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Repetition Priming and Anomia: An Investigation of Stimulus Dosage

Catherine A. Off

**A dissertation
submitted in partial fulfillment of the
requirements for the degree of**

Doctor of Philosophy

University of Washington

2008

**Program Authorized to Offer Degree:
Department of Speech and Hearing Sciences**

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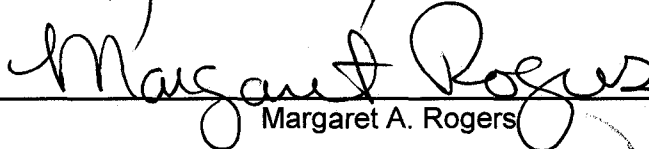


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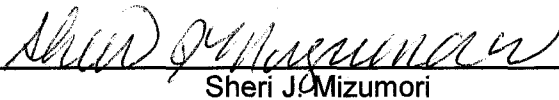
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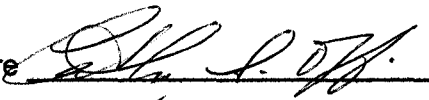
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Abstract

Repetition Priming and Anomia: An Investigation of Stimulus Dosage

Catherine A. Off

Chair of the Supervisory Committee:
Assistant Professor Kristie A. Spencer
Department of Speech & Hearing Sciences

In a recent review of anomia management, Maher & Raymer reported that 30% of aphasia intervention research from 1946 to 2001 focused on naming; however, “despite this proliferation of case reports and small group studies, there is still no clear agreement on how best to manage these deficits” (Maher & Raymer, 2004, p. 13). The inconsistency of acquisition, maintenance, and generalization effects observed across participants and types of treatment protocols is likely to stem from an inadequate knowledge base about how subject and treatment variables influence learning.

One treatment variable that has received increasing attention over the past two or three years is treatment intensity. Principles of neurobiological learning across both animal and human research suggest that the intensity of treatment is a significant factor for learning. Additional research exploring experience-dependent neural plasticity involved in memory and learning indicates that a large number of trials per session are required to elicit behavioral and/or neural change. Despite a considerable amount of literature examining overall treatment intensity, data are not available regarding the frequency (i.e., stimulus dosage) of treatment at which individuals with aphasia will maximally benefit.

A single-subject A-B design with replication across four individuals with aphasia and one healthy non-brain injured gender-matched control participant was used to assess the influence of repeated attempts at picture-naming, coupled with repeated exposure to hearing and reading target words, on the acquisition and maintenance of trained stimuli, and generalization to untrained stimuli. Individuals with chronic aphasia participated in a multi-week repetition priming protocol designed to investigate the influence of stimulus dosage on naming accuracy and latency. Results revealed positive repetition priming effects for trained items across both acquisition and maintenance phases; such positive effects were not observed for untrained stimuli or alternate exemplars. Stimulus dosage manipulations did not consistently influence naming performance for individuals with aphasia.

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DEDICATION

This dissertation is dedicated to my family...

To my husband who has provided me with years of friendship, love, and loyalty, endless hours of encouragement, and a limitless supply of dark chocolate.

To my son who is teaching me how to put life's pursuits into perspective.

To my parents and my brother... Thank you. I promise that you will never have to sit through another one of my graduations!

CHAPTER I: LITERATURE REVIEW

Approximately 700,000 people in the United States survive cerebral vascular accidents (CVA), or strokes, per year, and approximately two-thirds of these stroke survivors require subsequent rehabilitation for a number of impairments including motor deficits, cognitive deficits, and speech and/or language deficits (e.g., NIH, 2006). Specifically, approximately 1,000,000 individuals in the United States suffer from aphasia, with the majority of these cases resulting from stroke (Holland, Fromm, DeRuyter, & Stein, 1996, ASHA, 2004). In a large prospective study involving over 1000 participants with a diagnosis of CVA, aphasia was observed to occur in 38% of the sample, with the incidence rising to 40% when only participants with left-hemisphere lesions were assessed (Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1995). Furthermore, Pedersen and colleagues found that of the participants with aphasia who survived the stroke, 44% completely recovered by the time they were discharged from the hospital. At a six month follow-up, 50% of participants with an initial diagnosis of aphasia continued to present with aphasia; that is after six months of recovery time, only an additional 6% of participants with aphasia had completely recovered their language function.

Formally, aphasia has been defined as follows:

Aphasia is a multimodality physiological inefficiency with [greater than loss of] verbal symbolic manipulations (e.g. association, storage, retrieval, and rule implementation). In isolated form it is caused by focal damage to cortical and/or subcortical structures of the hemisphere(s) dominant for such symbolic manipulations. It is affected by and affects other physiological information processes to the degree that they support, interact with, or are supported by the symbolic deficits (McNeil & Pratt, 2001, p.907).

Clinically, aphasia is characterized by impairments of expressive and receptive language functions across some or all modalities including writing, speaking, drawing, and gesturing; the severity of these expressive and receptive deficits typically varies across modalities. Traditionally, classifications of aphasia stemmed from a localizationist perspective, suggesting a one-to-one mapping of neural structure to linguistic function. However, enough evidence has emerged to reject a

simple one-to-one mapping of lesion and deficit, instead revealing a widely distributed network that is activated differentially across linguistic tasks (for a recent discussion of this topic, see Poeppel & Hickok, 2004). Despite this change in perspective, traditional classifications continue to be used by aphasiologists to describe the relative linguistic strengths and weaknesses presented by an individual with aphasia. Table 1.1 provides a brief description of the classification of aphasic symptoms and related neural correlates (adapted from Helm-Estabrooks & Albert, 1991, p. 42). Naming impairments, the focus of this project, present across all categories of aphasia.

