehension involvement	2	-
Mean post onset	84	-
Mean age (yrs.)	54	33.4
Gender	Female-2	Male-5
Handedness	2	5
Normal or corrected to normal vision	2	5
Passed hearing screening	2	5

The participants with no brain damage were asked to complete a modified version of the RTT that consisted of seven subtests with ten trials in each subtest. Each participant's head dimensions were measured and fitted with the "best fitted" electrode cap. Measurements were taken by measuring the head from nasion (bridge of the nose) to the inion (mid-occipital ridge of the head) and from the left and right preauricular point of the ears. The electrode cap size was selected based on the above-mentioned measurements. Once the electrode cap was selected, it was fitted on the participant's head. Electrodes were secured to the electrode cap. Each electrode was filled with Signa conduction gel to decrease impedance. Electrode placement was based upon the traditional 10-20 International System (Jasper, 1958; Handy, 2005; Rugg& Coles 1997).

Once the skullcap with electrodes was placed on the participant's head, they were escorted into a soundproof room and seated in front of a computer touch monitor. The participant was asked to attend to the white sample that appeared on the touch monitor. An example of the observational white sample is presented in Figure 2.1.

Fig. 2.1 Observational White Sample

The participant was asked to touch the white square in the middle of the black screen to initiate the task. Once the participant touched the white square, a spoken sentence length message was presented via speakers placed at 34 cm from the participant. Spoken messages increased in length and complexity as the test progressed. A display of choices appeared on the screen. An example of the visual display of choices is presented in Figure 2.2.

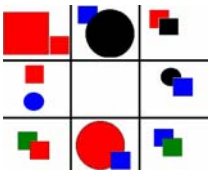


Fig. 2.2 Visual Display of Choices

The participant was instructed to listen to and respond to the spoken message by touching the

matching visual choice. An example of a spoken sentence length message with the matching visual choice is presented in Figure 2.3.

"Touch the big red circle and the little green square"

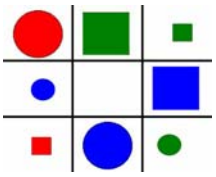


Fig. 3 Auditory Message with Matching Visual Choice

After the participants with no brain damage completed the test, feedback was provided to the principal investigator regarding fatigue experienced during the experimental task due to the length of the test. Based on this feedback, only the three subtests with the highest percent of correct responses were used with the individuals with aphasia. The three subtests were repeated three times resulting in ninety trials per participant. Trials were randomized to avoid memorization of the auditory messages. All other procedures remained the same as those used for the participants with no brain damage.

Data Collection and Analysis for Pilot Study

Correct responses and average reaction time were calculated using the stimulus presentation software program Superlab. Electrical activity was recorded from 64 electrodes using ActiveTwo from Bio Semi. ERP data was filtered and analyzed off-line with the Brain Vision Analyzer from Cortech Solutions (2008). Artifact rejection was completed in order to clean up the ERP signal. The sampling rate for filtering of data was set at 512 Hz. Artifacts included muscle artifacts, eye movements and eye blinks. Trials with any artifacts were rejected prior to averaging. Average waveforms were computed for each individual. A grand average was computed for each group.

Results of Pilot Study

Behavioral data showed that the average reaction time for the individuals with aphasia 2793.06 ms longer than that of the participants with no brain damage. The response accuracy for the individuals with aphasia was approximately 13% below that of the participants with no brain damage. The behavioral data suggests that the group with aphasia performed different from the participants with no brain damage. Behavioral data is summarized in Table 2.4. Electrophysiologic data is presented in Figure 2.4.

Table 2.4 Pilot Study Behavioral Results

	Reaction Time (msec)	Response Accuracy (%)
Participants with no brain damage (N=5)	5733.28	99.15
Individuals with Aphasia (N=2)	8526.34	86.46

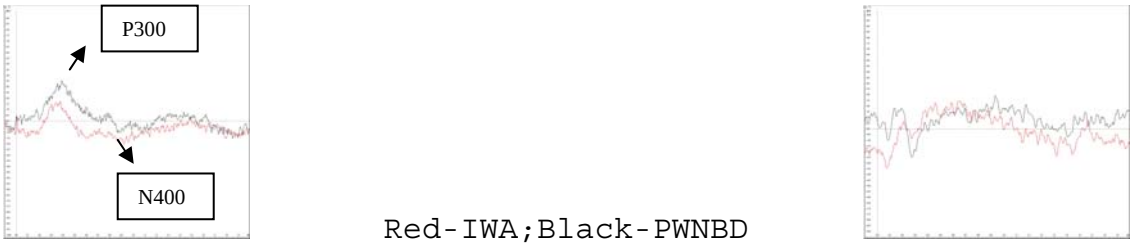
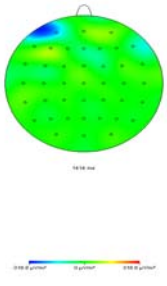


Fig.. 2.4 ERP Wave Form for the Participants with no Brain Damage and the Individuals with Aphasia

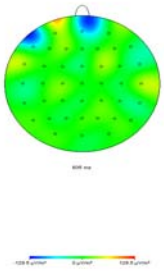
Electrophysiologic data shows that a similar pattern between the participants with no brain damage exists across the sample. The participants with no brain damage exhibited a negative deflection with latency at 400-600 ms suggesting the presence of an N400 ERP component. This suggests that the test is producing a semantic reaction. The waveform for the individuals with aphasia shows a delayed onset of negativity suggesting that no clear N400 ERP component was present.

In addition, there is a consistent pattern of a high P300 for the group with no brain damage. The ERP waveform for the individuals with aphasia showed a consistent deviation from the participants with no brain damage. This includes a consistent pattern of delayed positivity and low amplitudes suggesting that no true P300 ERP component was present.

Topographic maps were derived in order to localize the ERP signal source relative to the electrode placement on the scalp. Topographic maps were obtained for T7 (electrode is located on the left temporal area) and T8 (electrode is located on the right temporal area). Figure 2.5 illustrates the topographic maps for the two groups.



Participants with no Brain Damage



Individuals with Aphasia

Figure 2.5 Topographic Map

The topographic map for the participants with no brain damage shows cortical activation in the frontal, right and left temporal and parietal areas. In contrast, the topographic map for the individuals with aphasia shows cortical activation patterns that are different from the participants with no brain damage.

Summary

Several issues were identified during the pilot study. Feedback provided by the participants with no brain damage regarding fatigue due to the length of the experimental test resulted in a reduction of subtests for the individuals with aphasia. The results of the pilot study suggest that individuals with aphasia and participants with no brain damage perform differently in tests of semantic processing. Pilot study results were considered preliminary due to the limited number of participants and lack of statistical data. The decision was made to conduct an experimental research study that increases the sample size for the two groups to investigate auditory comprehension of spoken sentence length messages in individuals with aphasia and participants with no brain damage.

Chapter 3 Methodology

The purpose of this quantitative study is to compare cortical electrophysiologic and behavioral activity individuals with aphasia and participants with no brain damage responding to sentence length spoken messages. Review of the literature provides evidence that auditory comprehension of single words has been well studied. The literature review also reveals that comprehension of spoken sentence length messages has not been well examined. The current study seeks to broaden the understanding of the processes that mediate the recovery of auditory comprehension of spoken sentence length messages in individuals with aphasia. In order to examine auditory comprehension we need to identify where in the temporal process the individual processes auditory messages. ERP measures electrical activity at the level of the cortex in response to internal and external stimuli over time because it can be time locked to an event. Therefore, it is a reasonable choice for use in the current study. In seeking to understand this phenomenon, I addressed several research questions: 1) What are the correct response rate differences between the individuals with aphasia and the participants with no brain damage responding to spoken sentence length messages? 2) What are the behavioral reaction time differences (measured from the onset of the spoken message to the time the participants touches the visual choice) between the individuals with aphasia and the participants with no brain damage responding to spoken sentence length messages? 3) What are the differences in peak amplitude and latency of the P300 and the N400 (deduced from ERP wave patterns) between individuals with aphasia and participants with no brain damage responding to spoken sentence length messages? 4) What are the differences in sites of cortical activation (deduced from topographic maps) in sites of activity between the individuals with aphasia and participants with no brain damage?

This chapter describes the study's research methodology, and includes discussions on the following areas: a) rationale for the study, b) study design, c) research sample, d) data collection and analysis and e) limitations of the study.

RATIONALE

Auditory comprehension deficits are present in all aphasia types to varying degrees. It is important to study recovery of auditory comprehension in aphasia due to the financial and quality of life implications associated with it. Understanding the functional processes that are at work during the recovery of auditory comprehension has the potential for treatment implications such as development of better treatment strategies and other clinical decisions such as candidacy for treatment as well as length and duration of treatment. In addition, the literature provides evidence to suggest that ERP is a useful tool to examine cognitive processes since it can be time locked to a specific event and provides temporal resolution in the millisecond time range. In order to examine the functional and temporal processes that mediate auditory comprehension of spoken sentence length messages we need to identify where in that temporal process, semantic processing occurs. Based on characteristics such as excellent temporal resolution and ERP's ability to monitor cognitive processes over time, it is a reasonable choice for use in the current study.

DESIGN OF STUDY

This study compares two groups of different individuals that are not randomly assigned. Therefore, this study is a quasi-experimental design. The groups consisted of six individuals with aphasia and included two participants from the pilot study. The participants with no brain damage consisted of twelve individuals and included five participants from the pilot study.

The independent variables are the individuals with aphasia and the participants with no brain damage. The dependent variables are the latency, amplitude of the N400 and P300, response reaction time and response accuracy. The N400 component was defined as occurring between 350 to 650 ms post stimulus onset. The P300 was defined as occurring between 250 and 350 ms post stimulus onset (Friederici et al., 1989; 1999; Handy, 2005; Luck, 2005).

RESEARCH SAMPLE

Recruitment

All participants were recruited from the El Paso area. Written informed consent was obtained from all participants under the provision of the University of Texas El Paso (UTEP) Institutional Review Board (IRB). Participants were selected based on the following inclusion and exclusion criteria. Inclusion criteria for the experimental group included:

(a) diagnosis of left hemisphere stroke;

The medical record for each perspective participant was obtained and reviewed for evidence supporting diagnosis of left CVA. Evidence included but was not limited to CT scan or MRI previously reviewed by a physician and/or statement of left CVA diagnosis by a physician on the history and physical of the medical record. Participants were asked to complete a self-report medical screening questionnaire (Appendix A) to ensure that the participant was healthy and able to participate in the study.

(b) diagnosis of aphasia;

The medical record for each perspective participant was obtained and reviewed for evidence supporting diagnosis of aphasia. Evidence included documentation and/or statement of aphasia diagnosis made by a physician and/or speech language pathologist.

(c) normal or corrected-to-normal vision;

(d) normal or corrected-to normal hearing;

Inclusion criteria for normal participants included:

(a) no documented history of brain damage;

(b) normal or corrected to normal vision;

(c) normal or corrected to normal hearing;

Table 3.1 Participant Inclusion and Exclusion Criteria

	History of Left (CVA)	Diagnosis of Aphasia	Normal or Corrected to Normal Vision	Normal or Corrected to Normal Hearing	No documented history of brain damage
Individuals with Aphasia	x	x	x	x	
Participants with no Brain Damage	.	.	x	x	X

Sample

Participants for this experiment are six English-speaking individuals with diagnosis of left CVA (cerebrovascular accident) and aphasia. These participants were assigned to the experimental group. The control group includes twelve English-speaking individuals with no documented history of brain damage.

METHODS

Participants were tested individually in the Voice, Brain and Language Laboratory in the Speech Language Pathology Program of the College of Health Sciences at the University of Texas at El Paso. The experimental test used in this project was a modified version of the RTT. The modifications were done so that the RTT could be used in an ERP experiment. The RTT

framework was selected because it uses semantic information that cannot be predicted. In addition, the RTT has well established validity and reliability measures (McNeil & Prescott, 1978). Therefore, it was a reasonable choice for use in an ERP experiment that examines auditory processing of spoken sentence length messages.

Stimuli

The purpose of this study is to compare cortical electrophysiologic and behavioral activity in individuals with aphasia and participants with no brain damage responding to spoken

sentence length messages. Therefore, several modifications of the RTT were necessary. The modifications are described below.

The first modification was the mode of presentation. The RTT is a behavioral assessment measure that presents visual stimuli on a tabletop using a 4 X 5 matrix. The visual stimuli consist of plastic tokens of two different sizes (big and little) and two different shapes (circles and squares). These plastic tokens are of different colors (red, black, blue, green, and white). The patient is required to touch or move the plastic tokens in response to a spoken command that is presented by the examiner.

For this study, the visual stimuli were presented on a touch monitor using a 3 X 3 matrix. The visual stimuli were presented using the Superlab Stimulus Presentation Software (Superlab, 2008). The spoken messages used in this study were computer generated and presented in order to ensure consistency with intensity, prosody and rate of presentation across the entire testing session.

The RTT consists of ten subtests with ten trials in each subtest. The modified version used in this study consisted of seven subtests with ten trials in each subtest. However, based on feedback gathered during the pilot study only the three subtests with the highest percent of correct responses were used in the current experiment. The feedback was reported by the individuals with no brain damage. The pilot study was carried out to assess equipment and test integrity.

AUDITORY STIMULI

The auditory stimuli used in the current study consist of spoken messages that vary in length and grammatical complexity. The messages contain five different syntactical units that include prepositions (directional, spatial), adjectives (size, color), nouns (circle, square), verbs (touch, put) and one conjunction (and). Examples of the spoken messages are “Touch the black circle”, “Put the big red square in front of the big white circle” and “Touch the red square and the blue circle. Auditory commands were presented at 77-79 db SPL measured off speakers using a

Radio Shack Sound Level Meter. Speakers were placed at 34 cm from the participant. See Appendix B for a complete list of the spoken messages.

VISUAL STIMULI

The visual stimuli in the current study were created using Microsoft Format Painter and saved as "j-peg" files for use with Superlab Stimulus Presentation Software (2008). There are two different visual stimuli in this experiment. These include an observational white sample and a visual display of choices. The observational white sample is presented as a white square in the middle of a black screen. The observational white sample is designed to ensure the participants are oriented to the monitor and initiates the beginning of a trial. An example of an observational white sample is shown in Figure 3.1.

Fig. 3.1 Observational White Sample

The visual display of choices consists of five different colored (red, black, green, black, white) two shapes (circles, squares) of two different sizes (big, little) that are presented on a 3 X 3 grid. An example of a display of choices is shown in Figure 3.2. See appendix C for a complete list of the visual stimuli.

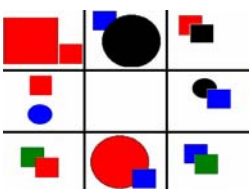


Fig. 3.2 Visual display of choices

EXPERIMENTAL PROCEDURE

The experimental procedure is described below. The participant was:

1. seated comfortably in a 6 X 6 soundproof room in front of the EntuitiveTouchmonitor.

2. instructed to place his/her hand on a mark on the edge of the table centered with the monitor at the start of the procedure. The mark is placed at a distance of 34 cm from the touch monitor.

3. instructed to return his/her hand to the mark after touching the visual display appearing on the monitor.

4. instructed to only move the hand to touch the screen when either a white sample or visual display of the choices appeared on the monitor.

5. instructed to look at the white sample (Fig.3.1) that appeared on a black screen on the monitor.

6. instructed to touch the white sample (Fig. 3.1) to initiate a trial.

7. instructed to listen to the auditory message (Appendix B) and respond to the message by touching the visual choice (Appendix C) that matches the auditory message. This procedure is repeated for each trial and each participant.