Table 1.1 Classification of Aphasic Symptoms

Non-Fluent Aphasia	Description	Anatomical Correlates	Fluent Aphasia	Descriptions	Anatomical Correlates
Global Aphasia	Poor auditory comprehension	Large perisylvian, extending into white matter	Wernicke's Aphasia	Poor auditory comprehension; poor repetition	Posterior third of supramarginal gyrus
Broca's Aphasia	Good auditory comprehension Poor repetition	Lateral frontal, suprasylvian, prerolandic, extending into white matter	Transcortical Sensory Aphasia	Poor auditory comprehension; good repetition	Posterior parieto-temporal; Wernicke's area is spared
Transcortical Motor Aphasia	Good auditory comprehension Good repetition	Anterior frontal; anterior and superior to Broca's	Conduction Aphasia	Good auditory comprehension; poor repetition	White matter pathways inferior to supramarginal gyrus
			Anomic Aphasia	Good auditory comprehension; good repetition	Angular gyrus, second temporal gyrus

Aphasia rehabilitation is considered to be efficacious and effective by practicing clinicians and clinical aphasiologists; however, according to the most recent Cochrane Review which evaluates randomized controlled trials, "speech and language therapy treatment for people with aphasia after a stroke has not been shown either to be clearly effective or clearly ineffective" (Greener, Enderby, & Whurr, 2004). Additionally, in a meta-analysis of aphasia treatment, Robey (1998) states, "the direct implication [of the lack of studies meeting criteria for a meta-

analysis] is that outcome research in aphasia treatment has not been consistent with the conventions of controlled clinical trials as practiced in the general clinical-outcome research community (p. 175),” and, “if the body of scientific evidence is to advance optimally, focused hypotheses must be tested on programmatically (e.g., replications on tests of dosage, specific populations, certain severities, and treatment protocols) (p. 183)”. Individually, single-subject and small group designs have demonstrated very large gains as a result of aphasia treatment (for a recent examination of lexical retrieval evidence see Robey & Beeson, 2005); however, reducing and synthesizing this enormous literature base in the context of a systematic review often leaves practicing clinicians wondering which treatment approach and/or delivery option will optimize progress during the stroke recovery process for their own clients. Maher & Raymer concur stating, “...despite this proliferation of case reports and small group studies, there is still no clear agreement on how best to manage these deficits” (Maher & Raymer, 2004, p.13). Consequently, aphasiologists are not able to (1) consistently differentiate among neurologically divergent forms of aphasia, and/or (2) reliably treat individuals who present with various manifestations of aphasia.

Past reviews that have examined the state of the evidence regarding aphasia treatment have necessarily focused on broad questions regarding aphasia treatment efficacy, without differentiating between types of treatment or what may ultimately be revealed as clinically divergent manifestations of aphasia (de Pedro-Cuesta, Widen-Holmqvist, & Bach-y-Rita, 1992; Greener, Enderby, & Whurr, 2004; Holland, Fromm, DeRuyter, & Stein, 1996; Robey, 1998). Although these broad explorations have provided essential preliminary information, the questions being asked about aphasia need to be refined to systematically investigate basic issues in aphasiology including: (1) neuroplastic mechanisms underlying rehabilitation and processes of learning in a recovering brain that may ultimately guide treatment delivery decision-making; and (2) subject and treatment delivery variables that ostensibly influence these learning processes. The primary aim of this project is to examine one treatment delivery variable, stimulus dosage, in the context of recovery from aphasia and to outline how this variable potentially interacts with processes of learning during the rehabilitation of aphasia. Results stemming from this

investigation will be interpreted using theoretical mechanisms of learning as they pertain to the acquisition, maintenance, and generalization of lexical retrieval in individuals with anomia.

NEUROPLASTICITY DURING RECOVERY FROM APHASIA

“...the brain is an organ of adaptation and the readiness for learning is intrinsic to its cells” – William Greenough

Historically, language functions were presumed to correspond in a one-to-one fashion with precise neural structures; that is, the lesion deficit hypothesis assumed a direct link between a focal lesion(s) and the ensuing impairment(s) of linguistic function. This hypothesis was initially based upon autopsy results combined with documented pre-morbid behavioral characteristics, thus leading to much of the currently used neuroanatomical terminology (e.g., Broca's and Wernicke's areas) and directing many early imaging studies using x-ray and computed tomography (CT) that sought to identify, locate, and verify the existence of language centers. Despite the advancement of imaging technologies that have become increasingly more sensitive to spatial features of the brain (e.g., magnetic resonance imaging (MRI)), the lesion deficit hypothesis is not able to provide a complete model of cognitive-linguistic processes for a number of reasons including: (1) it cannot provide information about which non-damaged structures are involved in the language tasks being assessed, (2) it assumes an inference between normal and damaged brains; that is, it assumes that a lesioned brain is simply a normal brain minus a region of tissue, and (3) it does not permit the development of theoretical neural circuits and/or networks that may be activated during language processes.

With the advent of functional imaging technologies that allow researchers to examine the living brain during the use of language processes, it has become even more apparent that a simple one-to-one mapping of lesion to deficit is erroneous. Instead, mounting evidence suggests that although language is typically dominant in the left hemisphere for most individuals, language processes involve a widespread network across both hemispheres and both cortical and subcortical tissue. Furthermore, this network appears to demonstrate significant variability across

individuals. As a result of this neural variability, in addition to other subject and treatment variables, understanding processes of learning in individuals with aphasia has proven difficult. In recent years, researchers have begun to explore this variability at both behavioral and neurobiological levels. Specifically, researchers have turned their attention to the neuroplastic mechanisms underlying recovery processes in order to better understand subject variability as it applies to recovery processes and amenability to rehabilitation protocols.

Although neuroplasticity has been discussed in the context of development and learning since the early 1800's, experience-dependent plasticity (i.e., neurobiological changes resulting from learning) wasn't systematically investigated in adult animal models until the 1980's. Accordingly, theories about neuroplastic mechanisms in adult humans emerged and have since been viewed as a significant component of functional recovery subsequent to stroke (e.g., Nudo, 2004). With the additional technological advancement of functional neuroimaging in the 1990's, investigations of cortical plasticity became possible in living adult humans and have conclusively demonstrated that the adult human brain is capable of significant neural change and functional reorganization following learning experiences including recovery and rehabilitation from stroke (Bruno, 2004; Hallett, 2001; Nudo, 2004).

During the acute stage of recovery, ranging from hours to days post-CVA, physiologic neural and metabolic changes occur in response to the ischemic event. Initially, local changes occur at the site of the infarct and the surrounding (peri-infarct) tissue (Nudo, 2004). At this stage of recovery effects of the ischemic event begin to resolve including a reduction of edema, a reuptake of toxins, improved or restored blood flow (i.e., reperfusion), a resolution of blood pressure, and possible repair of damaged but not destroyed cells (Bruno, 2004; Hallett, 2001; Hillis & Heidler, 2002; Papathanasiou & Whurr, 2000). In addition to these local physiologic changes, the ischemic event can also disrupt the excitability of individual neurons or groups of neurons both proximal and distal to the lesion (Hallett, 2001; Herholz & Heiss, 2000). For example, cellular degeneration or denervation supersensitivity may occur as a result of a loss of connections (Kean, 2005; Papathanasiou & Whurr, 2000). Denervation supersensitivity is defined as an enhanced or

exaggerated response to neurotransmitters, and occurs when a smaller than normal amount of neurotransmitter is available at the synapse (Kean, 2005). As a result of these rapid physiologic responses to the ischemic event, neurons and supporting cells exist in a state that is particularly predisposed for functional change in the context of rehabilitation and/or learning; that is, the brain exists in a state of reactive plasticity (Nadeau, Gonzalez Rothi, & Rosenbek, in press). These early mechanisms of recovery are typically referred to under the umbrella term spontaneous recovery, and are typically thought to be relatively independent of neural changes occurring as a result of rehabilitative protocols and/or language-based experiences following stroke. Although a few authors have begun to explore cortical reorganization at early stages of spontaneous recovery (Hillis, 2006; Saur, Lange, Baumgaertner, Schraknepper, Willmes, Rijntjes, & Weiller, 2006), aphasiologists have yet to systematically explore the acute effects of early re-training on neuroplastic mechanisms. A programmatic line of research is needed to further delineate cortical reorganization during spontaneous recovery and reorganization that ostensibly results from rehabilitation during the acute stage of recovery.