Events

A trial is composed of the following events. These events are described in the sequence in which they occurred within the trial. The observational white sample (Fig.1) was displayed on a black screen. The participant touched the white sample.The spoken message (Appendix B) was presented immediately after the participant touched the white sample.This was followed by a blank screen displayed for 1000 ms.Immediately following the 1000 ms the visual display of choices (Fig. 2) was displayed on the screen. The participant responded by touching the appropriate cell that contained the choice that matched the auditory message.Immediately following the participant's response, the screen went blank for 3000 ms.At the end of the 3000 ms, the observational white sample was displayed on the black screen initiating the next trial.

This cycle was repeated for each trial. There were ten trials per grammatical level of complexity. Each level of grammatical complexity was presented three times resulting in ninety trials per participant. Levels and trials were randomized for each participant to decrease the possibility that the participant would memorize the spoken messages. A schematic representation of a trial sequence is presented in Fig. 3.3.

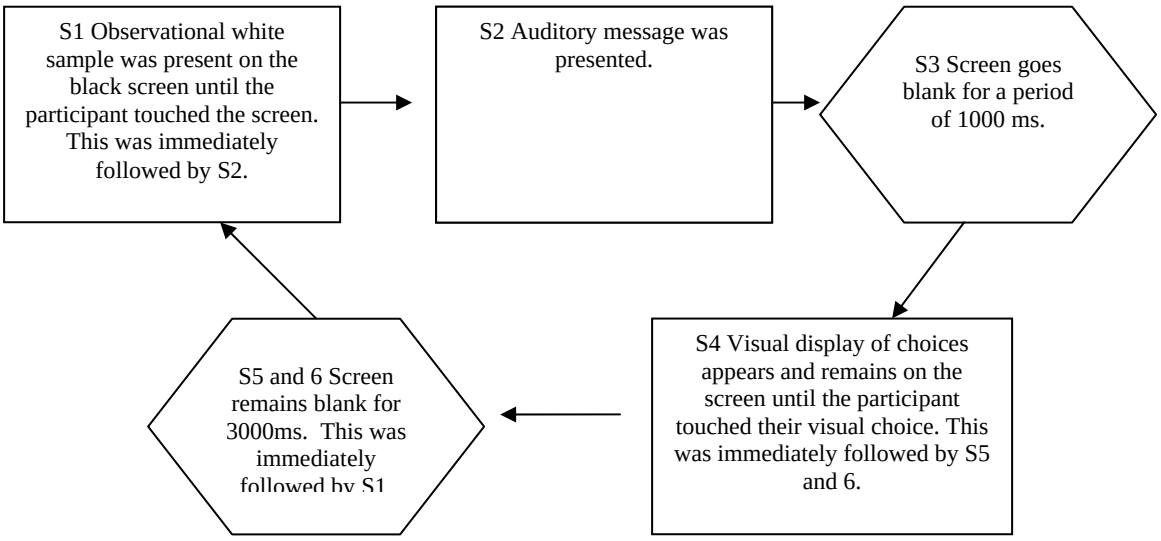


Fig. 3.3 Schematic representation of a trial sequence.

EVENT MARKERS

In order to measure time segments in an EEG waveform, event markers or triggers are placed at designated time windows within a trial. These time windows are the events contained in a trial. Event markers mark events within a trial and are time-locked to each event. The timeline for an individual trial begins with an event marker. For the current study, event markers were assigned as follows.

The event markers did not appear on the touch screen monitor but were marked on the ERP recording for measurement purposes. S1 marked the beginning of a trial (observational white sample). The S refers to an event marker used for recording purposes. S1 was followed by

S2. S2 marked the onset of the auditory message. S3 marked the onset of an ISI (interstimulus interval) that lasted for 1000 ms. S4 followed and marked the onset of the visual choices. This was followed by S5 and S6. S5 marked an ISI that lasted 1500 ms. This was followed by S6 that marked another ISI that lasted for 1500 ms and indicated the end of a trial. Following S6, the next trial began with S1indicating the beginning of a new trial. Figure 3.4 illustrates a schematic representation of the timeline sequence for an individual trial with corresponding events.

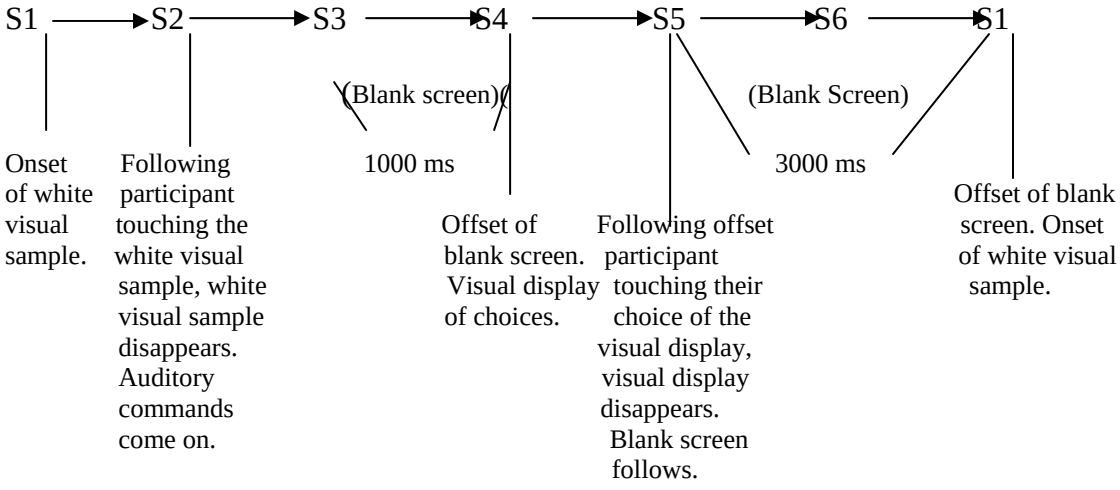


Fig.. 3.4 Schematic representation of the timeline for a trial with corresponding triggers (event markers)

ELECTROPHYSIOLOGIC METHOD

Each participant's head dimensions were measured and fitted with the "best fitted" electrode cap. Measurements were taken by measuring the head from nasion (bridge of the nose) to the inion (mid-occipital ridge of the head) and from the left and right preauricular point of the ears. The electrode cap size was selected based on the above-mentioned measurements. Once

the electrode cap was selected, it was fitted on the participant's head. Electrodes were secured to the electrode cap. Each electrode was filled with Signa conduction gel to decrease impedance. Electrode placement was based upon the traditional 10-20 International System (Jasper, 1958; Handy, 2005; Rugg& Coles 1997).

DATA COLLECTION

Several steps were taken to collect data from the ERP waveforms. The steps are outlined below.

ERP Recordings

Electrical activity was recorded from the scalp with 64 electrodes placed according to the International 10-20 System. Two reference electrodes were placed on the left and right mastoids. Assuch, all electrodes were referenced to the mastoids. Vertical and horizontal eye movement were tracked using four additional electrodes placed on the lower outer canthi and the orbital ridge of the right eye as well as the right and left temple (Handy, 2005; Jasper, 1958; Rugg& Coles 1997).

Electrical signals were recorded from the 64 scalp locations with a custom software program called ActiveTwo from Bio Semi. The electrodes transmitted electrical signals at a sampling rate of 2048 Hz. Bandpass was set at 0.1 Hz for the low cut off with a 12 dB slope, and a high cut off at 30 Hz. The notch filter was set at 60 Hz.

DATA FILTERING

ERP data was filtered and analyzed off-line using the Brain Vision Analyzer from Cortech Solutions (2008) with the sampling rate set at 512 Hz. Artifacts in the ERP wave were eliminated using the filtering feature of Brain Vision Analyzer (2008) before the data was averaged. Artifacts are defined as noise or electrical activity that was not associated with the stimuli. The most common sources of artifacts are eye blinks and/or eye movements (Handy, 2005; Luck, 2005). A notch filter was used to filter out noise coming from electrical power lines (Handy, 2005; Luck, 2005).

DATA SEGMENTATION

Segmentation is the separation of ERP data into temporal blocks. Segmentation was performed from event marker to event marker to create epochs or time windows. These time windows are used for individual trial analysis and averaging. Figure 3.5 shows a schematic representation of segmentation per trial.

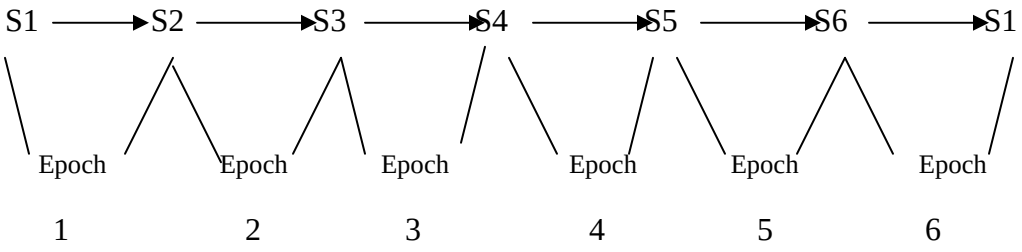


Fig. 3.5 Schematic representation of segmentation per trial.

DATA ANALYSIS

The number of correct responses were automatically recorded and saved to the computer using Superlab Software Program for later analysis for the individuals with aphasia and the participants with no brain damage. The mean behavioral reaction time (measured from the onset of the spoken message to the time the participant touches their visual choice on the touch screen monitor) as well as the mean latency and amplitude of the N400 and P300 were measured and analyzed for all participants.

Statistical Analysis

Correct response rate, behavioral reaction time (measured from the onset of the spoken message to the time when the participant touches their visual choice on the touch screen monitor), latency and amplitude of the P300 and N400 were calculated for the two groups and subjected to statistical analysis using SPSS version 19. Examination for distributional assumptions and potential outliners was completed for all variables. This was done by using frequency analysis, scatter plots and histograms. Kurtosis and skew were calculated to check for violations of normality. Inspection and analysis of missing data was completed in order to check for reliability. Outliers were corrected when required. Variables that violated distributional assumptions were corrected by using appropriate transformations. The results from the data transformations were checked.

Descriptive statistics were then performed and reported. Descriptive statistics include means and standard deviations using an Independent Samples T-Test to determine if performance variations were statistically significant between the two groups.

Analysis of ERP Data

Cognitive response latencies for the visual sample were determined by the occurrence of the P300 waveform. A P300 waveform was defined as a positive deflecting peak occurring 250-350 ms after the onset of the visual sample. Cognitive latencies for the choices were determined by the occurrence of N400 waveform. An N400 waveform was defined as the largest negative deflecting peak occurring 350-650 ms after the onset of the visual choices. Amplitude was measured by the voltage variations that occurred at the point where the P300 and N400 ERP peak latencies were measured.

ERP data was examined, corrected and analyzed using the Brain Vision Analyzer (Cortech Solutions, 2008). Sampling rate was set at 512 Hz using the Brain Vision Analyzer settings. Electrode channels were edited to eliminate unwanted noise from disturbing electrodes. All electrodes were referenced to the mastoids. EEG data was filtered at 0.1 Hz for the low cut

off and at 30 Hz for the high cut off. The notch filter was active at 60 Hz. Segmentation was performed from event marker to event marker to create epochs. Segmentation of S1 and S4 was performed to create two-second epochs (500 ms before stimulus onset and 1500 ms post stimulus onset) that facilitated analysis of the electrophysiologic data. Grand averages were calculated. Additionally, bad intervals were skipped.

Spatial Analysis

Spatial analysis was completed in order to generate cortical activation patterns. Spatial analysis is in the form of topographic (surface) maps. Topographic mapping allows for localization of the ERP signal source relative to the electrode placement on the scalp. Electrode Cz was utilized to derive cortical activation patterns because it is located centrally. This makes it the best electrode to illustrate overall activation. Topographic maps were generated at the P300 positive and N400 negative peaks.

HUMAN SUBJECT PROTECTION

Permission to conduct this study was obtained from the University of Texas at El Paso's Institutional Review Board prior to beginning the study. Participants were recruited from the El Paso Stroke Support Group, the UTEP Speech and Hearing Clinic, outpatient rehabilitation centers andindependent health care practitioners. Face-to-face meetings were used for recruitment purposes. The principal investigator maintained contact with participants via telephone or mail.

Once selected, participants were provided with verbal explanation as to the purpose of the investigation, all procedures, benefits and/or risks associated with the experiment. Participants were given an opportunity to read the informed consent and ask questions regarding their participation in the study as well as the research project itself. Participants were provided with an explanation as to their right to participate and/or withdraw from the project at any time. Once all questions were answered to the participant's satisfaction, they were asked to sign the informed consent.

Data collection, data entry and data analysis are the responsibility of the principal investigator. All data, including identifying information in both paper and computer format are kept in a locked cabinet in the Voice and Brain Laboratory in the Speech Pathology Department located in Campbell hall. Only the principal investigator and her faculty advisors have access to the data and the Voice and Brain Laboratory located at 1101 N. Campbell, El Paso, Tx. 709902. The door to the laboratory is locked and only the principal investigator and her faculty advisors have access to the key.

For added protection, electronic data is stored in a computer that may only be accessed by a password known only to the principal investigator and her faculty advisors. Data collected and used for this experiment will be stored five years after the termination of the study. Data will be destroyed after this five-year time-period.

Any information obtained from any participant was shared only between the principal investigator and her faculty advisors. This information was used only for the purposes of this research project. The Voice and Brain Language lab has a door that can be closed to maintain the participant's privacy when filling out forms and/or during preparation for the study. For added privacy, the Voice and Brain Language Laboratory is located in an area that can be closed off from others by an additional door that is locked. Only the principal investigator and her faculty advisors have access to the key for that additional door.

The principal investigator was responsible for collecting and analyzing the data during the length of this study. Participants were identified by a letter and number code that was also used to identify participant data. No video and/or auditory recordings were completed during this study.

There are no known risks associated with this research. Testing procedures were non-invasive. However, participants experienced mild fatigue during the testing situation. Participants were provided opportunities to rest if they voice complaints of fatigue. Participants may benefit from this study by knowing the outcome of their performance using event related potentials. The data obtained from this study may lead to development of improved evaluation

and treatment techniques for use in aphasia therapy. In addition, the data obtained in this study may result in the development of improved prognostic indicators.

There were no other specific sites or agencies, other than The University of Texas at El Paso involved in this research project. There were no other IRB approvals, other than The University of Texas at El Paso IRB requested for this project.

LIMITATIONS OF THE STUDY

A number of limitations were found in this study. Recruiting pathological participants is difficult. Therefore, the sample size for the experimental group was small. In addition, because recruitment of pathological participants is difficult, this study did not control for age, time post onset, site of lesion or gender. Finally, a participant fatigue was a limitation because prospective participants that felt fatigued during the initial phase of the procedure (taking head measurements for fitting of skullcap) dropped out of the study before completing the experimental task. Other participants provided feedback about fatigue during the experimental procedure. Participants A1 and A6 were provided with a five-minute rest break.

SUMMARY

The current study proposed to use Event Related Potentials to compare auditory comprehension of spoken sentence length messages in individuals with aphasia and participants with no brain damage. This study compared two groups of individuals; individuals with aphasia and participants with no brain damage. This is a quasi-experimental design since it lacked random assignment. The independent variables were the individuals with aphasia and the participants with no brain damage. The dependent variables were correct response rate, reaction time, latency and peak amplitude of the P300 and the N400 ERP components.

Sample size for the experimental group consisted of six adults with left-hemisphere stroke diagnosed with aphasia by a physician and certified speech language pathologist. Review of participant's medical record was used to verify diagnosis of left CVA and aphasia. The control group included twelve adults with no documented history of brain damage. Two of the

individuals with aphasia and four of the participants with no brain damage had previously participated in the pilot study.

A modified version of the RTT (McNeil & Prescott, 1978) was used to assess the participant's auditory comprehension of spoken sentence length messages. Electrical activity was recorded from 64 electrodes using Active-Two from BioSemi. Group means of correct response rate, reaction time, latency and peak amplitude were compared for performance variations.