During the sub-acute and chronic stages of recovery, lasting weeks to years after a CVA, functional recovery begins to occur. At this time, both behavioral compensation (i.e., the use of alternative strategies for use of the impaired function) and adaptive plasticity are thought to play a role in observable behavioral changes (Nudo, 2004). In terms of adaptive plasticity, functional changes appear to result from a complex coordination of multiple levels of neural plasticity, ranging from local molecular changes to system-wide reorganization (e.g., Gazzaniga, 2000, see chapters 9-16). Changes in synaptic potential are likely to provide relatively immediate functional changes; for example, connections previously inhibited may be unmasked as a result of ischemic events, or existing connections may be strengthened or weakened through processes of long-term potentiation (LTP) and long-term depression (LTD), respectively (Bruno, 2004; Buonomano & Merzenich, 1998; Hallett, 2001; Johansson, 2000; Keefe, 1995; Nudo, 2004). Neuro-anatomical changes are likely to emerge later during the recovery process, including synaptogenesis in the form of increased dendritic arborization (Buonomano &

Merzenich, 1998; Grossman, Churchill, Bates, Kleim, & Greenough, 2002; Keefe, 1995; Nudo, 2004). Such anatomical changes appear to be primarily experience-dependent, and are thus more likely to occur in the context of rehabilitation or intensive use of the impaired function (Greenough, 2005; Grossman, Churchill, Bates, Kleim, & Greenough, 2002). New connections such as these may ultimately provide access to regions that were initially isolated from the lesion (Hillis & Heidler, 2002). These theories of rehabilitation have emerged from animal studies; to date, however, systematic investigations of the effects of aphasia rehabilitation and associated treatment variables on neuroplastic mechanisms have yet to be published.

Non-neuronal supporting cells have also been examined in animal models to determine their role in neuroplasticity and have subsequently shown that plasticity is not exclusive to neurons. For example, oligodendrocytes have been shown to produce more myelin, thus creating an increased number of high-speed connections in adult animals that have been exposed to complex or enriched environments relative to those not exposed to enriched environments (Greenough, 2005; Grossman, Churchill, Bates, Kleim, & Greenough, 2002). Animals exposed to enriched environments have also demonstrated an increased capillary volume, reflecting an improved cerebrovascular system, and an increased number of glial cells in general, thus providing increased nourishment for the neural system (Greenough, 2005; Grossman, Churchill, Bates, Kleim, & Greenough, 2002).

Large scale, system-wide cortical plasticity observed following stroke in adult humans is typically referred to as functional neural reorganization, which results from a realignment of the relative interaction between cortical structures and cognitive functions (Hillis & Heidler, 2002). Such representational changes appear to emerge later in the recovery process (i.e., chronic stages of recovery), and manifest differently across recovering individuals. Cortical reorganization is thought to result from a coordination of the abovementioned neuroplastic mechanisms as well as a regression of diaschisis (Seitz, Azari, Knorr, Binkofski, Herzog, & Freund, 1999), the repair and/or restitution of partially damaged pathways (Hillis & Heidler, 2002), and the use of existing, undamaged, or redundant pathways. Cortical reorganization during stroke recovery is thought to include recruitment or use of: (1)

task-related regions in the ipsilateral hemisphere, (2) perilesional undamaged tissue, and (3) homologous regions in the unimpaired contralateral hemisphere (e.g., Rijntjes, 2006). If the functional network is partially preserved, compensation is likely to take place for the lost function; that is, the remaining tissue of the functional network may take over the function that was previously assigned to the lost tissue (Barker & Dunnett, 1999; Gonzales Rothi, 2000). Alternatively, if the functional network is severely damaged, another system may adapt to take over or substitute the lost function (Barker & Dunnett, 1999; Gonzales Rothi, 2000; Grafman, 2000).

Although the investigation of cortical reorganization in adult humans is in its infancy, the processes underlying reorganization are likely to be sensitive to the extent to which and the context in which the stroke survivor uses the impaired function (Bruno, 2004; Hallett, 2001). However, this theory of reorganization has yet to be systematically investigated in the context of aphasia rehabilitation; specifically, although subject variables (e.g., site/size of lesion, stage of recovery) have been investigated to some degree, studies examining treatment variables such as task, stimuli, and treatment intensity are currently absent from the literature base.

For aphasiologists, the crucial piece of information to be gained from the neural plasticity literature is that experience drives neural change (Ivanco & Greenough, 2000). Furthermore, neuroplastic recovery mechanisms following brain injury appear to be sensitive to a number of intrinsic (e.g., size/site of lesion, stage of recovery) and extrinsic variables (e.g., treatment variables). However, little is understood about how either of these variables relate to neuroplastic recovery processes, or whether they are responsible for priming the system for some particular path of cortical reorganization. In theory, different combinations of intrinsic and extrinsic variables are likely to determine these different paths of reorganization and subsequent degrees of functional recovery. As such, it is imperative that aphasiologists systematically investigate aphasia recovery to determine the subject and treatment variables that can be manipulated to result in behavioral change.

MECHANISMS OF LEARNING

An early study examining the learning abilities of individuals with aphasia demonstrated that patients exhibit many of the same behavioral correlates relative to learning as those without brain damage (Carson, Carson, & Tikofsky, 1968). Specifically, individuals with aphasia were able to (1) learn new tasks (as demonstrated by decreased response time and/or increased accuracy across trials), (2) retain newly learned information and/or skills over time, and (3) process complex stimulus material. Relative to non-brain injured controls, however, individuals with aphasia demonstrated generally slower response times and lower levels of achievement.

The rehabilitation of language is inherently supported by mechanisms of learning. However, despite calls for aphasiologists to include a discussion of principles of learning thought to underlie their treatment protocols (Baddeley, 1993; Ferguson, 1999; Laine, 2000; Martin, 1996; Stark, 2005), few authors have done so (although, for example, see Baddeley, 1993; Breitenstein & Knecht, 2002; Fillingham, Sage, & Lambon-Ralph, 2006; Plaut, 1996)¹. This lack of a theoretical framework of learning upon which to base rehabilitative protocols is particularly noteworthy considering the amount of evidence suggesting that cortical reorganization is modulated in response to a multitude of intrinsic and extrinsic variables, ostensibly as a result of inherent mechanisms of learning. Furthermore, animal, and more importantly, human studies of motor learning following stroke have provided a significant body of literature upon which to model language learning (for a recent review, see Dobkin, 2004). Although the neural correlates underlying language processes are significantly different from those supporting motor functions, the neural processes that enable and facilitate learning are likely to be influenced by similar variables across both motor and cognitive-linguistic functions.

Given evidence from both neuroimaging studies of cortical reorganization following stroke, and behavioral or cognitive-behavioral studies of learning and

¹ Breitenstein & Knecht provide a brief model of implicit language learning (purely behavioral); Fillingham et al. discuss a protocol based on errorless learning, but do not propose a complete theory of (re)learning. Plaut proposes a cognitive neuropsychological theory of learning based upon connectionism. Baddeley provides a review of cognitive theories of learning, in general, from the standpoint that models of working memory may help explain learning processes in an impaired system.

rehabilitative processes, aphasiology, as a science, is in the optimal position to tie neural, neuropsychological, cognitive, and behavioral theories together to propose a complex theory(s) of learning during recovery from stroke². As such, this chapter will explore the rehabilitation process in terms of principles inherent to learning and specific neurobiological mechanisms that appear essential for learning following stroke. Coupled with neuroplastic mechanisms of cortical reorganization, mechanisms of learning can provide a theoretical framework upon which the manipulation of treatment variables can be motivated.

Neurobiological Principles of Rehabilitation

The goal of any rehabilitation program is to reduce the behavioral, motor, and/or cognitive effects of a neurological disease or disorder such as stroke (Dobkin, 2004). In a review article that assessed models of rehabilitation for cognitive impairments subsequent to brain injury, Lillie and Mateer outlined four outcomes of rehabilitation: (1) restoration of damaged function, (2) optimization of residual function, (3) compensation for lost function, and (4) substitution of intact function (Lillie & Mateer, 2006, p.119). Without question, the most desirable outcome is to restore the impaired function to its original state; however, complete repair of neural damage is unlikely in most cases. As such, a more realistic goal may be to activate intact neural connections and promote cortical reorganization by optimizing the residual function.

In rehabilitative medicine, the past two decades or so of research shifted its focus from primarily impairment-based treatment approaches to those that help the individual compensate for their lost or impaired abilities. For example, in physical therapy, patients were encouraged to use the unimpaired hand to complete activities of daily living (for a review, see Bruno, 2004). Similarly, individuals with aphasia have been coached to use modalities other than spoken language such as gesturing, drawing, and writing to facilitate functional communication during conversation (Elman & Bernstein-Ellis, 1999; Kagan, 1995; Rogers, Alarcon, &

² Just before this paper was printed an in press book chapter was obtained from neurologist Dr. Steven Nadeau that provides a compelling neural model of language rehabilitation that incorporates a connectionist neural network model with neuropsychological phenomena and both neural and behavioral correlates of language and language impairments (Nadeau, Gonzalez Rothi, & Rosenbek, in press).

Olswang, 1999; Simmons-Mackie & Damico, 1995). Such rehabilitative approaches certainly serve their purpose to help stroke survivors interact with their environment in a meaningful way as soon as possible after the stroke. However, compensatory approaches may also be selected by practicing clinicians because of a lack of consistent and/or compelling evidence that supports impairment-based approaches. Therefore, as researchers, we are obligated to provide theoretically motivated, but clinically relevant evidence that details variables germane to each of these types of rehabilitative outcomes. Currently, the variables that may influence restoration and/or optimization of the residual language function(s) are poorly understood; however, a systematic line of research based upon principles of learning and their influence on cortical reorganization should ultimately shed light on the factors that are necessary and essential for successful rehabilitation, regardless of the desired outcome (i.e., restoration and/or optimization vs. compensation).

With the relatively recent explosion of animal and human studies examining neuroplastic mechanisms of recovery, researchers have begun to explore how various rehabilitative approaches are associated with neuroplastic processes during recovery. Based upon a review of the motor learning literature as it applies to rehabilitation, Elbert and Rockstroh have proposed four guiding neurobiological principles necessary for successful cortical reorganization to occur³ (Elbert & Rockstroh, 2004, p. 132). The first principle, *practice makes perfect*, is based on studies that examine skill learning. This principle proposes that intensive, massed stimulation of a particular motor skill results in expanded cortical representational maps, indicative of an increased level of importance for that particular skill. The amount of practice necessary to result in learning is well studied in the motor learning literature and is cited by Schmidt and Lee as the "most important condition" when learning a new skill (Schmidt & Lee, 1999, pp. 285-286). The second principle, *use it or lose it*, is based on investigations of limb or digit amputation and/or loss and/or ablation of innervation subsequent to brain injury. In instances during which decreased or completely ablated innervation to a particular cortical region occurs, nearby groups of cells may be recruited during learning, thus

³ Cortical reorganization and/or neuroplasticity, in general, are discussed in the context of motor learning using animal models. Elbert and Rockstroh do not discuss cortical reorganization in the context of cognitive-linguistic regions of interest.

expanding an adjacent cortical representational map or narrowing the previously used representational map. The third principle, *fire together, wire together*, is based on the Hebbian theory of learning, stating that the persistent stimulation of one cell during skill learning will eventually lead to the simultaneous stimulation of adjacent cells; eventually this process is thought to spread to several neighboring cells, thus expanding the representational map (Hebb, 1949). A basic assumption underlying this principle is that the initial stimulation of the cell must be associated with a functionally important behavior to the individual being trained and/or rehabilitated (Keefe, 1995, p. 91). Finally, the fourth principle, *you have to dream it to achieve it*, is based on studies that have demonstrated that not only is intensive and prolonged practice of the skill required to result in cortical reorganization, but the behavior being learned or relearned must be of such interest or importance to the individual that the brain continues to process the new skill during sleep (e.g., Stickgold & Walker, 2005). Collectively, these tenets provide a neurobiological framework that can be applied to the investigation of learning during stroke recovery in individuals with aphasia⁴.