Chapter 4 Results

The review of the literature shows evidence to support the hypothesis of cortical neuroplasticity and that recovery from aphasia occurs even months post onset. In addition, the literature shows that ERP is useful in the study of auditory comprehension in individuals with aphasia due to its non-invasive nature. In addition, it can be time-locked to a specific event allowing for continuous monitoring of cortical activity over time. The present study proposes to use ERP to compare cortical electrophysiologic activity in individuals with aphasia and participants with no brain damage responding to a spoken message. Behavioral responses will also be collected, measured and analyzed.

The aim of the current study was to compare cortical electrophysiologic and behavioral activity in individuals with aphasia and participants with no brain damage responding to spoken sentence length messages. A better understanding of the temporal characteristics underlying the mechanisms of auditory comprehension of spoken sentence length messages may contribute to the development of enhanced treatment strategies.

In seeking to understand the processes involved in the recovery of auditory comprehension in aphasia several research questions were addressed: 1) What are the correct response rate differences between the individuals with aphasia and participants with no brain damage responding to spoken sentence length messages? 2) What are the behavioral reaction time differences (measured from the onset of the spoken message to the time the participant touches their visual choice on the touch screen monitor) between the individuals with aphasia and the participants with no brain damage responding to spoken sentence length messages? 3) What are the differences of the peak latency and amplitude of the P300 and N400 ERP components (deduced from ERP wave patterns) between the individuals with aphasia and participants with no brain damage responding to spoken sentence length messages? 4) What are the site of activation differences (deduced from topographic maps) between the individuals with aphasia and participants with no brain damage responding to spoken sentence length messages?

DESCRIPTION OF PARTICIPANTS

Characteristics for the study participants are as follows. Individuals with aphasia included six individuals with left hemisphere stroke and aphasia with auditory comprehension impairment. The individuals with aphasia ranged in age from 34 to 71 years with a mean age of 55 years. Time post onset ranged from 16 to 120 months with a mean of 92.7 months. All individuals with aphasia were diagnosed with left hemisphere stroke and aphasia by a medical physician and this was verified through review of their medical records. The group included 3 (50%) males and 3 (50%) females. Individuals with aphasia were recruited from the community Stroke Support Group and outpatient rehabilitation clinics. The control group included twelve individuals with no documented history of brain damage. The participants with no brain damage ranged in age from 23 to 49 years with a mean age of 35.17 years. The participants with no brain damage included eight males (67%) and four females (33%). All participants indicated right-handedness on the medical history interview. The participants with no brain damage were recruited from the university and community groups. Two of the individuals with aphasia and four of the participants with no brain damage had previously participated in the pilot study. Participant characteristics are summarized in Table 4.1

Table 4.1 Participant Characteristics

	Individuals with Aphasia	Participants with no Brain Damage
Gender	3 males (50%) 3 females (50%)	8 males (67%) 4 females (33%)
Age	X=55	X=35.17

RESULTS

Statistical Analysis

Correct response rate per level of complexity was calculated for all participants. The data for correct response rate for all participants was collected using Superlab Stimulus Presentation Software. The data for the individuals with aphasia group was compared to the data from the

participants with no brain damage in an Independent Samples T-Test to determine if the individuals with aphasia performed statistically different from the participants with no brain damage. Behavioral results are summarized below. Means, standard deviations and results of the Independent Samples T-Test are summarized in Table 4.2.

Table 4.2 Correct Response Rate, Means, SD, Independent Samples T-Test

	X	SD	T-value	DF	P- value	Level of Significance .05
Level 1			2.82	5.12	.04*	<.05
Individuals with Aphasia	76.43	18.79				
Participants with no Brain Damage	98.15	2.86				
Level 2			2.83	5.20	.03*	<.05
Individuals with Aphasia	74.40	19.10				
Participants with no brain damage	96.67	3.73				
Level 3			2.83	6.07	.03*	<.05
Individuals with Aphasia	76.81	14.85				
Non-Brain Damaged Participants	94.81	6.78				

Significant (*); Non-Significant (NS)

There was a statistically significant difference in correct response rate between the two groups for level 3, 4 and 5 as determined by the Independent Samples T-Test. There was a statistically significant difference in the scores for level 3 correct response rate for individuals with aphasia (M= 76.4300, SD=18.78947) and participants with no brain damage (M=98.1492, SD=2.85554); t(5.116)=2.815, p=.036. There was a statistically significant difference in the scores for level 4 correct response rate for individuals with aphasia (M= 74.3983, SD=19.10056) and participants with no brain damage (M=96.6683, SD=3.73169); t(5.192)=2.829, p=.035. There was a statistically significant difference in the scores for level 5 correct response rate for individuals with aphasia (M= 76.8050, SD=14.85442) and participants with no brain damage (M=94.8092, SD=6.77692); t(6.065)=2.825, p=.030. The level of significance was .05. The null hypothesis is that all subjects will perform equally. The null hypothesis was rejected. Thus, the participants did not perform equally. The individuals with aphasia made more errors than the participants with no brain damage across the entire test. In addition, the participants with no

brain damage decreased their level of accuracy as the complexity increased while the individuals with aphasia maintained performance throughout the test.

Behavioral reaction times were calculated for all participants for every level of complexity. Behavioral reaction time was measured from the onset of the spoken message to the time the participant touched their visual choice on the touch screen monitor. Behavioral reaction times data for all participants was collected using Superlab Stimulus Presentation Software. The behavioral reaction time data for the individuals with aphasia was compared to the data from the participants with no brain damage in an Independent Samples T-Test to determine if the individuals with aphasia performed statistically different from the participants with no brain damage. Significance level was .05. There was a no statistically significant difference in the behavioral reaction time for level 3 scores for individuals with aphasia (M=6143.8850, SD=1520.09438) and participants with no brain damage (M=5590.6517, SD=401.84675); t(5.353)=-.876, p=.418. In addition, there was no statistically significant difference in behavioral reaction time for level 4 for individuals with aphasia (M= 6931.5267, SD=19.45982) and participants with no brain damage (M=6146.5792, SD=633.3310); t(5.537)=-.963, p=.376. There was no statistically significant difference in the behavioral reaction time for level 5 for individuals with aphasia (M= 4881.3283, SD=2164.69835) and participants with no brain damage (M=5425.8583, SD=13664.47188. 6.77692); t(16)=.657, p=.521. Reaction time means, standard deviation and results of the Independent Samples T-Test are summarized in Table 4.3.

The null hypothesis is that all subjects will perform equally. Based on the evidence at the alpha level of significance (.05) for reaction time, we failed to reject the null hypothesis. Thus, all participants performed equally.

Table 4.3 Means, SD, and Independent Samples T-Test

Reaction Time	X	SD	T value	DF	P value	Level of Significance .05
Level 1			-.88	5.35	.42 (NS)	<.05
Individuals with Aphasia	6143.89	1520.09				
Participants with no Brain Damage	5590.65	401.85				
Level 2			-.96	5.54	.38 (NS)	<.05
Individuals with Aphasia	6931.53	1945.45				
Partipants with no brain damage	6146.58	633.33				
Level 3			.66	16	.52 (NS)	<.05
Individuals with Aphasia	4881.33	2164.70				
Non-Brain Damaged Participants	5425.86	13664.47				

Significant (*); Non-Significant (NS)

Peak latencies and amplitudes were calculated for all participants for the P300 and N400 ERP components. The P300 component was expected to occur at the observational white visual sample at theinitiation of a trial. Peak latency for the P300 was defined as a positive deflecting peak occurring between 250-350 ms after the onset of the observational white visual sample.

Peak latency for the N400 component was defined as the largest negative deflecting peak occurring 350-650 ms after the onset of the visual choices. Peak amplitude was measured by the voltage variations that occurred at the point where the P300 and N400 ERP peak latencies were measured. The peak latency and amplitude for the P300 and N400 ERP components for the individuals with aphasia were compared to the data from the participants with no brain damage in an Independent Samples T-Test to determine if the individuals with aphasia performed statistically different from participants with no brain damage. Peak latency and amplitude means and standard deviations for the P300 and N400 are summarized in Table 4.4. Results of Independent Samples T-Test for peak latency and amplitude of the P300 and N400 are summarized in Table 4.5.

Table 4.4 Means and SD for Latency and Amplitude of the N400 and P300

	Mean	SD
Individuals with Aphasia		
Latency N400	765.17	250.78
P300	529.50	367.10
Amplitude N400	-1.98	1.29
P300	4.32	3.44
Participants with no brain damage		
Latency N400	725.58	384.69
P300	425.83	266.33
Amplitude N400	-1.41	1.38
P300	4.15	3.03

Table 4.5 Independent Samples T-Test Results for Latency and Amplitude of the N400 and P300

	T-value	DF	P-value	Level of Significance
Latency N400	-.23	16	.82 (NS)	<.05
Latency P300	-.69	16	.50 (NS)	<.05
Amplitude N400	.87	10.75	.40 (NS)	<.05
Amplitude P300	-.11	16	.92 (NS)	<.05

Significant (*); Non-significant (NS)

There was no statistically significant difference in peak latency of the N400 for individuals with aphasia (M=765.17, SD=250.783) and participants without brain damage (M=725.58, SD=384.691); t(16)=-.227, p=.823. There was no statistically significant difference in peak amplitude of the N400 for individuals with aphasia (M= -1.983, SD=1.291781) and participants with no brain damage (M=-1.408.5792, SD=1.380990); t(10.751)=.870, p=.403. There was no statistically significant difference in peak latency of the P300 for individuals with aphasia (M=529.50, SD=367.103) and participants with no brain damage (M=425.83, SD=266.39); t(16)=-.688, p=.501. There was no statistically significant difference in the peak amplitude of the P300 for individuals with aphasia (M= 4.31767, SD=3.436978) and participants with no brain damage (M=4.14667, SD=3.3033040); t(16)=.108, p=.915.

The null hypothesis is that all subjects will perform equally. Based on the results at the alpha level of significance (.05) for peak latency and amplitude of the N400 and P300, we failed to reject the null hypothesis. Thus, all participants performed equally.

Results of the Independent Samples T-Tests for peak latency and amplitude of the P300 and N400 made it necessary to examine individual participant data. Individual participant data for all participants for peak latency and amplitude of the P300 and N400 ERP component is summarized in Table 11. The absence of a statistically significant difference may have been

influenced by individual participant data. For example, participant NBD3 exhibited peak latency of the P300 at 1199 ms. Participant NBD6 exhibited peak latency of the N400 at 1299 ms. In addition, participant NBD9 exhibited delayed latency of the N400 at 1734 ms. To determine if the data from these participants influenced the absence of a statistically significant difference between the two groups for latency of the P300 and N400, an Independent Samples T-Test was completed without the data from those participants.

There was a no statistically significant difference in the peak latency of the N400 for individuals with aphasia (M=765.17, SD=250.783) and participants with no brain damage (M=567.40, SD=59.560); t(5.341)=-1.900, p=.112. There was no statistically significant difference in the peak latency for P300 for individuals with aphasia (M= 529.50, SD=367.103) and participants with no brain damage; (M=353.00, SD=89.456); t(5.326)=-1.159, p=.296.

The null hypothesis is that all subjects will perform equally. Based on the evidence at the alpha level of significance (.05) for peak latency and amplitude of the N400 and P300 and after excluding the outliers NBD 3, 6, and 9, we failed to reject the null hypothesis. Thus, all participants performed equally.

Table 4.6 Individual Participant Latency and Peak Amplitude of P300 and N400

	P300 Latency (ms)	P300 Amplitude (mv)	N400 Latency (ms)	N400 Amplitude (mv)
NBD1	342	3.227	615	-1.853
NBD2	381	9.735	609	-0.818
NBD3	1199	8.924	611	-0.732
NBD4	461	1.529	596	-2.026
NBD5	387	6.337	590	1.333
NBD6	275	2.969	1299	-0.400
NBD7	252	1.708	553	-1.810
NBD8	529	0.059	514	-4.083
NBD9	383	6.565	1734	-0.287
NBD10	357	3.098	434	-1.487
NBD11	252	2.964	533	-2.932
NBD12	264	2.645	619	-1.810
A1	391	8.559	1148	-0.875
A2	1227	3.482	881	-0.667
A3	465	0.843	557	-3.446
A4	402	1.588	451	-1.721
A5	402	8.627	699	-3.697
A6	252	2.807	855	-1.496

A=individuals with aphasia NBD=Participants with no brain damage

ms=milliseconds mv=microvolts

The results of this Independent Samples T-Test show that while some of the participants without brain damage had delayed latencies for the N400, there was no statistically significant difference between the two groups.

The latency and amplitude for the P300 and N400 were collected from electrode site T7 (located over the left temporal area) and T8 (located over the right temporal area) and analyzed for all participants. Means and standard deviations for the peak latency of P300 and N400 at electrode site T7 and T8 for both groups are summarized in Table 4.7. Independent Samples T-Tests results for peak latency at P300 and N400 at electrode site T7 andT8 are summarized in Table 4.7. The results of the Independent Samples T-Test for peak amplitude of the P300 and N400 at electrode T7 and T8 are summarized in Table 4.9. The results of the Independent Samples T-Test for the latency and amplitude of the N400 and P300 at electrode site T7 and T8 shows that a statistically significant difference between the two group means exists for the peak latency of the P300 at electrode site T7 (located over the right temporal lobe).

Table 4.7 Means and Standard Deviations P300 and N400 at electrode Sites T7 and T8

			Mean	SD
Individuals with Aphasia	P300 Latency			
		T7	1192.8	390.5
		T8	710.0	324.3
Participants with no brain damage	P300 Latency	T7	516.3	454.8
		T8	408.6	194.7
Individuals with Aphasia	N400 Latency			
		T7	1053.8	222.5
		T8	961.7	357.6
Participants with no brain damage	N400 Latency	T7	600.0	268.9
		T8	593.6	147.4

Table 4.8 Independent Samples T-Test results for latency P300 and N400 at electrode site T7 andT8.

Latency	T-value	DF	P-Value	Level of Significance
P300 (T7)	-3.11	16	.007*	<.05
P300 (T8)	-2.48	16	.024*	<.05
N400 (T7)	-3.56	16	.003*	<.05
N400 (T8)	-3.141	16	.006*	<.05

Significant (*); Non-Significant (NS)

Table 4.9 Independent Samples T-Test results for Amplitude P300 and N400

Amplitude	T-value	DF	P-Value	Level Of Significance
P300 (T7)	-.12	16	.90 (NS)	<.05
P300 (T8)	.16	16	.87 (NS)	<.05
N400 (T7)	1.11	16	.28 (NS)	<.05
N400 (T8)	1.26	16	.23 (NS)	<.05

Significant (*); Non-Significant (NS)

In addition, a statistically significant difference between the two group means was found for the peak latency of the N400 at electrode site T7 and T8.

The null hypothesis is that all subjects will perform equally. The null hypothesis was rejected. Thus, the participants did not perform equally. In addition, the results of the Independent Samples T-Test shows that while a statistically significant difference in peak latency was found, a statistically significant difference for amplitude was not. Specifically, these results suggest differences in sites of activation exist between the two groups.

The statistical analysis shows that the two groups performed different for correct response rate. There was no statistically significant difference for behavioral reaction time (measured from the onset of the spoken message to the time the participant touches their visual choice on the touch screen monitor), peak latency and amplitude of the P300 and N400 even when individual participant data was examined and outliers eliminated. A statistically significant difference was found for peak latency of P300 and N400 at electrode site T7 and T8 but not at electrode site Cz. In addition, a statistically significant difference for peak amplitude of the P300 and N400 was not found at electrode site T7 and T8 between the two groups.

The statistical analysis for the cortical electrophysiologic data on performance variations between the two groups based on level of grammatical complexity could not be completed because of the design of the experimental procedure. Levels and trials were randomized so that participants would not memorize the spoken messages. As a result, it was not possible to separate the levels of complexity when they were recorded by BioSemi. Therefore, the EEG for the levels of grammatical complexity could not be analyzed by Vision Analyzer.

ELECTROPHYSIOLOGIC DATA ANALYSIS

Peak latencies for the response to the spoken sentence length message were determined by the occurrence of N400 ERP component because those waves are reflective of semantic processing. An N400 ERP was defined as a negative deflection occurring at 350-650 ms after onset of the visual display of choices. Peak latencies for the observational white sample were determined by the occurrence of P300 ERP waveforms because those waves are reflective of attention. A P300 ERP component was defined as a positively deflecting peak occurring 250-350 ms after the onset of the visual sample. Amplitude was measured by the voltage variations that occurred at the point where the P300 and N400 ERP peak latencies were measured.

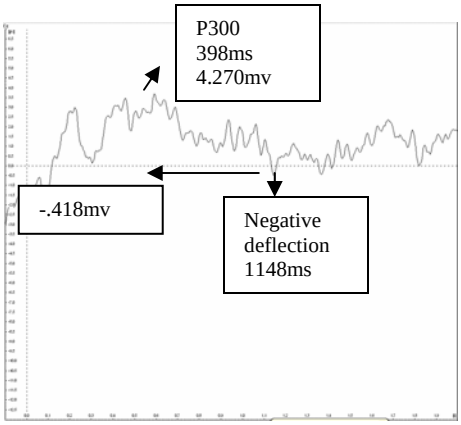


FIG. 4.1 ERP WAVEFORM SHOWINGAN N400
Individuals with aphasia
Latency at 1148 ms Amplitude -0.418 mv

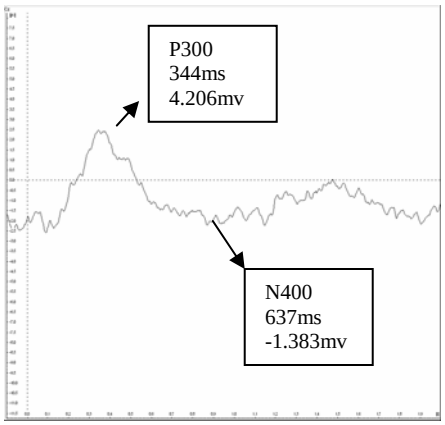


FIG. 4.2 ERP WAVEFORM SHOWING AN N400
Participants with no Brain Damage
Latency at 637 ms Amplitude -1.383 mv

The nature of activation also differed across the duration of each sentence in each of the two groups of participants. Each trial began with an observational white sample. The purpose of the observational white sample was to get the participant to attend to the screen and to signal the beginning of a trial. The participant was instructed to touch the observational white sample to initiate the trial. The start of a sentence was marked once the participant touched the white sample since this initiated thespoken message to be played. The P300 was expected to occur at this time window. Figure 4.1 shows the ERP waveform for the individuals with aphasia. As can be seen from this waveform, there is a delayed onset of the P300 and a delayed onset of the N400 component. In addition, the amplitude for the N400 component is -.418 mv. Figure 4.2 shows the ERP waveform for the participants without aphasia. As can be seen, the P300 occurred earlier than in individuals with aphasia. In addition, the N400 occurred at 637 ms post stimulus onset with larger negative deflection. To summarize, Figures 4.1 and 4.2 show that the individuals with aphasia showed delayed onset of positivity and reduced attentional activity at the start of each sentence as well as an increased latency and reduced amplitude when processing the sentence. The individuals with no brain damage showed quicker attentional response to the onset of each trial and a shorter latency and increased amplitude when processing the sentence. ERP waveforms for the N400 ERP component are shown in Figures 9a and 9b.

The individuals with aphasia exhibited slightly higher amplitude than the participants with no brain damage. The participants with no brain damage displayed a quicker attentional response within the time that the P300 was expected to occur. This suggests that the individuals with no brain damage exhibited a P300 ERP component.

In addition, the participants with no brain damage displayed a similar pattern across the sample suggesting that a semantic response had occurred. The participants with no brain damage exhibited negative deflections at 637 ms suggesting the presence of an N400 component. The individuals with aphasia exhibited negative deflection at 1148 ms with amplitude of -0.418 suggesting that a clear N400 component was not present. The ERP waveforms for the P300 measured at electrode site Cz are shown in Figures 10a and 10b.

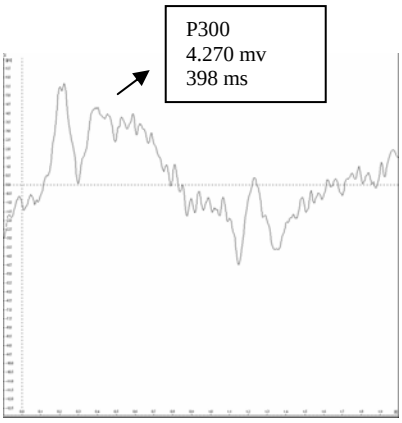


Fig. 4.3 Individuals with Aphasia
P300
Latency at 398ms Amplitude 4.270 mv

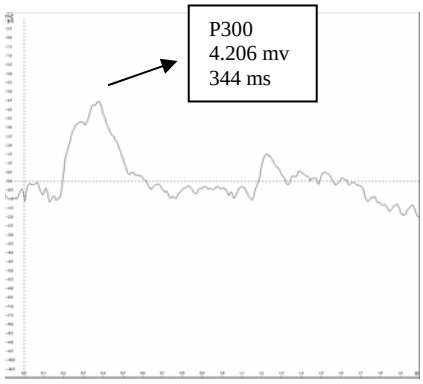


Fig. 4.4 Participants with no brain damage
P300
Latency at 344ms Amplitude 4.206 mv

The ERP waveforms seen in Figure 10a shows the P300 waveform for the individuals with aphasia showing a P300 waveform measured at Cz. Figure 10b shows an ERP waveform for the participants with no brain damage showing a P300 measured at Cz.

CORTICAL ACTIVATION PATTERNS:

Cortical activation patterns were derived from electrode Cz. This electrode was chosen

for its central location, which makes it one of the best single electrodes to illustrate overall cortical activation. Cortical activation patterns were observed using topographic CDS maps generated at the N400 peak (350-650 ms post stimulus onset) for the visual display of choices and the P300 peak (250-350 ms post stimulus onset) for the observational white sample.

Individual participant waves were averaged and CDS maps were generated based on the group average. Figure 4.5 and 4.6 illustrates the topographic maps for the two groups generated at the N400 peak latency (choice response). On the topographic maps, red areas indicate areas of high positive activation. Yellow areas indicate moderate positive activation whereas blue means areas of negative activation. Areas of neutral or minimal activation are shown in green.

The most significant observation is that the individuals with aphasia exhibited activation in the frontal, right and left temporal electrode sites at the N400 peak latency (choice response). The participants with no brain damage displayed some degree of activation in the left frontal electrodes. In addition, the non-brain damaged individuals exhibited negative activation in the central frontal electrodes. Figure 4.7 and 4.8 illustrates the topographic maps for the two groups generated at the P300 component.

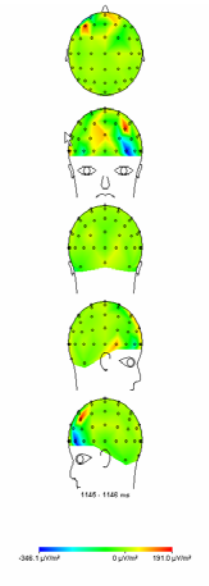


FIG. 4.5 INDIVIDUALS WITH APHASIA
N400
Latency at 1148 ms amplitude -0.418 mv

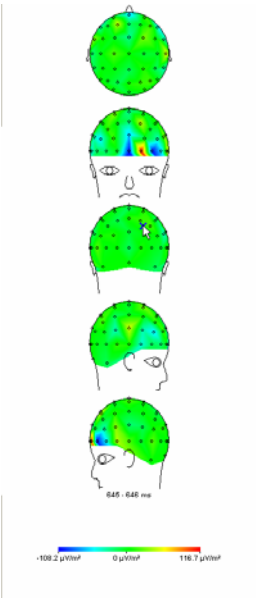


FIG. 4.6 PARTICIPANTS WITH NO BRAIN DAMAGE
N400
Latency at 637 ms amplitude -1.383 mv

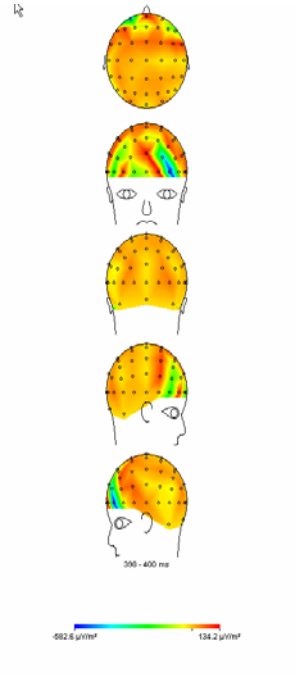


FIG. 4.7 INDIVIDUALS WITH APHASIA
P300 (Attentional Response)
Latency at 398 ms Amplitude 4.270 mv

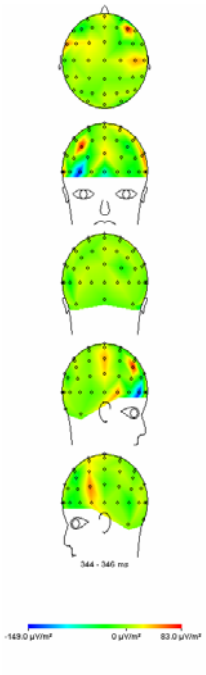


FIG. 4.8 PARTICIPANTS WITH NO BRAIN DAMAGE
P300 (Attentional Response)
Latency at 344 ms Amplitude 4.206 mv

The most notable observation is that individuals with aphasia exhibit high areas of activation throughout the cortex. This could be attributed to the individuals with aphasia recruiting all area available to compensate for damage to localized areas. The participants with no brain damage displayed a temporal parietal distribution.

SUMMARY

This chapter presents the behavioral and electrophysiological results from two groups of participants responding to spoken sentence length messages; individuals with aphasia and participants with no brain damage. The results show that the two groups performed differently in correct response rate. The individuals with aphasia made more error responses than did the participants with no brain damage. Behavioral results show that the participants with no brain damage showed reduced accuracy as the complexity of sentence was increased while the individuals with aphasia maintained the same level of accuracy throughout the test. Though no

statistically significant difference in peak latency and amplitude of the P300 and N400 components was found between the two groups, visual examination of the waveforms showed that the individuals with aphasia exhibited longer ERP latencies with smaller peaks than participants with no brain damage.

Since no statistically significant difference was found for peak latency and amplitude for the P300 and N400 measured at Cz, the peak latency for the two groups was measured and analyzed at electrode sites T7 and T8 to determine if a statistically significant difference was found. While, a statistically significant difference was not found for amplitude of the P300 and N400 measured and analyzed at T7 and T8, a statistically significant difference in peak latency was found between the two groups. This suggests that the two groups performed differently and that the individuals with aphasia may not be using the left temporal areas to comprehend spoken messages.

Cortical activation patterns differed between the two groups. The individuals with aphasia exhibit high areas of activation distributed throughout all electrodes for the P300 while the participants with no brain damage displayed activation in the frontal, right and left temporal electrodes. This suggests that the individuals with aphasia access all cortical areas to perform attentional tasks. The non-brain damaged individuals displayed some degree of activation in the left frontal electrodes for the N400 while the individuals with aphasia exhibited activation in the frontal, right and left temporal electrode sites. The pattern of cortical activity for the two groups support the results of a statistically significant difference in peak latency for the N400 at T7 and T8.

The nature of activation also differed across the duration of each sentence in each of the two groups of participants. The individuals with aphasia showed reduced attentional activity at the start of each sentence and an increased latency and reduced amplitude when processing the sentence. The participants with no brain damage showed quicker attentional response to the onset of each trial and a shorter latency and larger amplitude when processing the sentence. These

findings suggest that individuals with aphasia require increased processing time to comprehend a spoken sentence length message.

Chapter 5 Discussion

INTRODUCTION

The purpose of this quantitative study is to compare the cortical electrophysiologic and behavioral responses of individuals with aphasia and participants with no brain damage responding to spoken messages. An understanding of the temporal aspects involved in auditory comprehension may lead to development of improved assessment and treatment strategies for individuals with aphasia. In addition, the knowledge gained may broaden the understanding of auditory comprehension of spoken messages. The questions undertaken in this study are: 1) What are the correct response rate differences between the individuals with aphasia and the participants with no brain damage responding to spoken sentence length messages? 2) What are the behavioral reaction time differences (measured from the onset of the spoken message to when the participant touches his/her visual choice on the touch screen monitor) between the individuals with aphasia and the participants with no brain damage responding to spoken sentence length messages? 3) What are the differences in peak latency and amplitude of the P300 and the N400 ERP components (deduced from ERP wave patterns) between the individuals with aphasia and participants with no brain damage? 4) What are the differences in sites of cortical activation (deduced from topographic maps) between the individuals with aphasia and participants with no brain damage?

This chapter presents a discussion of the implications based on the data collected.

DISCUSSION

The current study showed that individuals with aphasia required additional time to comprehend a spoken message but given this additional time, they were able to respond with a moderate degree of accuracy. Additionally, the individuals with aphasia displayed a delayed onset of positivity suggesting a delayed P300. Similar results were seen in a study by Swaab, Brown and Hagoort (1997). Fourteen individuals with aphasia were examined in a task that required them to count low tones that were presented randomly along with high tones. The

individuals with aphasia were grouped for analysis according to their comprehension performance. The results show that the high comprehension group participants exhibited a delayed P300 with reduced amplitude. In contrast, the low comprehensiongroup participants did not display a P300 waveform. The results of the current study show that individuals with aphasia demonstrate delayed P300 with decreased amplitudes. This suggests that individuals with aphasia display delayed attentional responses. This is important because some evidence exists in the literature that suggests that poor attention may influence auditory comprehension problems in individuals with aphasia and that treating attention may improve auditory comprehension performance (Murray, Keeton, Karcher, 2006).

The results also showed that the individuals with aphasia demonstrated increased elctrophysiologic latencies for the N400. This has previously been reported in the literature (Swaab et. al., 1997). However, previous ERP studies on auditory comprehension of spoken messages have used explicit semantic judgment that requires the participant to decide whether sentences were congruous or incongruous. For example, Connolly, et. al., (1999) used ERP to examine auditory comprehension in a 27-year old male with global aphasia. The patient was presented with sentences that were either semantically appropriate or semantically incorrect. The participant was tasked with silently judging whether the sentences were appropriate or not. A latency of up to 500 ms was exhibited by the participant suggesting the presence of an N400 ERP component. Based on the explicit semantic and silent judgment and the ERP waveforms exhibited by the patient, Connolly et. al., (1999) concluded that the patient understood the semantic differences between the semantically appropriate and semantically inappropriate sentences. The current study added a novel procedure that required the participant to produce an overt respond to indicate understanding of the spoken message. Therefore, it is reasonable to believe that the novel experimental procedure contributed to the delayed electrophysiologic latencies displayed by the individuals with aphasia. Furthermore, behavioral results show that the individuals with aphasia maintained correct response rate across the levels of complexity but exhibited increased behavioral reaction times (measured from the onset of the spoken message to

the time the participant touched their visual choice on the touch screen monitor) as the complexity of the spoken messages increased. Increasing the complexity, may result in overtaxing the system thus resulting in delayed response latencies. Because the levels and trials were randomized so that the participants did not memorize the spoken messages, the cortical electrophysiologic latencies based on level of grammatical complexity for the P300 and N400 could not be measured or analyzed.