As a result of increased evidence supporting neuroplastic mechanisms during recovery, a significant amount of research has been conducted to better understand how restoration or optimization of function may be achieved. For example, in the context of physical rehabilitation, practitioners and researchers alike provide intense, repetitive treatment of the impaired limb (i.e., constraint-induced movement therapy), which is hypothesized to facilitate neurological and behavioral recovery, prevent learned non-use of the limb, and directly influence functional reorganization (Bruno, 2004; Kunkel, Kopp, Muller, Villringer, Villringer, Taub, & Flor, 1999). The National Institute of Health (NIH) concurs, stating that "there is a strong consensus among rehabilitation experts that the most important element in any rehabilitation program is carefully directed, well-focused, repetitive practice - the same kind of practice used by all people when they learn a new skill, such as playing the piano or pitching a baseball" (NIH, 2006). This model of rehabilitation has gradually filtered into the field of aphasiology with researchers now beginning to

⁴ See also Dobkin (2004) for a synopsis of specific intrinsic and extrinsic neuroplastic mechanisms likely to influence restoration of function.

explore ideas of constraint and intense practice that may be relevant to language rehabilitation following stroke (Lillie & Mateer, 2006; Pulvermuller, Neininger, Elbert, Mohr, Rockstroh, Koebbel, & Taub, 2001); this renewed interest in the restoration and optimization of function is also reflected by the type of studies currently being funded by NIH.⁵ However, until recently (Kleim & Jones, 2008; Raymer, Beeson, Holland, Maher, Martin, Murray, Rose, Thompson, Turkstra, Altmann, Boyle, Conway, Hula, Kearns, Rapp, Simmons-Mackie, & Gonzales Rothi, 2008) a discussion about how treatment variables are theorized to modulate the processes of learning to ultimately optimize neurobiological mechanisms of recovery was strikingly absent from the literature base. In the past, aphasiologists had recognized the importance of the interaction of subject and treatment variables in the context of rehabilitation; however, no discussion of *how* these variables modulate the learning process, relative to either cognitive-behavioral or neurobiological models of learning, was provided. With the recent attention to using principles of experience-dependent plasticity⁶ aphasiologists can begin to answer questions about (1) the way that individuals with aphasia learn and (2) the nature of neurological recovery (specifically, cortical reorganization) that allows for treatment variables to matter.

Principles of Learning to Consider for Rehabilitation

Learning can be broadly defined as the process or processes necessary to acquire new information or new capabilities; whereas, memory can be broadly defined as information or a capability that is acquired and subsequently persists to be retrieved or used at a time distant from the moment of acquisition (Gazzaniga, Ivry, & Mangun, 2002). As such, learning and memory are intimately connected, manifesting as different stages of the same processing mechanism. For the purposes of this paper, memory and learning are discussed collectively as learning,

⁵ See <http://clinicaltrials.gov/ct/gui/action/SearchAction?term=Aphasia> for a current list of NIH funded clinical trials in the area of aphasiology. Currently, two out of nine clinical trials are recruiting participants to examine constraint-induced aphasia treatment (CIAT).

⁶ Kleim & Jones (2008) propose ten principles of experience-dependent plasticity relative to neuro-rehabilitation: (1) use it or lose it; (2) use it and improve it; (3) specificity; (4) repetition matters; (5) intensity matters; (6) time matters; (7) salience matters; (8) age matters; (9) transference; (10) interference. Results from the current investigation presented will be discussed in light of both Elbert & Rockstroh's and Kleim & Jones' principles of experience-dependent plasticity.

and defined as a process occurring as a result of experience that results in the acquisition or reacquisition of a capability, as demonstrated by long-lasting behavioral changes (Schmidt & Lee, 1999; Stark, 2005).

In the broadest sense, learning has been modeled as behavioral, cognitive, or cognitive-behavioral in nature (Ferguson, 1999). While behavioral theories are based solely on how the environment affects overt behavioral responses (i.e., stimulus-response); cognitive and cognitive-behavioral theories of learning make hypotheses about internal influences (i.e., emotional, cognitive, and psychosocial factors) on learning. More recently, cognitive neuropsychological theories have provided a framework within which neurological processes and neural substrates of various cognitive functions can make predictions about impairments stemming from neurological disorders and diseases (Gazzaniga, 2000). In aphasia therapy, most treatment protocols appear to be based upon a cognitive neuropsychological or cognitive-behavioral theory of learning, whether this is directly stated or not. Typically, however, investigators use these cognitive-behavioral or cognitive-neuropsychological theories to guide their model of language processing, rarely discussing how these theories of learning motivate the particular learning paradigm or treatment protocol that they have selected to investigate⁷ (Howard, 2006).

Behavioral learning paradigms generally include habituation, simple conditioning, priming, and skill learning (Ferguson, 1999; Laine, 2000). These paradigms have been extensively examined in healthy participants and patients with various memory impairments (e.g., Knowlton, Mangels, & Squire, 1996; Squire, 1992); yet, systematic investigations of the manner in which individuals with aphasia respond to various behavioral paradigms of learning have yet to be conducted. The goal, then, for aphasiologists, is to develop a theory of learning that considers: (1) neurobiological principles of learning⁸, (2) models of cortical reorganization during aphasia recovery, and (3) cognitive-behavioral or cognitive neuropsychological

⁷ Although, one line of research that has explicitly asserted a cognitive neuropsychological theory of (re)learning includes authors who examine connectionist models of lexical retrieval (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Martin, Fink, Laine, & Ayala, 2004; Nadeau, Gonzalez Rothi, & Rosenbek, in press; Plaut, 1996).

⁸ For example, refer to Martin and colleagues for a detailed hypothesis about the nature of LTP/LTD and their role in learning; specifically, this hypothesis addresses associative processes of learning, storage capacity, and the permanence of memories (Martin, Grimwood, & Morris, 2000).

principles of learning. Within the context of such a theory, hypotheses can be made about how variables of interest are likely to modulate the recovery process. Before such a complex theory of rehabilitation can be advanced, however, behavioral learning paradigms need to be systematically investigated in individuals with aphasia.

Repetition Priming: An Implicit Learning Paradigm

Learning can be broadly categorized as either implicit or explicit in nature. Explicit learning is defined as requiring conscious or controlled attention to the learning process and conscious recollection of prior learning experiences (Tulving & Schacter, 1990). Implicit learning, on the other hand, does not require intentional or conscious awareness of the learning process (i.e., recall and recognition). Instead, implicit learning is defined as an unconscious or automatic process⁹ that results in relatively abstract knowledge (Butler & Berry, 2004; Reber, 1989). Automatic processes of attention have been shown to be fast, effortless, and unavailable to conscious awareness, and are developed through practice (as discussed in Logan, 1988).