The statistical analysis showed that there was no statistically significant difference between the two groups for latency of the attentional (P300) and choice (N400) responses. Close examination of the data provides a possible explanation for the failure to find a statistically significant difference for latency of the P300 and N400. Individual participant data shows that one of the non-brain damaged participants, NBD3 demonstrated a latency for the P300 component at 1199 ms. Furthermore, non-brain damaged participant 6 exhibited a latency for the N400 at 1299 ms and non-brain damaged participant 9 displayed a latency for the N400 at 1734. These three participants behaved more like the individuals with aphasia than the other participants with no brain damage. In addition, one of the individuals with aphasia, BD4 exhibited a latency for the N400 at 451 ms while BD6 exhibited a latency for the P300 at 252 ms. These two individuals with aphasia, performed more like the participants with no brain damage. Given the small sample size, it is reasonable to believe that failure to find a statistically significant difference could have been influenced by the individual participant data.

The results of this study did not show a statistically significant difference for latency of the N400 exhibited at electrode site Cz. However, a statistically significant difference between the two groups was found for latency of the N400 at electrode sites T7 and T8. Electrode site T7 is located on the left side and sits over the temporal lobe. Electrode site T8 is located on the right side and sits over the temporal lobe. The cortical activation patterns of the individuals with aphasia showed more activation in the right temporal electrodes than the left. In addition, the cortical activation patterns seen in the left temporal electrodes in the individuals with aphasia were different from the participants with no brain damage. This phenomenon may

be attributed to the theory of neuroplasiticity. Reorganization of brain function has been reported in the literature (Basso, et. al., 1999; Vanlancker-Sidtis, 2004).

The presence of the N400 component in the participants with no brain damage suggests that a semantic reaction had occurred. This is a reaction commonly found in ERP studies of auditory comprehension in individuals with no brain (Kutas&Hillyard, 1984; Neville, et. al., 1991).

Finally, activation observed in topographic maps of the aphasic group differed significantly from the controls. The most significant observation is that the individuals with aphasia exhibited activation in the frontal and right temporal electrodes with some activation seen in the left temporal electrode sites measured at 1148 ms and derived from electrode Cz. Cortical activation patterns measured at 398 ms and derived from electrode Cz shows that individuals with aphasia had high areas of activation throughout all electrodes while the participants with no brain damage displayed a temporal parietal distribution. This could be attributed to the increased attentional demands required for this novel experimental procedure since a late occurring but high P300 was exhibited by individuals with aphasia. A high and late occurring P300 is significant of attention.

It should be noted that the ERP waveforms are averages of many individual trials and, as such important individual differences may be masked. In addition, these individual differences may influence the statistical analysis. For example, in the present study, individual differences exhibited by three participants with no brain damage. Participants with no brain damage may have influenced the results that yielded no statistically significant differences for reaction time, latency and amplitude of the N400 and P300 components.

Furthermore, such a small sample is not considered sufficient to override results such as the ones obtained in this current study. However, given the difficulties in recruiting pathological populations, this study was undertaken in order to increase the knowledge presently available in the literature regarding the auditory comprehension of spoken sentence length

messages. Therefore, future research should focus on replication of the study, increasing number of participants and examining complexity using ERP by redesigning the experimental procedure.

In summary, ERP was used in the current study to investigate the temporal characteristics of auditory comprehension of spoken sentence length messages in individuals with chronic aphasia. The results indicate that individuals with aphasia comprehend the spoken message but require increased processing time.

IMPLICATIONS

The implication of this study is that the individuals with aphasia processed sentence length spoken messages with a moderate degree of accuracy. A statistically significant difference was found between the individuals with aphasia and the participants with no brain damage in correct response rate. Though no statistically significant difference was found in peak latency and amplitude of the P300, visual examination of the waveforms revealed that individuals with aphasia exhibited a delayed onset of the P300 with slightly higher amplitudes than waveforms exhibited by the participants with no brain damage. In addition, no statistically significant difference was found between the two groups for the peak latency of the N400, though visual inspection of the ERP waveform showed delayed latencies with decreased amplitudes for the individuals with aphasia. Although a statistically significant difference was not found for amplitude of the P300 or N400 at electrodes T7 and T8, a statistically significant difference was found for the peak latency of the P300 and N400 at electrodes T7 and T8 suggesting that reorganization of brain functions has occurred.

Results of this study have the implications for development of improved assessment, treatment techniques and prognostic indicators. The development of improved prognostic indicators may improve the allocation of rehabilitation and financial resources.

RECOMMENDATIONS

Several design issues were identified during the completion of this study. First, a statistically significant difference between the individuals with aphasia and the participants with no brain

damage was not found for behavioral reaction time (measured from the onset of the spoken message to the time that the participant touches their visual choice on the touch screen monitor). In addition, a statistically significant difference for peak latency and amplitude of the P300 and N400 ERP components was not found. The absence of a statistically significant difference, could have been attributed to the heterogeneity of the participants. That is, three of the participants with no brain damage, demonstrated latencies similar to those of the participants with no brain damage. For example, participant NBD5 displayed an onset of positivity for the attentional response at 1199 ms. NBD6 participant 6 exhibited a delayed onset of negativity for the N400 at 1299 ms while NBD participant 9 exhibited a delayed onset of negativity for N400 at 1734 ms. These participants with no brain damage behaved more like the individuals with aphasia. Secondly, the sample size for the individuals with aphasia was six and the group with the participants with no brain damage was twelve. With such a small number of participants, extreme individual participant differences such as those displayed by NBD participants 5, 6 and 9, lack of statistical representation may be an issue. Finally, this study did not control for variables such as age, gender, time post onset and site of lesion. Not controlling for variables such as the ones listed may result in experimental bias and obscure important differences between the groups. Future research should focus on increasing sample size and controlling for variables such as gender, age, site of lesion, and time post onset across the pathological population.

SUMMARY

This chapter discusses the results obtained from individuals with aphasia and participants with no brain damage responding to sentence length spoken messages using electrophysiologic methods. Increased knowledge about the temporal characteristics displayed

by individuals with aphasia may assist in the development of more effective rehabilitation strategies thus improving their quality of life. This chapter also presented recommendations for future research in auditory comprehension.

References

[American Heart Association Statistical Fact Sheets. (2010). Retrieved on Jan. 15, 2010 from http://www.americanheart.org/downloadable/heart/1260905040318FS10WM10.pdf.

Aten, J. L., Caligiuiri, M. , Holland, A. L. (1982). The efficacy of functional communication therapy for chronic aphasic patients. Journal of Speech and Hearing Disorders, 47, 93-96.

Bailey, Dale L. (2005). Positron Emission Tomography: Basic Sciences. Springer. New York, New York.

Basso, A., Capitani, E., Moraschini, S. (1982). Sex differences in recovery of aphasia. . Cortex, 18, 469-475.

Basso A., Gardelli M., Grassi, M.P., & Mariotti, M. (1989). The role of the right hemisphere in recovery from aphasia. Two case studies. Cortex, 25, 555-566.

Belin, P., Zilbovicius, M., Crozier, S., Thivard, L., & Fontaine, A. (1998). Lateralization of speech and auditory temporal processing. Journal of Cognitive Neuroscience, 10, 4. 536-540.

Benson, D. F. &Ardilla, A. (1996). Aphasia a Clinical Perspective. OxfordUniversity Press. New York, New York.

Brookshire, R. H. (2003). Introduction to Neurogenic Communication Disorders. Mosby, St. Louis, Missouri.

Brust, J. C., Shafer, S. Q., Richter, R. W. &Bruun, B. (1976). Aphasia in acute stroke. Stroke, 7, 167-174.

Buxton, Richard. (2002). Introduction to Functional Magnetic Resonance Imaging:

Principles and Techniques. CambridgeUniversity Press. New York, New York.

Cao, Y. Vidingstad E.M., George, K.P., Johnson, A.F., & Welch K.M. (1999). Cortical language activation in stroke patients recovering from aphasia with functional MRI. Stroke, 30, 2331-2340.

Chapey, R. (2008). Language Intervention Strategies in Aphasia and Related Neurogenic Communication Disorders (5th ed.). Lippincott Williams & Wilkins. Baltimore, Maryland.

Connolly, J., Mate-Kole, C. & Joyce, B. (1999). Global aphasia; an innovative assessment approach. Archives of Physical Medicine and Rehabilitation, 80, 1309-1315.

Cortech Solutions, Inc. (2008). Retrieved on April 30, 2008 from http://www.cortechsolutions.com./

D'Arcy, R. C. N., Marchand, Y., Eskes, G., Harrison, E. R., Phillips, S. J. Major, A. & Connolly, J. (2003). Electrophysiological assessment of language function following stroke. Clinical Neurophysiology, 114, 662-672.

Darley, F. L. (1982). Aphasia. W. B. Saunders Company. Philadelphia, P.A.

De Boissezon, X., Marie, N., Castel-LaCanal, E., Marque, P., Bezy, C., Gros, H., Lotterie, J.A., Cardebat, D., Puel, M. &DeMonet, J. F. (2009). Good recovery form aphasia is also supported by right basal ganglia: a longitudinal controlled PET study. European Journal of Physical and Rehabilitation Medicine, 45, 1-12.

DeLong, K., Urbach, T., &Kutas, M. (2005). Probabilistic word pre-activation during language comprehension inferred from electrical brain activity. Nature Neuroscience, 8, 8, 1117-1121.

Demonet, J. F., Chollet, F., Ramsay, S., Cardebat, D., Nespoulous, J.L, Wise, R., Rascol,

A., &Frackowiak, R. (1992). The anatomy of phonological and semantic processing in normal subjects. Brain, 115, 6, 1753-1778

Demeurisse, G., Demol, O., Derouck, M., De Beucklaer, R., Coekaerts, M. J., & Capon, A. (1980). Quantitative study of the rate of recovery from aphasia due to ischemic stroke. Stroke, 11, 5, 455-458.

DeRenzi, E. & Vignolo, L. A. (1962). The Token Test: a sensitive test to detect disturbances in aphasics. Brain, 85, 665-678.

Doyle, P. J., Goldstein, H., Bourgeois, M. S., &Nakles, K. O. (1989). Facilitating generalized requesting behavior in Broca's aphasia: An experimental analysis of a generalization training procedure. Journal of Applied Behavior Analysis, 22, 157-170.

Dunn, L. M. & Dunn, L. M. (1981). Peabody Picture Vocabulary Test. American Guidance Service. Minnesota.

Ferro, J. M. & Crespo, M. (1988). Young adult stroke: neuropsychological dysfunction and recovery. Stroke, 19, 982-986.

Friederici, A., Yves von Cramon, D. &Kotz, S. (1999). Language related brain potentials in patients with cortical and subcortical left hemisphere lesions. Brain, 122, 1033-1047.

Friederici, A &Kilborn, K. (1989). Temporal constraints on language processing: syntactic priming in Broca's aphasia. Journal of Cognitive Neuroscience, 1 (3), 262-272.

Friederici, A., Ruschemeyer, SA., Hahne, A., &Fiebach, C. J. (2003). The role of the left inferior and superior temporal cortex in sentence comprehension: localizing syntactic and semantic processes. Cerebral Cortex, 13,2, 170-177.

Goodglass, H. & Kaplan, E. (1972). Boston Diagnostic Aphasia Examination. The Psychological Corporation. Boston, Mass.

Grafman, J. (2000). Conceptualizing functional neuroplasticity. Journal of Communication Disorders, 33, 345-356.

Handy, Todd. (2005). Event-Related Potentials A Methods Handbook. MIT Press. Cambridge, Ma.

Hagoort, P., Wassenaar, M., & Brown, C. (2003). Real-time semantic compensation in patients agrammatic comprehension: electrophysiological evidence for multiple-route plasticity. PNAS, 100, 7, 4340-4345.

Haynes, W. o., &Pindzola, R. H. (2004). Diagnosis and Evaluation in Speech Pathology (6th ed.). Pearson Education, Inc., Boston, Mass.

Heiss, W. D., Kessler, J., Thiel, A., Ghaemi, M. &Karbe, H. (1999). Differential capacity of left and right hemispheric areas for compensation of post stroke aphasia. Annals of Neurology, 45, 430-438.

Hohnsbein, J., Falkenstein, M. &Hoormann, M. (1995). Effects of attention and time-pressure on P300. Biological Psychology, 40 (1-2), 73-81.

Holland, A. L., Frattali, C. M., & Fromm, D. (1999). Communication Activities of Daily Living. Pearson Assessment, Oxford, England

Holland, A. L., Swindell, C. S., & Forbes, M. M. (2009). The evolution of initial global aphasia: implications for prognosis. Retrieved May 24, 2009 from http://aphasiology.pitt.edu/archive/00000850/01/15-21.pdf.

Inhgam, R. (2007). Neuroimaging in Communication Science and Disorders. Plural Publishing, Inc.

Jasper, H. H. (1958). Report of the committee on methods of clinical examination in electromyography. Electroencephalography and Clinical Neurophysiology, 10, 60-99.

Kang, E. K., Sohn, H.M., Han, M.K., Kim, W., Han, T.R.&Paik, N.J. (2010). Severity of post-stroke aphasia according to aphasia type and lesion location in Koreans. Journal of Korean Medical Science, 25, 123-127.

Karbe, H., Thiel, A., Weber-Luxenburger, G., Herholz, K., Kessler, J., Heiss, W.D. (1998). Brain plasticity in poststroke aphasia: what is the contribution of the right hemisphere? Brain and Language, 64, 215– 230.

Kertesz, A. (2009). Recovery from aphasia and non-verbal impairment in stroke. Retrieved May 24, 2009 from http://aphasiology.pitt.edu/00000376/01/09-08.pdf.

Kertesz, A. (1982). Western Aphasia Battery. Grune&Statton, New York, New York.

Kertesz, A. (1988). What do we learn from recovery from aphasia. In: Waxman, SG., editor. Functional Recovery in Neurological Disease. Vol. 47. New York: Raven Press, p. 897-903.

Kertesz, A. & McCabe, P. (1977). Recovery patterns and prognosis in aphasia. Brain, 100, 1-18.

Kim, H. & Na, D.L. (2004). Normative data on the Korean version of the Western Aphasia Battery. Journal of Clinical and Experimental Neuropsychology, 26, 1011-1020.

Kutas, M. &Hillyard, Steven A. (1984). Event-related brain potentials (ERP) elicited by novel stimuli during sentence processing. Annals of the New YorkAcademy of Sciences, 425, 236-241.

Kutas, M. & King, Jonathan W., (1996). The potentials for basic sentence processing; differentiating integrative proceses. Attention and Performance. XVI ed. MIT Press.

Lendrem, W. & Lincoln, N. (1985). Spontaneous recovery of language in patients with aphasia between 4 and 34 weeks after stroke. Journal of Neurology, Neurosurgery and Psychiatry, 48, 743-748.

Liles, B. Z. & Brookshire, R. (1975). The effects of pause time on auditory comprehension of aphasic subjects. Journal of Communication Disorders, 8, 221-235.

Luck, S.J. (2005). An Introduction to the event related potential technique. MIT Press, Cambridge, Mass.

McNeil, M. R. & Prescott, T. E. (1978). Revised TokenTest.University Park Press, Baltimore, Maryland.

Murray, L.L., Keeton, R.J., &Karcher, L. Treating attentionin mild aphasia: evaluation of attention process training. Journal of Communication Disorders, 39, 1, 37-61.

Musso, M., Weiller, C., Muller, S. P., Bulau, P. Rijntjes, M. (1999). Training induced brain plasticity in aphasia. Brain, 122, 1781-1790.

Naeser, M. A., Gaddie, A., Palumbo, C. &Stiassny-Eder, D. (1990). Late recovery of auditory comprehension. Archives of Neurology, 47, 425-432.

National Aphasia Association. (2007). Retrieved May, 24, 2009, from http://www.aphasia.org

National Institute of Neurological Disorders and Stroke. (2008, October). Retrieved May 24, 2009, from http://www.nids.nih.gov/disorders/aphasia/aphasia.htm

Neville, H., Nicol, J., Barss, A., Forster, K., & Garrett, M. (1991). Syntactically based sentence processing classes: evidence from event-related brain potentials. Journal of Cognitive Neuroscience, 3(2), 151–165.

Nicholas, L.E., & Brookshire, R. H. (1986). Consistency of the effects of rate of speech

on brain-damaged adults' comprehension of narrative discourse. Journal of Speech and Hearing Research, 29, 462-470.

Nigam, A., Hoffman, J.E., Simons, R.F. (1992). N400 to semantically anomalous pictures and words. Journal of Cognitive Neuroscience, 4,1, 15-22.

Parr, Susie. (2007). Living with severe aphasia: tracking social exclusion. Aphasiology, 21, 98-123.

Pashek, G. V., & Brookshire, R. H. (1982). Effects of rate of speech and linguistic stress on auditory paragraph comprehension of aphasic individuals. Journal of Speech and Hearing Research, 25, 377-383.

Pedersen, P. M., Jorgensen, H. S., Nakayama, H., Raaschou, H. O., & Olsen, T. S. (1995). Aphasia in acute stroke: incidence, determinants, and recovery. Annals of Neurology, 38, 4, 659-666.

Petrea, Re., Beiser, A. S., Seshadri, S., Kelly-Hayes, M., Kase, C.S., Wolf, P.A. (2009). Gender Differences in Stroke Incidence and Poststroke Disability in the Framingham Heart Study. Stroke, 40, 1032-1037.

Porch, B. (1967). Porch Index of Communicative Ability. Consulting Psychologists Press, Palo Alto, California.

Richter, M., Miltner, W. H. R. &Straube, T. (2008). Association between therapy outcome and right hemispheric activation in chronic aphasia. Brain, 1-11.

Rugg, M. D. & Coles M. G. H. (1997). Electrophysiology of Mind: Event-Related Brain Potentials and Cognition. OxfordUniversity Press. Oxford, England.

Salmon, N., & Pratt, H. (2002). A comparison of sentence- and discourse-level semantic processing: An ERP study. Brain and Language, 83, 367-383

Sarno, M. T. (1969). The functional communication profile: manual of direction. New York, Institute of Rehabilitation Medicine, New York, New York.

Sarno, M. T. & Levita, E. (1979). Recovery in treated aphasia in the first year post-stroke. Stroke, 10, 663-670.

Scandinavian Stroke Study Group. (1985). Scandinavian Stroke Scale, multicenter trial of Hemodilution in ischemic stroke—background and study protocol. Stroke, 16, 5, 885-890.

Shewan, C. &Kertesz, A. (1984). Effects of speech and language treatment on recovery from aphasia. Brain and Language, 23, 272-299.

Superlab. (2008), Retrieved on April 30, 2008 from http://www.superlab.com

Swaab, T., Brown, C., &Hagoort, P. (1997). Spoken sentence comprehension in aphasia: Event-related potential evidence for a lexical integration deficit. Journal of Cognitive Neuroscience, 9, 39-66.

Swindell, C., Holland, A., Fromm, D. (2007) Classification of aphasia: WAB type versus clinical impression. Retrieved May 24, 2009, from http://aphasiology.pitt.edu/archive/00000795/01/14-07.pdf.

TerKeurs, M., Brown, C.M., Hagoort, P. &Stegeman, D. F. (1999). Electrophysiological manifestations of open-and closed-class words in patients with Broca's aphasia in patients with agrammatic comprehension. an event related brain potential study. Brain, 122, 839-854.

Torbey, M. T. &Selim, M. H. (2007). The stroke book. CambridgeUniversity Press, Cambridge, Mass.

Vanlancker-Sidtis, D. (2004). When only the right hemisphere is left: studies in language and when only the right hemisphere is left. Brain and Language, 91, 2, 199-211.

Warburton, E., Price, C.J., Swinburn, K. & Wise, R.J.S. (1999). Mechanisms of recovery from aphasia: evidence from positron emission tomography studies. Journal of Neurology, Neurosurgery and Psychiatry, 66, 155-161.

Wertz, R. T. (2009). Panel: aphasia with and without adjectives classifying the aphasias: commodious or chimerical. Retrieved May 24, 2009, from archive/000001360/01/13-44.pdf.

Wertz, R. T., Deal, J. L. & Robinson , A. J. (1984). Classifying the aphasias: a comparison of the Boston Diagnostic Aphasia Examination and the Western Aphasia Battery. Retrieved May, 24, 2009, from http://aphasiology.pitt.edu/archive/00000794/.

Zahn, R. Drews, E., Specht, K., Kemeny, S., Reith, W., Willmes, K., Schwarz, M. & Huber, W. (2004). Recovery of semantic word processing in global aphasia: a functional magnetic resonance imaging study. Cognitive Brain Research, 18, 322-336.

Zahn, R., Huber, W., Drews, E., Specht, K., Kemeny, S., Reith, W., Willmes, K. & Schwarz, M. (2002). Recovery of semantic word processing in transcortical sensory aphasia: a functional magnetic resonance imaging study. Neurocase, 8, 376-386.

Zatorre, R. J. &Belin, P. (2001). Spectral and temporal processing in human auditory cortex. Cerebral Cortex, 11, 946-953.

Zurif, E., Swinney, D., Prather, P., Solomon, J. &Bushell, C. (1993). An on-line analysis of Syntactic processing in Broca’s and Wernicke's aphasia. Brain and Language, 45, 448-464.

Appendix

A

SELF-REPORT MEDICAL QUESTIONNAIRE

Self-Report Medical History Questionnaire
UTEP
Brain, Voice and Language Laboratory

The following information is required by the Institutional Review Board to screen for possible participation in EEG studies. We must know if you have had any medical problems that might keep you form participating in this research project. It is important that you be as honest as you can. Information provided will be kept confidential.

Participant ID# _____Age_____Gender_____

1. Since birth have you ever had any medical problems? If yes, please explain.

2. Since birth have you ever been hospitalized? If yes, please explain.

3. Have you ever hit your head and experienced a concussion? If yes, please explain.

4. Did you ever have problems where you saw a counselor, psychologist or psychiatrist? If yes, please explain.

5. Have you ever suffered from seizures? If yes, please explain.

6. Do you use tobacco (smoke, chew)? If yes, please explain.

7. Have you had any hearing problems? If yes, please explain.

8. Have you had any vision problems? If yes, please explain.

9. What is your current weight and height?

10. Do you currently have or have you ever had any of the following? (circle yes or no) Please explain any yes answers.

Yes No strong reaction to cold weather
Yes No circulation problems
Yes No tissue disease
Yes No skin disorders (other than facial acne)
Yes No arthritis
Yes No asthma
Yes No lung problems
Yes No heart problems/disease
Yes No diabetes
Yes No hypoglycemia
Yes No hypertension
Yes No low blood pressure
Yes No hepatitis
Yes No neurological problems
Yes No epilepsy or seizures
Yes No brain disorder
Yes No stroke

11. Have you ever been diagnosed formally to have had?

Yes	No	learning deficiency or disorder
Yes	No	reading deficiency or disorder
Yes	No	attention deficit disorder
Yes	No	attention deficit hyperactivity disorder

12. Do you have

Yes	No	claustrophobia (high fear of small closed rooms)
Yes	No	high fear of needles

13. List any over the counter prescription medications you are presently taking.

14. Do you have or have you ever had any other medical conditions that you can think of? If yes, please note them below.

APPENDIX
B

LIST OF AUDITORY COMMANDS

SUBTEST 3

1. Touch the green square and the black square
2. Touch the blue circle and the green square
3. Touch the white circle and the blue square
4. Touch the black circle and the white square
5. Touch the green circle and the red square
6. Touch the red square and the white circle
7. Touch the white square and the green circle
8. Touch the black square and the red circle
9. Touch the red circle and the white circle
10. Touch the blue square and the black circle

SUBTEST 4

1. Touch the big green square and the little black square
2. Touch the big black square and the little red circle
3. Touch the big blue circle and the little green square
4. Touch the big white circle and the little blue square
5. Touch the little blue square and the big black circle
6. Touch the little green circle and the big red square
7. Touch the little black circle and the little white square
8. Touch the little white square and the big green circle

9. Touch the little red circle and the big blue circle

10. Touch the big red square and the big white circle

SUBTEST 5

1. Put the black square by the red circle

2. Put the black circle above the white square

3. Put the blue square before the black circle

4. Put the red circle on the blue circle

5. Put the blue circle behind the green square

6. Put the green square under the black square

7. Put the white circle below the blue square

8. Put the white square next to the green circle

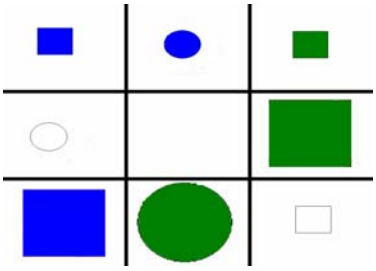
9. Put the red square in front of the white circle

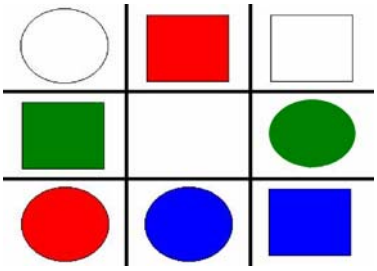
10. Put the green circle beside the red square

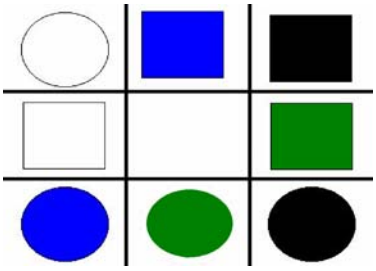
APPENDIX

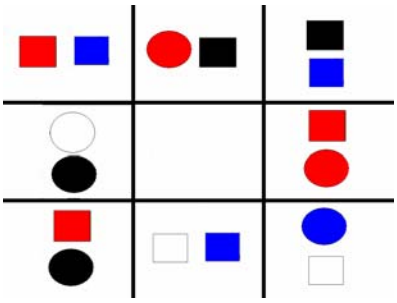
C

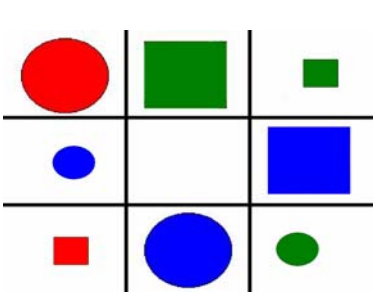
VISUAL STIMULI

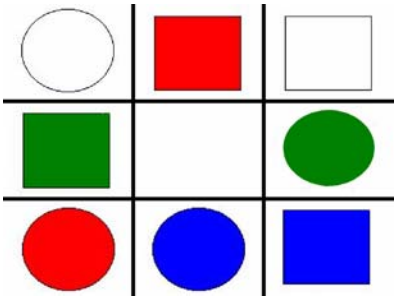


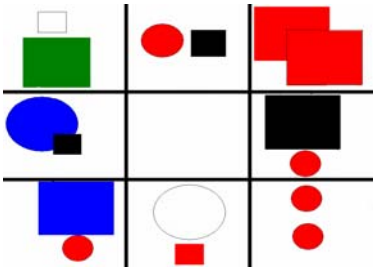


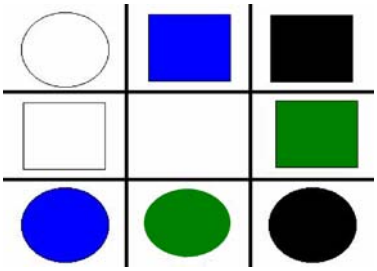


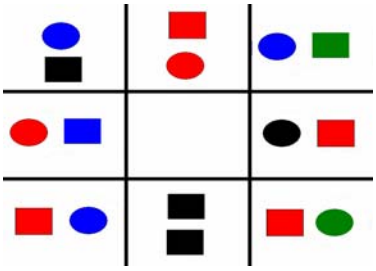


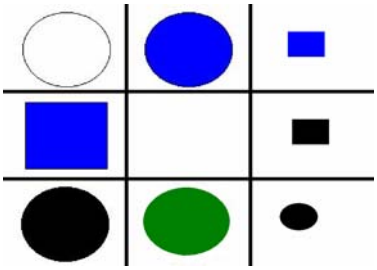


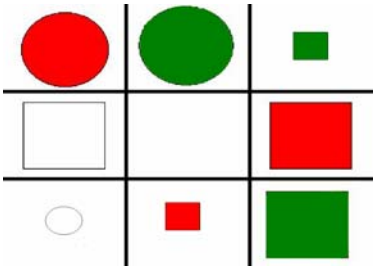


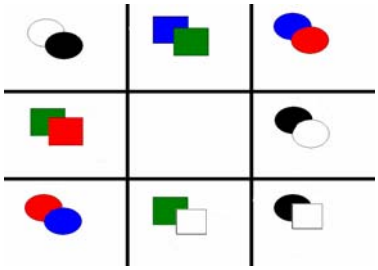


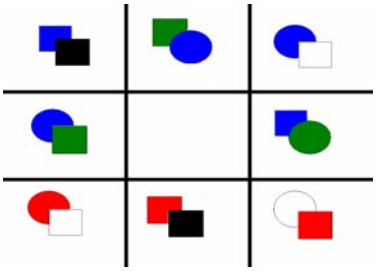


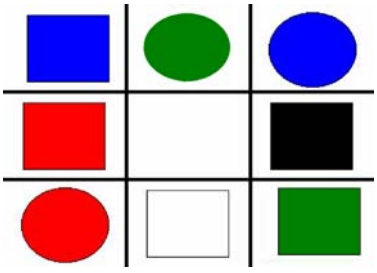


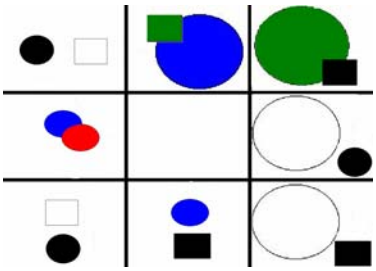


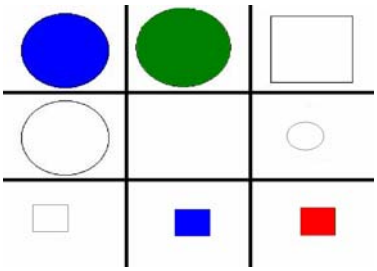


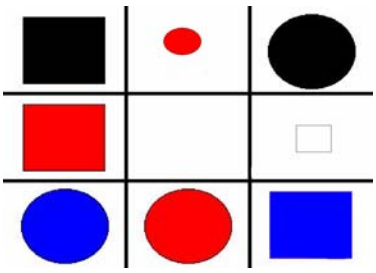


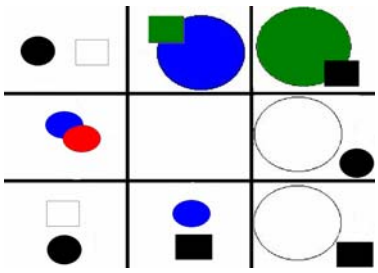


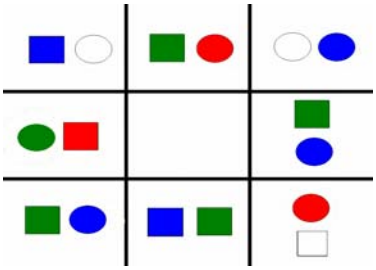


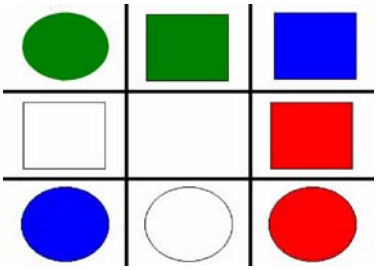


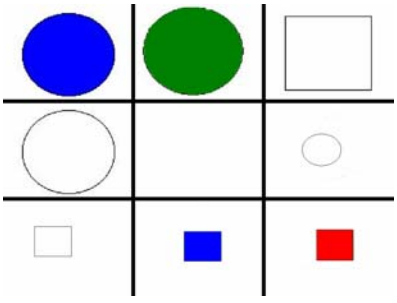


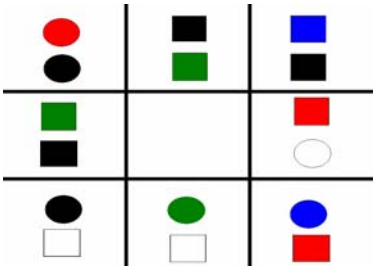


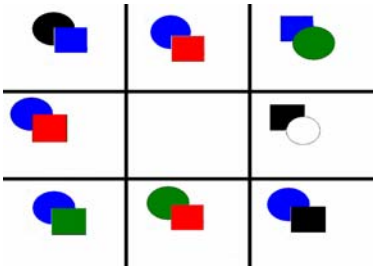


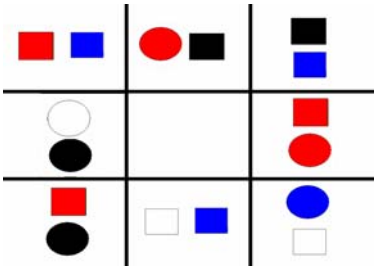












APPENDIX

D

IRB RESEARCH PROPOSAL

Title: Assessing Electrophysiological and Behavioral Activity in Aphasic and Normal Subjects Responding to Spoken Commands