One type of implicit learning is priming, which results in faster and/or more accurate responses; priming occurs in response to a single exposure of a stimulus (Badgaiyan, Schacter, & Alpert, 2001; Henson, Shallice, & Dolan, 2000; Poldrack & Gabrieli, 2001; Tulving & Schacter, 1990). By definition, priming occurs in response to a single exposure of a stimulus, in the absence of explicit learning or controlled processes of attention (Badgaiyan, Schacter, & Alpert, 2001; Butler & Berry, 2004; Henson, Shallice, & Dolan, 2000; Poldrack & Gabrieli, 2001; Reber, Gitelman, Parrish, & Mesulam, 2004; Tulving & Schacter, 1990). This single exposure is not thought to contribute to expertise or automaticity of a particular skill (Reber, Gitelman, Parrish, & Mesulam, 2004). Repetition priming, on the other hand, refers to the priming effect(s) observed as a result of more than one presentation of a given stimulus (Reber, Gitelman, Parrish, & Mesulam, 2004; Schwartz & Hashtroudi, 1991; Tulving & Schacter, 1990). While a single prime may initiate the learning process via encoding, repetition priming, also referred to as *direct priming* (Tulving &

⁹ Shiffrin and Schneider conducted a series of studies differentiating automatic and controlled processes of attention (Shiffrin & Schneider, 1977).

Schacter, 1990), *identity priming* (Hutchinson, Neely, Neill, & Walker, 2004), or *item-specific priming* (Mitchell & Brown, 1988), has been proposed as an elementary mechanism of learning leading towards the development of automaticity that typically accompanies expertise; that is, expertise is hypothesized to develop after the second stimulus response (Poldrack, Wagner, Prull, Desmond, Glover, & Gabrieli, 1999; Reber, Gitelman, Parrish, & Mesulam, 2004). Although repetition priming has been shown to be independent of skill learning (Schwartz & Hashtroudi, 1991; van Turennout, Bielschewsky, & Martin, 2003), some believe that repetition priming shares the same underlying processes and neural substrates with skill learning (Dennis & Schmidt, 2003). The difference between repetition priming and skill learning appears to evince the level of learning; skill learning reflects a general improvement of a capability, while repetition priming is thought to reflect learning at the level of a stimulus item.

An Information-Processing Model of Repetition Priming

Spreading-activation or strength-based models (based upon the original PDP model by McClelland & Rumelhart, 1981) contend that priming temporarily increases the magnitude of activation of the item being responded to, thus allowing improved access to the stimulus on subsequent trials. Interactive activation models are usually discussed in the context of a particular cognitive process, such as lexical retrieval (Dell & O'Seaghdha, 1992; Martin, Fink, Laine, & Ayala, 2004; Nadeau, Gonzalez Rothi, & Rosenbek, in press) or facial recognition (Burton, Bruce, & Johnston, 1990). Burton and colleagues suggest that repetition priming reflects a strengthening of an excitatory connection between the stimulus node and the response node. Once the stimulus node has reached threshold (i.e., the correct response has been made), the strength of the connection between the stimulus and response nodes increases. Ostensibly, because residual activation of the first trial persists for a short time, the next trial should have a higher level of activation. Although the activation of the second trial will decay, the remaining residual activation should be stronger than when the item was first encountered. Thus, the connection between the stimulus and response nodes becomes increasingly stronger with subsequent correct trials. Essentially, the residual activation of the

connection increases with each trial, resulting in faster and less variable responses.¹⁰

Although this model of learning has been put forth as an hypothesis about the cognitive representations and processes underlying repetition priming, it remains underspecified, and is unable to address many of the behavioral characteristics of repetition priming (e.g., repetition is long lasting). Furthermore, most cognitive theories have yet to address or incorporate neurobiological substrates of learning and neural processes of reorganization following stroke into their models¹¹.

Behavioral Characteristics of Repetition Priming

Repetition priming experiments that investigate lexical retrieval typically use tasks such as picture naming or lexical decision tasks. Participants are presented with a stimulus item (e.g., picture, written word) and asked to do some task (e.g., picture-naming, lexical decision); this item is then repeated later in the experiment. Repetition priming experiments can be massed¹² (i.e., the stimulus is repeated in succession with no intervening stimuli) or spaced/distributed (i.e., the stimulus is repeated with one or several items intervening between trials). In traditional motor and/or skill learning paradigms, massed practice is assumed to occur within one training session, while spaced or distributed practice is defined as training sessions with one or more stimuli intervening between repetitions or some time interval between repetitions (Nadeau, Gonzalez Rothi, & Rosenbek, in press). Behavioral changes associated with repetition priming are observed as decreased latencies, improved accuracy (Cornelissen, Laine, Tarkiainen, Jarvensivu, Martin, & Salmelin, 2003; Henson & Rugg, 2003; Henson, Shallice, & Dolan, 2000; Tulving & Schacter,

¹⁰ Other connectionist authors have suggested that repetition priming reflects a permanent modification (reduction) of the threshold level for activation of the lexical representations (e.g., Morton, 1969). More generally, Nadeau and colleagues suggest that *learning* manifests in a connectionist network as changes in the strength of connections within a neural network; neural networks support knowledge (represented as long term memory at the level of connection strengths), working memory, and processing (Nadeau, Gonzalez Rothi, & Rosenbek, in press).

¹¹ Although, the recent Nadeau et al. chapter (in press) provides a more sufficiently detailed account and appears to be the first to integrate neurological substrates of linguistic functioning with cognitive models of memory (which include processes of long term memory and working memory) and behavioral and rehabilitative data.

¹² Note: Many investigators claim "massed" practice for intensive treatment (i.e., many trials) as opposed to many repetitions of the same trial.

1990; Wiggs & Martin, 1998) or reduced response variability (Dennis & Schmidt, 2003) across trials.

Persistence of Repetition Priming Although early theorists of repetition priming suggested that repetition priming was the result of “transient variations in activation level of pre-existing memory representations” (Versace & Nevers, 2003, pp. 389-390) and early studies indicated that priming effects were relatively transient in nature (e.g., Graf, Squire, & Mandler, 1984), decreased latencies have since been found to persist in unimpaired subjects for up to 48 weeks with only a single exposure to the stimulus (Cave, 1997; Durso & Johnson, 1979; Lachman & Lachman, 1980; Mitchell & Brown, 1988). For example, Cave (1997) investigated the duration of priming effects with a total of 204 non-brain-injured subjects, using a picture-naming paradigm. During the first session, each subject was presented with 130 black and white drawings (from Snodgrass & Vanderwart, 1980) and asked to name the pictures as quickly and accurately as possible. The subject pool was then divided into nine groups, each including 20-29 subjects. Each of these groups returned for a second session at various intervals (6, 8, 10, 12, 16, 24, 32, 40, and 48 weeks) relative to the initial session. Subjects were not informed that they would be seeing the same pictures at the later date. During the second session, subjects were presented with 200 drawings, including 100 of the previously seen pictures and 100 novel pictures. For all nine delay conditions, pictures named during the first session were responded to significantly faster than those pictures not previously named. The reaction time difference between naming previously seen and novel targets ranged from approximately 78 ms to 25 ms, with the mean difference decreasing as the delay interval increased. Thus, priming effects have been observed up to 48 weeks, but these effects are not equivalent across delay periods; that is, the priming effect decreases or decays across time.