I. Investigators (co-investigators): Patricia Lara, M. A. CCC-SLP
Supervisor: Dr. Anthony P. Salvatore, CCC-SLP, Chair of the Rehabilitation Sciences Dept., Director of the Speech Pathology Department
Dissertation Committee Members: Samuel C. Riccillo, Ph. D., Director Biosemiotic Research Laboratory, Adjunct Faculty College of Health Sciences;
Joe Tomaka, Ph. D. Associate Professor Department of Health Promotion

II. Hypothesis, Research Questions, or Goals of the Project

The purpose of this study is to determine if ERP N400 and peak latency are significantly different between mild aphasic individuals vs. non-brain damaged individuals during an auditory comprehension task. The following questions will be addressed:

1. What are the ERP characteristics such as the N400 and peak latency for non-brain damaged individuals vs. mild aphasic individuals?
2. What is the relationship between the two?
3. Do N400 and peak latency of both groups differ based on linguistic complexity?

III. Background and Significance:

Aphasia is language disorder that affects all modalities including speech, sign, reading, writing and auditory comprehension (Darley, 1982). Aphasia is caused by damage to the language areas of the brain. In most people, this is the left hemisphere. While aphasia is caused by a variety of different etiologies, the most common is stroke (Benson &Ardilla, 1996). It is estimated that approximately one million people in the United States suffer from aphasia and that 25 to 40% of individuals who survive a stroke will acquire the disorder. This means that approximately 100,000 Americans will acquire aphasia each year (National Aphasia Association, 2007, National Institute of Neurological Disorders and Stroke, 2008).

Aphasia can lead to social isolation, depression, financial hardships, loss of personal relationships and stigma on the victim and their families (Parr, 2007). As a result, research has focused on finding an effective means of assessing

and treating the disorder. The difficulty lies in that recovery is being influenced by several factors including initial severity of the disorder, gender, age, type of aphasia, time post-onset and type and extent of aphasia treatment provided. Additional confusion arises from the various theories regarding neuroplasticity, the brain's ability to reorganize itself and its effect on recovery.

In the past, behavioral methodology has been used to measure recovery of aphasia and to predict recovery patterns. However, these methods do not provide correlation between anatomical structures and the functional and temporal processes involved in recovery. In addition, many aphasic patients suffer from physical disabilities that greatly limit the efficacy of behavioral assessment instruments (D'Arcy, et.al., 2003). Advances in neuroimaging technology have provided new avenues to explore aphasia. The neuroimaging options most commonly used in aphasia research are Functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), and Event Related Potentials (ERP) (Handy, 2005).

This study proposes to use ERP to examine the functional brain activity reflected in the electrical activity that is produced in the brain in response to spoken messages. The electrical activity is measured through the skull with electrodes that are attached to a skullcap. While fMRI and PET are excellent tools to localize anatomical structures, they do not provide information regarding the functional and temporal processes that occur in response to stimuli. ERP, on the other hand, provide real-time information in the millisecond range since it is time-locked to a specific stimulus event. Cost and availability make the use of fMRI and PET prohibitive in clinical and research settings. Additionally, fMRI and PET are considerably more invasive than ERP (Handy, 2005). These characteristics make ERP an excellent tool for the study of aphasia.

Test Description

The visual stimuli and auditory commands that will be created for this experiment will be a modification of the stimuli used in The Revised Token Test (RTT) (McNeil & Prescott, 1978). The RTT (1978) is a behavioral assessment instrument that detects mild comprehension deficits in brain injured individuals. The RTT (1978) framework was selected for this experiment because it is based on the theory that a test of language comprehension should be based on a language and not on an intellectual level. Modifications of the stimuli are necessary so that they can be used in an electrophysiologic experiment. Among the modifications described below is the mode

of presentation. The RTT (1978) presents visual stimuli (plastic tokens-squares and circles of different colors-blue, red, white, black and green) on a tabletop using a 4 X 5 matrix. This experiment will present stimuli on a touch monitor using a 3 X 3 matrix. Plastic tokens will not be used in this EEG experiment. Electrophysiologic procedures require that the visual stimuli and auditory commands used in the RTT (1978) be modified so that triggers can be added and presented using an EEG stimulus presentation software. Triggers are markers that are time-locked to each event within the trial and tell the program what event to present next within the trial. Events within a trial contain the features that make a particular trial unique from other trials. The software program Superlab from Cedrus Corporation (Superlab, 2008) will be utilized to present the auditory commands and visual stimuli that will be used in this experiment.. Another modification made to the RTT (1978) for use in this EEG experiment is the use of seven subtest rather than the ten subtests used in the RTT (1978). This modification was selected because of repetitive and redundant trials in the RTT (1978). A reduction in the number of subtests and therefore, trials was necessary due to complaints of fatigue voiced by non-brain damaged subjects. As a result, only the three high scoring subtests will be used for this experiment.

Experimental Task

The patient will be seated comfortably in a 6 X 6 soundproof room in front of the EntuitiveTouchmonitor. The participant will be asked to place his/her hand on a mark on the table at the start of the procedure. The participant will be instructed to return his/her hand to the mark after touching the visual display appearing on the monitor. The mark will be placed at a distance of 34 cm from the touch monitor. Participants will be instructed to move only to touch the screen when either a white sample (Fig. 1.) or visual display of the choices (Fig. 2.) appears on the touch monitor.

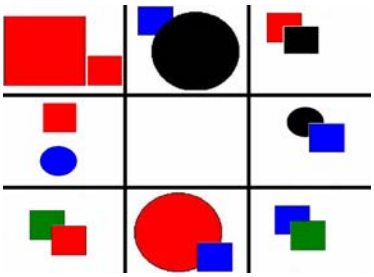
Participant will be instructed to look at the observational white sample (Fig. 1.) that will appear on a black screen on the touchmonitor. Participant will be instructed to touch the white sample (Fig. 1.) to initiate an experimental trial. Participant will be instructed to listen to the auditory command (examples listed below) and carry out the command by touching the visual choice (Fig. 2.) that matches the auditory command.

Individual Trial Sequence

The white sample (Fig.1.) will appear on a black screen. The participant will touch the white sample. Immediately, the auditory command will be presented via speakers. This will be followed by a blank screen indicating a rest period of 1000 ms. Following the 1000 ms rest period, the visual display of choices will appear. The participant will respond by touching the appropriate cell that contains the choice that matches the auditory command. Immediately following the participant response, the screen will go blank for a rest period of 3000 ms. This cycle will repeat for each individual trial.

Fig. 1. Example of an observational white sample

Fig. 2. An example of a visual choice



Examples of Auditory Commands

1. Touch the black circle
2. Touch the big green circle
3. Touch the little red square
4. Touch the green square and the black square
5. Touch the blue circle and the green square
6. Touch the big green square and the little black square
7. Touch the big black square and the little red circle
8. Put the black square by the red circle
9. Put the black circle above the white square
10. Put the big red square in front of the big white circle

Information to be Derived from Test

Comprehension of spoken commands requires the combination of the meaning of individual content words into a global meaning of that command. Event related potentials provide information as to the neural processes and networks that are involved in comprehension on a real-time basis. Therefore, this study seeks to determine what the differences are between the neural processes and networks of individuals with aphasia and non-brain damaged individuals when processing auditory commands. Some of the information that may be obtained includes differences in latency as the spoken commands increase in length and complexity.

Summary

The ability to use and comprehend language is one of the most valuable tools that human beings possess. It sets us apart from other living organisms. An injury to the left hemisphere may result in a loss of comprehension ability as is seen in

aphasia. The desire to help these individuals recover from aphasia and live a more fulfilling life is shared by many clinicians. Finding assessment approaches and therapeutic interventions that will provide accurate diagnosis, prognosis and speed up the recovery process will decrease the frustration felt not just by the patient and family members but also clinicians. Additionally, identification of effective diagnostic and treatment methods may provide an index for allocation of rehabilitative and financial resources. In spite of the advances that provide new insights into the structural changes that occur during the recovery of Wernicke's aphasia, the functional and temporal processes involved in those changes are still not clearly understood.

IV. Research Method, Design, and Proposed Statistical Analysis:

Experimental Design:

The current study proposes to use Event Related Potentials to determine if ERP N400 and peak latency are significantly different between mild aphasic individuals vs. non-brain damaged individuals during an auditory comprehension task. This study compares two groups of different individuals. Therefore, this study is a between subjects design. The independent variable is dichotomous (aphasic vs. non-brain damaged). The dependent variable is continuous (performance as measured by response time, selected ERP components, and peak latency).

Participants:

Participants will be ten English-speaking aphasic patients and ten non-brain damaged individuals from the El Paso area. Based on existing literature, participants will be selected based on the following criteria (Liles & Brookshire, 1975; Pashek& Brookshire, 1982).

Inclusion criteria for the aphasic participants will be:

(a) diagnosis of left hemisphere stroke;
(b) diagnosis of aphasia;
(c) making more than 3 errors on the RTT (Revised Token Test) (McNeil & Prescott, 1978);
(d) normal or corrected-to-normal vision;
(e) normal or corrected-to normal hearing;

Inclusion criteria for Partipants with no brain damage will be:

(a) no documented history of brain damage;
(b) normal or corrected to normal vision;
(c) normal or corrected to normal hearing;

Aphasic participants will be assigned to the experimental group. Partipants with no brain damage will be assigned to the control group.

Participants in both groups will complete the following tasks:

(1) self-report medical history questionnaire;
(2) experimental task;

Partipants with no brain damage will be asked to fill out the self-report medical history questionnaire in order to ensure that the control group is as near as possible to the healthy normal population.

Principal investigator will complete the following tasks:
(1) Administer the Revised Token Test (RTT) (McNeal & Prescott, 1978) to both groups;
(2) Take head measurements;
(3) Apply electrode cap according to Biosemi procedure of conduction gel and amplified electrodes;
Performance between groups will be assessed and analyzed.

Proposed Statistical Analysis:
Based on the research questions outlined below, data analysis will be completed as follows:

1. Is there a statistically significant difference in N400 time, location and peak latency between mild aphasic individuals vs. non-brain damaged individuals responding to spoken commands?
   Comparison ANOVAs will be used to determine if time, location and peak latency variation is statistically significant between the two groups.
2. Is there a statistically significant difference in N400 and peak latency of both groups based on linguistic complexity?
   Comparison ANOVAs will be used to determine if linguistic complexity produces differences in N400 and peak latency.

Spatial analysis will also be completed using Vision Analyzer (Cortech Solutions, Inc., 2008) in order to generate cortical activation. Analysis will be completed offline. Spatial analysis will be in the form of topographic (surface) maps. Topographic mapping will allow for localization of the ERP signal source relative to the electrode placement on the scalp. Topographic maps will be compared across participants.

V. Human Subject Interactions

A. This sample consists of 20 participants. The sample will contain ten participants with left-hemisphere stroke and resulting aphasia and ten participants with no history of brain damage. Participants assigned to the control group will demonstrate less than three errors on the RTT (McNeil & Prescott, 1978). Any indication of auditory comprehension difficulties in the control group participants will disqualify them from participating in this study. Those individuals will be referred to their physician for follow-up. Participants will be tested from November 2010 to March 2011. Participants assigned to the experimental group will be left-hemisphere stroke and resulting aphasia. They will demonstrate three or more errors on the RTT (McNeil & Prescott, 1978).
B. Participants will be recruited from the El Paso Stroke Support Group, the UTEP Speech and Hearing Clinic, outpatient rehabilitation centers and independent health care practitioners. Face-to-face meetings will be used for recruitment purposes. The principal investigator will maintain contact with participants via telephone or mail.
C. Once selected, participants will be provided with verbal explanation as to the purpose of the investigation, all procedures, benefits and/or risks associated with the experiment. Participants will be given an opportunity to read the informed consent. They will also be given opportunity

to ask questions regarding their participation in the study as well as the research project itself. Participants will be provided with an explanation as to their right to participate and/or withdraw from the project at any time. Once all questions have been answered to the participant's satisfaction, they will be asked to sign a letter of informed consent.

D. Participants will be asked to come to the Voice, Brain and Language Laboratory in the Speech and Hearing Clinic at UTEP. Participants will be asked to come to the lab for two visits. During the first visit, participants will be provided with a brief explanation regarding the use of ERP. Participants will fill out the self-report medical and handedness questionnaires. During this first visit, the participant will be assessed using the RTT (McNeil & Prescott, 1978) to determine level of auditory comprehension deficit (high, low performance).

Participant will be asked to come in for a second visit. At the second visit, the principal investigator will take head measurements and apply the electrode cap according to the Biosemi procedure of conduction gel and amplified electrodes. The patient will then be seated comfortably in a 6 X 6 soundproof room in front of a computer touch monitor to complete the experimental task. The experimental task requires that the participant listen to an auditory command and follow the command by touching the visual choice that matches the auditory command using the touch monitor.

E. Data collection, data entry and data analysis will be the responsibility of the principal investigator. All
data, including identifying information in both paper and computer format will be kept in a locked cabinet in the Voice and Language Laboratory in the Speech Pathology Department. Only the principal investigator and her faculty advisors will have access to the data and the Voice and Language Laboratory (1101 N. Campbell, El Paso, Tx. 709902). For added protection, electronic data will be stored in a computer that may only be accessed by a password known only to the principal investigator and her faculty advisors. Data collected and used for this experiment will be stored five years after the termination of the study. Data will be destroyed after this five-year time period.

F. Any information obtained from any participant will be shared only between the principal investigator and her faculty advisors. This information will be used only for the purposes of this research project. All participants will be tested in the Voice, Brain and Language Laboratory in the Speech and Hearing Clinic at the UTEP. The laboratory is equipped with a table and chairs for the participants to sit and fill out their self-report forms. In addition, this area can be closed off from others in the area for privacy purposes. The principal investigator will be responsible for collecting and analyzing the data during the length of this study. Participants will be identified by a number code that will also be used to identify participant data. Data collected during this study will be stored for a period of five years, after which, the data will be destroyed. No video and/or auditory recordings will be completed during this study.

G. All participants will be interviewed and tested in the Voice and Language Laboratory of the UTEP Speech and Hearing Clinic. Tables and chairs are available for the comfort of the participants. This area is equipped with a door that locks for confidentiality and privacy. The stimuli presentation and ERP recording equipment available for this project includes the software program Superlab (2008) from Cedrus Corporation, custom software program ActiveTwo from Bio Semi (Cortech, 2008), two computers, a printer and a touchmonitor. Additionally, electrode caps in three different sizes, amplified electrodes, conduction gel, gloves, measuring tape and locking cabinet are available for use by the principal investigator during this study.

H. Testing will take place from August to November 2010. Two days during the week will be designated as testing days. Additional days will be added if needed.

VII. There are no known risks associated with this research. Testing procedures are non-invasive. However, participants may experience mild fatigue during the testing situation. Participants will be provided opportunities to rest should they voice complaints of fatigue.

VIII. Participants may benefit from this study by knowing the outcome of their performance using event related potentials. This may lead to the use of this tool for assessment of mild aphasia providing for a more accurate diagnosis and prognosis for future recovery.

IX. There are no other specific sites or agencies, other than The University of Texas at El Paso involved in this research project.

X. There are no other IRB approvals, other than The University of Texas at El Paso IRB requested for this project.

References

Benson, D. F. &Ardilla, A. (1996). Aphasia a Clinical Perspective. OxfordUniversity Press
New York, New York.
Cortech Solutions, Inc. (2008). Retrieved on April 30, 2008 from http://www.cortechsolutions.
com./
Darley, F. L. (1982). Aphasia. W. B. Saunders Company. Philadelphia, P.A.
Handy, Todd. (2005). Event-Related Potentials A Methods Handbook. MIT Press.
Cambridge, Ma.
Liles, B. Z. & Brookshire, R. (1975). The effects of pause time on auditory comprehension of
aphasic subjects. Journal of Communication Disorders, 8, 221-235.
McNeil, M. R. & Prescott, T. E. (1978). Revised TokenTest.University Park Press, Baltimore, Maryland.
National Aphasia Association. (2007). Retrieved May, 24, 2009, from http://www.aphasia.org

National Institute of Neurological Disorders and Stroke. (2008, October). Retrieved May 24, 2009, from http://www.nids.nih.gov/disorders/aphasia/aphasia.htm
Parr, Susie. (2007). Living with severe aphasia: tracking social exclusion. Aphasiology, 21, 98-123.
Pashek, G. V., & Brookshire, R. H. (1982). Effects of rate of speech and linguistic stress on auditory paragraph comprehension of aphasic individuals. Journal of Speech and Hearing Research, 25, 377-383.
Superlab. (2008), Retrieved on April 30, 2008 from http://www.superlab.com.

APPENDIX

E

INFORMED CONSENT

University of Texas at El Paso (UTEP) Institutional Review Board
Informed Consent Form for Research Involving Human Subjects

Protocol Title: Comparing N400 and Peak Latency in Non-Brain Damaged and Mild Aphasic Participants when Responding to Spoken Messages

Principal Investigator: Patricia Lara M.A., C.C.C.-SLP

Advisor: Dr. Anthony P. Salvatore, C.C.C.-SLP, Chair of the Rehabilitation Sciences Department and Director of the Speech Pathology Department

Dissertation Committee Members: Samuel C. Riccillo, Ph. D., Director Biosemiotic Research Laboratory, Adjunct Faculty College of Health Sciences; Joe Tomaka, Ph. D. Associate Professor Department of Health Promotion

UTEP College of Health Sciences: Interdisciplinary Health Sciences Ph.D. Program-Brain Voice and Language Laboratory

---

In this consent form "you" always means the study subject. If you are a legally authorized representative (such as a parent or guardian), please remember that "you" refers to the study subject.

1. **Introduction**

You are being asked to take part voluntarily in the research project described below. Please take your time making a decision and feel free to discuss it with your friends and family. Before agreeing to take part in this research study, it is important that you read the consent form that describes the study. Please ask the study researcher or the study staff to explain any words or information that you do not clearly understand.

2. Purpose of the Study

You have been asked to take part in a research study that uses event related potentials to compare N400 and peak latency in non-brain damaged and mild aphasic participants when responding to spoken messages. This study examines brain activity in response to spoken messages and visual pictures.

The rationale: Aphasia is language disorder that affects speech, sign, reading, writing and auditory comprehension. Aphasia is caused by damage to the language areas of the brain. In most people, this is the left side of the brain. While aphasia may result from a variety of different causes, the most common is stroke. It is estimated that approximately one million people in the United States suffer from aphasia and that 25 to 40% of individuals who survive a stroke will acquire the disorder. This means that approximately 100,000 Americans will acquire aphasia each year.

Aphasia can lead to social isolation, depression, financial hardships, loss of personal relationships and social stigma on the victim and their families. As a result, research has focused on finding better assessment and treatment options. This has been a difficult task because many factors such as age, male vs. female, time since stroke and the brain's ability to reorganize itself may affect recovery. However, some factors have not been completely investigated. Finding better evaluation and treatment options may help in adjusting rehabilitation process and the cost of rehabilitation. For these reasons, we need to understand the processes involved in recovery of aphasia.

Approximately, 20 subjects (10 individuals with aphasia and 10 healthy individuals) will be enrolling in this study at UTEP. You are being asked to be in the study because you have been diagnosed with (1) a left hemisphere stroke, (2) aphasia, (3) a healthy subject without brain damage.

If you decide to enroll in this study, your involvement will last about approximately two 45-minute sessions on two separate days.

3. Procedure

If you agree to take part in this study, you will be provided with an explanation regarding the use of event related potentials. Also during your first visit, you will be asked to fill out the self-report medical questionnaire. In addition, you will be assessed by the principal investigator using an

Approved on:	11/03/2010
Expires on:	11/03/2011
Study number:	189351-1

aphasia test to determine whether you are a candidate for this study. You will be asked to come in for a second visit. At the second visit, the principal investigator will measure your head to find the cap that fits you best. The principal investigator will fit you with the electrode cap, apply the conduction gel and attach the electrodes. You will then be seated in a soundproof room in front of a computer touch monitor to complete the experimental task. The experimental task requires that you listen to commands presented through speakers and then follow the command by touching the appropriate visual picture that is shown on the touch screen monitor. Examples of commands that you will hear are "touch the black square", "put the little red circle below the big black square".

4. Risks, Discomforts and Benefits

There are no known risks associated with this research. However, you may experience slight fatigue during the testing conditions. If you feel fatigued, you will be given the opportunity to rest.

5. What will happen if I am injured in this study?

The University of Texas at El Paso and its affiliates do not offer to pay for or cover the cost of medical treatment for research related illness or injury. No funds have been set aside to pay or reimburse you in the event of such injury or illness. You will not give up any of your legal rights by signing this consent form. You should report any such injury to Patricia Lara at (915) 256-6309 and to the UTEP Institutional Review Board (IRB) at (915-747-8841) or rb.orsp@utep.edu.

6. Benefits

There will be no direct benefits to you for taking part in this study. However, you may benefit from this study by knowing the outcome of your performance using event related potentials. This research may lead to better understanding of what is involved in the recovery of aphasia and that may lead to better assessment and treatment options.

7. Options

You have the option not to take part in this study. There will be no penalties involved if you choose not to take part in this study.

8. Funding

Internal Funding:

Funding for this study is provided by UTEP Department of Speech Pathology.

9. Costs

There are no direct costs to you. However, you will be responsible for travel to and from the research site and any other incidental expenses.

10. Compensation

You will not be paid for taking part in this research study.

11. Refusal or Withdrawal

Taking part in this study is voluntary. You have the right to choose not to take part in this study. If you do not take part in the study, there will be no penalty. If you choose to take part, you have the right to stop at any time. However, we encourage you to talk to a member of the research group so that they know why you are leaving the study. If there are any new findings during the study that may affect whether you want to continue to take part, you will be told about them. The researcher may decide to stop your participation without your permission, if he or she thinks that being in the study may cause you harm, and/or there is not sufficient effort on your part to

Approved on:	11/03/2010
Expires on:	11/03/2011
Study number:	189351-1

complete the testing.

12. Contact Information

You may ask any questions you have now. If you have questions later, you may call Patricia Lara at (915) 256-6309 or *plara2@miners.utep.edu*. You may also contact the principal investigator's advisor, Dr. Anthony P. Salvatore at (915) 747-7265 or at *asalvatore@utep.edu*. If you have questions or concerns about your participation as a research subject, please contact the UTEP Institutional Review Board (IRB) at (915-747-8841) or irb.orsp@utep.edu.

13. Confidentiality

Your part in this study is confidential therefore, all information collected in this study will remain confidential. Only the principal investigator (Patricia Lara) and her research advisor (Dr. Anthony Salvatore) will have access to this information. In addition, none of the information will identify you by name. Instead, identification numbers will be used. All records will be stored in a locked cabinet in the Brain, Voice and Language Lab at the UTEP Speech Clinic (1101 N. Campbell, El Paso, Tx. 79902). For further protection, only the principal investigator and her advisor will have access to the locked cabinet. Computer information will be stored in the lab computers and password secured. Only the principal investigator and her advisor will have access to the password. The results of this research study may be presented at meetings or in publications; however, your identity will not be disclosed in those presentations.

14. Mandatory Reporting

If information is revealed about abuse or neglect to the elderly or disabled, the law requires that this information be reported to the proper authorities.

15. Authorization Statement

I have read each page of this paper about the study (or it was read to me). I know that being in this study is voluntary and I choose to be in this study. I know I can stop being in this study without penalty. I will get a copy of this consent form now and can get information on results of the study later if I wish.

Participant Name: _____ Date: _____

Participant Signature: _____ Time: _____

Consent form explained/witnessed by: _____

Signature

Printed name: _____

Date: _____ Time: _____

Approved on:	11/03/2010
Expires on:	11/03/2011
Study number:	189351-1

APPENDIX

F

IRB APPROVAL LETTER

THE UNIVERSITY OF TEXAS AT EL PASO
Office of the Vice President for Research and Sponsored Projects
Institutional Review Board
El Paso, Texas 79968-0587
phone: 915 747-8841 fax: 915 747-5931

DATE:	November 3, 2010
TO:	Patricia Lara, M.A., CCC-SLP
FROM:	University of Texas at El Paso IRB
STUDY TITLE:	[189351-1] Comparing N400 and Peak Latency in Non-Brain Damaged and Mild Aphasic Participants when Responding to Spoken Messages
IRB REFERENCE #:	189351-1
SUBMISSION TYPE:	New Project
ACTION:	APPROVED
APPROVAL DATE:	November 3, 2010
EXPIRATION DATE:	November 3, 2011
REVIEW TYPE:	Expedited Review

Thank you for your submission of New Project materials for this research study. University of Texas at El Paso IRB has APPROVED your submission. This approval is based on an appropriate risk/benefit ratio and a study design wherein the risks have been minimized. All research must be conducted in accordance with this approved submission.

This study has received Expedited Review based on the applicable federal regulation.

Please remember that informed consent is a process beginning with a description of the study and insurance of participant understanding followed by a signed consent form. Informed consent must continue throughout the study via a dialogue between the researcher and research participant. Federal regulations require each participant receive a copy of the signed consent document.

Please note that any revision to previously approved materials must be approved by this office prior to initiation. Please use the appropriate revision forms for this procedure.

All SERIOUS and UNEXPECTED adverse events must be reported to this office. Please use the appropriate adverse event forms for this procedure. All FDA and sponsor reporting requirements should also be followed.

Please report all NON-COMPLIANCE issues or COMPLAINTS regarding this study to this office.

Please note that all research records must be retained for a minimum of three years after termination of the project.

Based on the risks, this project requires Continuing Review by this office on an annual basis. Please use the appropriate renewal forms for this procedure.

If you have any questions, please contact Athena Fester at (915) 747-8841 or afester@utep.edu. Please include your study title and reference number in all correspondence with this office.

APPENDIX

G

STROKE SUPPORT GROUP SUPPORT LETTER

August 9, 2012

UTEP Institutional Review Board
ORSP Admin-209
El Paso, TX78868

Dear Committee Members:

The purpose of this letter is to grant Patricia Lara, a Ph.D. candidate at the University of Texas at El Paso permission to recruit perspective participants for her research from the Stroke Support Group. The project, "Comparing N400 and Peak Latency in Non-Brain Damaged and Mild Aphasic Participants when Responding to Spoken Messages" entails comparison of brain activity in aphasic individuals and non-br3in damaged individuals in an auditory comprehension task using ERP (event related potentials). As Ms. Lara has explained, her intent is to recruit ten participants for the experimental group and ten participants for the control group. The Stroke Support Group was selected because individuals with aphasia secondary to left-hemisphere stroke attend meetings regularly. Ms. Lara has previously held the position of coordinator for the support group and is therefore, familiar with the organization's mission. The Stroke Support Group strives to provide education regarding new advances and resources in stroke recovery to its members, their families and the El Paso area community. Ms. Lara's research may provide some relief in terms of new and innovative assessment and treatment approaches that may assist these individuals live a more fulfilling life. Therefore, Ms. Lara will be asked to share the results of her research with the group membership during one of the regularly scheduled meetings after the project is completed. I, David Baquera, M.S., CCC-SLP, coordinator for the Stroke Support Group do hereby grant permission for Patricia Lara, to recruit perspective participants from the Stroke Support Group.

Sincerely,

David Baquera, M.S. CCC-SLP

Coordinator, Stroke Support Group (577-6561)

Director, Rehabilitation Services SPHN

Coordinator, Stroke Support Group (577-6561)

Director, Rehabilitation Services SPHN

Vita

Patricia Lara was born in El Paso, Tx. where she received her Bachelor of Arts in Speech Language Pathology from the University of Texas in El Paso in 1982. She continued her education in El Paso and received her Master of Arts degree in Speech Language Pathology. She worked in the CanutilloSchool District and El PasoSchool District as a speech language pathologist where she worked with school aged populations. She left the educational system and went to work in the medical field of speech pathology. She has wide clinical experience in dysphagia and adult language disorders.
She worked as staff speech pathology in several hospitals. In addition, Ms. Lara spent 10 years in hospital administration. She entered the doctoral program in Interdisciplinary Health Sciences at the University of Texas at El Paso in 2006. Ms. Lara taught at the university level while pursing her doctoral degree. Ms. Lara's area of research is auditory comprehension, aphasia and event related potentials.

Ms. Lara has presented her research at the Texas Speech Hearing Convention held in San Antonio, Tx. in 2012. She has been a guest speaker at various community meetings and has lectured on acquired adult communication disorders.

Current research
Ms. Lara's current research involves comparing cortical electrophysiologic and behavioral activity in individuals with aphasia and individuals with no brain damage responding to spoken messages. She plans to continue her work in this area including looking at the influence of complexity on spoken message comprehension.

Education:
Ph.D., Interdisciplinary Health Sciences- in progress; University of Texas at El Paso, El Paso, Texas, expected date of completion August 2012
Concentrations: Aphasia, Auditory Comprehension
Dissertation: Assessing Electro-Physiological and Behavioral Activity in Aphasic and Normal Participants Responding to Spoken Messages

M.A., Speech Language Pathology, University of Texas at El Paso, El Paso, Texas, 1985
Concentrations: Child Language Development

B.A, Speech Language Pathology, University of Texas at El Paso, El Paso, Texas, 1983

Teaching and Related Experience:
Instructor, Fall 2011
University of Texas at El Paso, El Paso, Texas
Course: Dysphagia

Instructor, Spring 2009
University of Texas at El Paso, El Paso, Texas
Courses: Disorders of Articulation and Phonology

Permanent address: 6824 WhisperCanyon .
El Paso, Tx. 79912

This thesis/dissertation was typed by Patricia Lara