Mitchell and Brown (1988) also investigated repetition priming by manipulating the duration between repetitions. The investigators conducted three separate experiments examining three different intervals: one week, four weeks, and six weeks. For all three experiments, non-brain-injured subjects ($n=60$) were presented with, and asked to name, 100 pictures during the initial session; during the second session participants were presented with 50 repeated pictures and 50

novel pictures. A recognition task was completed in addition to the naming task to assess explicit memory of the stimulus items. Results indicated that repetition priming led to facilitation of picture-naming, with priming effects ranging between 70 and 80 ms, regardless of the interval between sessions. In this study, the priming effect did not decay as a result of increasing the interval between sessions. Additionally, repetition priming was present regardless of whether the subjects remembered seeing the pictures or not, thus indicating that repetition priming is independent of explicit memory and learning processes.

In summary, repetition priming effects can persist up to 48 weeks¹³. However, the magnitude of the effect may be susceptible to manipulations of inter-stimulus interval. While the abovementioned research has provided conclusive evidence for a persistence of repetition priming effects across time, few studies have systematically examined the sensitivity of the priming effect to the number of intervening stimuli. In a recent study examining lexical decision in older, non-brain-injured adults and individuals with aphasia, Blumstein and colleagues found that repetition priming effects are relatively insensitive to the number of stimuli that intervene between the repeated trials, although the magnitude of change is greatest when no stimuli intervene between the first and second presentation (Blumstein, Milberg, Brown, Hutchinson, Kurowski, & Burton, 2000).

Sensitivity to the Number of Repetitions Although a significant amount of research has substantiated a positive relationship between the magnitude of learning (i.e., the number of trials) and subsequent learning and/or retention in the realm of motor learning (Keefe, 1995; Schmidt & Lee, 1999), the same relationship has been found to be less robust and relatively inconsistent in the context of implicit learning tasks such as repetition priming. Brown, Jones, and Mitchell (1988) explain this issue as a difference between “single test priming” during which the target stimulus is presented only once before the subject is probed, and “multiple test priming” during which the subject is presented with the target stimulus more than once before the test probe (p.160). The confusion surrounding this issue appears to stem from the myriad variables that may or may not be manipulated

¹³ For lexical decision studies that demonstrate long-lasting effects of repetition priming, refer to (Dannenbring & Briand, 1982; Kirsner & Smith, 1974; Scarborough, Cortese, & Scarborough, 1977).

during repetition priming tasks, including, at the very least: (1) the interval or duration between the repetitions (i.e., massed/blocked vs. spaced), (2) the number of repetitions (i.e., a large number of repetitions vs. a small number of repetitions); (3) the task itself (e.g., word fragment completion, word stem completion, perceptual identification, object decision, picture naming), (4) the stimulus materials (e.g., line drawings, words, photographs), and (5) the manner in which the stimuli are presented (e.g., low tech vs. computer presentation).

Brown, Jones & Mitchell (1988) find the discrepancies across studies to be noteworthy, and suggest that such inconsistencies may reveal something about the underlying nature of implicit learning mechanisms. As such, the authors conducted an investigation in which they varied the number of repetitions and the interval between repetitions during a picture naming task. Sixty-four non-brain-injured subjects were presented with 125 black-and-white line drawings (from Snodgrass & Vanderwart, 1980) and asked to name the pictures as quickly and accurately as possible. Subjects then participated in two additional sessions during which some pictures presented were novel and some were single repetitions of previously seen pictures; the number of previously seen and novel pictures was not reported. In total, the participants were presented with the pictures four times. Half of the subjects were administered the second and third session immediately after the initial session; the other half returned one week later for the second session and two weeks later for the third session. For the first repetition of the stimuli (i.e., second presentation), significant repetition priming effects were observed across both massed and spaced conditions. During the massed interval condition, additional significant decreases in latency were observed at the third but not fourth presentation. Results were less clear for the spaced interval condition; although the priming effect remained significantly greater for multiple presentations than for a single presentation, repetition priming effects decayed across weeks. Thus, for multiple presentations in the spaced interval condition, priming effects decay to a lesser degree than for single presentations. The authors did not comment on generalization to untrained items, but reaction times for naming novel items remained relatively stable across the course of the experiment (i.e., three sessions).

That is, unpracticed items were not responded to faster across the course of the experiment, providing further evidence that repetition priming is item-specific.

Reber and colleagues (Reber, Gitelman, Parrish, & Mesulam, 2004) investigated repetition priming in the context of object identification across 8 repetitions. Forty-two non-brain-injured individuals were presented with 72 pictures of everyday objects for 750 ms, with an inter-stimulus interval of two seconds. The participants were asked to determine whether each picture was a picture of a baseball or not via button press. Those pictures that were not baseballs (i.e., non-target stimuli) were presented 1, 2, 3, 4, 5, 6, 7 or 8 times during the session. Participants responded significantly faster to items that were repeated the second time than upon the initial repetition (an approximate 100 ms decrease in reaction time was observed from the first to the second presentation of the stimulus), but no additional significant decreases of reaction time were observed from the third to eight presentation. The authors proposed that eight repetitions is not enough to "establish behaviorally detectable expertise" during this object identification task because of the simplicity of the task. For an additional study using object identification, see also (Koutstaal, Wagner, Rotte, Maril, Buckner, & Schacter, 2001).

In summary, multiple test repetition priming has not been sufficiently examined. While a single repetition of the stimulus item has been shown to decrease reaction time and increase accuracy, the magnitude of continued behavioral priming is less clear across increasing numbers of repetitions. While Brown and colleagues (Brown, Jones, & Mitchell, 1996) suggest that manipulation of the repetition priming task itself (i.e., massed vs. spaced practice) is likely to influence the magnitude of priming effects observed across multiple repetitions, Reber and colleagues (Reber, Gitelman, Parrish, & Mesulam, 2004) recommend systematic investigation of multiple test repetition for greater than eight repetitions. The most obvious conclusion to draw from the extant literature is that the most significant priming effects are observed for the first and second repetitions, with subsequent repetitions beginning to plateau; however, priming effects may become significant after eight repetitions.

Generalization Repetition priming is assumed to be, by definition, item-specific; that is, repetition priming effects are not expected to facilitate response latency and accuracy for untrained items (refer to the previous discussion by Brown, Jones, & Mitchell, 1996). For example, in a word-fragment completion task, Schwartz and Hashtroudi, distinguished repetition priming from skill learning on the basis of a lack of generalization to untrained items in the case of repetition priming (Schwartz & Hashtroudi, 1991). At the perceptual level, repetition of the target stimulus with minor color or texture manipulations were not shown to influence priming effects during a picture naming task (Cave, Bost, & Cobb, 1996); however, Koutstaal and colleagues found that presentations of two different exemplars of the same target stimulus (i.e., two different pictures of an umbrella) resulted in moderate priming effects relative to novel pictures (Koutstaal, Wagner, Rotte et al., 2001). Although repetition priming is unlikely to generalize from trained to untrained items, a large number of trials (i.e., greater than 8 repetitions) has yet to be conducted in the context of lexical access.

Neural Substrates of Repetition Priming

Neuropsychological research, including non-human primate single-cell recordings (Desimone, 1996) and human functional neuroimaging studies (Dobbins, Schnyer, Verfaellie, & Schacter, 2004; Henson & Rugg, 2003; Henson, Shallice, & Dolan, 2000; James, Humphrey, Gati, Menon, & Goodale, 1999; Kassubek, Schmidtke, Kimmig, Lucking, & Greenlee, 2001; Poldrack & Gabrieli, 2001; Reber, Gitelman, Parrish, & Mesulam, 2004; van Turennout, Ellmore, & Martin, 2000), strongly suggests that repetition priming results in a reduced level of cortical activity, referred to as repetition suppression or neural priming. Repetition suppression was first submitted as a hypothesis in 1987 after Brown and colleagues found decreased neuronal activity in the inferior-medial temporal cortex in monkeys for repeated exposures to a familiar stimulus relative to the initial presentation of the stimulus (Brown, Wilson, & Riches, 1987). Since this original documentation, numerous studies have examined the behavioral characteristics related to repetition suppression. Wiggs and Martin review this extensive literature and summarize the properties of repetition suppression as follows (Wiggs & Martin, 1998, p. 229):

- (1) comparable to behavioral correlates of repetition priming, repetition suppression is stimulus specific;
- (2) comparable to behavioral correlates of repetition priming, repetition suppression is relatively insensitive to intervening stimuli;
- (3) comparable to behavioral correlates of repetition priming, repetition suppression is long lasting (up to 24 hours between presentations);
and
- (4) comparable to some behavioral evidence, repetition suppression is graded; that is, with each repetition, neurons demonstrate additional reduction in firing rate (this has been demonstrated up to eight presentations).

The specific cortical region demonstrating neural suppression appears to reflect the type of stimulus presented (e.g., pictures of objects vs. faces) and the modality of stimulus presentation (e.g., visual vs. auditory stimuli). For example, in the aforementioned study, Reber and colleagues observed repetition suppression while using fMRI to investigate object identification (Reber, Gitelman, Parrish, & Mesulam, 2004). Nine non-brain-injured participants were presented with pictures of everyday objects and asked to determine whether each picture was a picture of a baseball or not via button press. Non-target stimuli were presented 1, 2, 3, 4, 5, 6, 7, or 8 times during the fMRI session. Cortical areas were analyzed for changes in hemodynamic response activity (1) between the first and second presentation, and (2) beyond the second presentation. The posterior fusiform cortex of the right hemisphere demonstrated decreased activity across all eight presentations. A larger region, including both right and left fusiform cortices demonstrated a significant decrease from the first to second presentation. Only the right posterior fusiform cortex demonstrated an incremental decrease of activity across all eight presentations, indicating that this region may be responsible for object identification expertise. This study indicates that repetition priming is associated with reductions of cortical activity; additionally, the authors provide initial evidence that the neural correlates of repetition priming associated with several repetitions are significantly different from the neural correlates associated with a single repetition of a visual stimulus.

One theory that has been put forth to explain the relationship between behavioral and neural correlates of repetition priming suggests that repetition reflects a honing or tuning of stimulus representation in the cortex which allows information to be more readily available (Desimone, 1996; Schnyer, Dobbins, Nicholls, Schacter, & Verfaellie, 2006; Wiggs & Martin, 1998). Under this view, the neurons that demonstrate reduced activity are those that are no longer needed to identify the stimulus. That is, the neurons that continue to be activated have become more attuned to the stimulus as a result of repeated presentations, thus access to the information associated with the stimuli becomes easier. Subsequently the peripheral neural activity that was associated with the stimulus response is no longer needed for performing the task with that particular stimulus item. Desimone (1996) suggests that this mechanism is an inherent characteristic of neurons that exists to allow individuals to be able to quickly and efficiently recognize and identify previously encountered stimuli.

Repetition priming, as a behavioral learning paradigm, provides an optimal foundation upon which to develop a theory of learning for individuals with aphasia; most importantly, this paradigm does not require conscious, explicit cognitive processes during learning, thus minimizing the number of internal cognitive factors that are likely to vary across individuals with aphasia. Furthermore, repetition priming, as a fundamental mechanism of learning, is particularly important to theories of rehabilitation when considering treatment intensity, or more specifically, stimulus dosage as a component of treatment intensity¹⁴.

Many investigators have begun to focus on the overall intensity of aphasia treatment (for a recent review, see Bhogal, Teasell, Foley, & Speechley, 2003); however, no systematic investigations of stimulus dosage, for any treatment protocols, have been conducted. Animal models of motor learning, as well as associated models of cortical reorganization, are based on hundreds of trials per day over the course of several weeks (Keefe, 1995, p. 91), and yet an investigation of stimulus dosage remains conspicuously absent from the aphasia rehabilitation literature. Given the current trend towards intensive, constraint-induced aphasia

¹⁴ Since repetition priming has been shown in healthy participants to be item-specific, it is the optimal paradigm upon which to investigate the number of trials required to elicit maximum behavioral and/or neural changes in any treatment protocol.

treatment (CIAT) protocols, a programmatic study of stimulus manipulation is warranted. Repetition priming is, therefore, an ideal tool by which to incrementally investigate acquisition and maintenance of trained items during spoken language production.

LEXICAL RETRIEVAL DEFICITS FOLLOWING STROKE

Speaking is one of the most complex motor skills that humans can perform. Ideas and intents are conceptualized, subsequently translated into linguistic representations, and finally converted into a motor code suitable for execution by the muscles of the each of the subsystems involved during speech production. In addition to processing that can accommodate variables such as para-linguistic attributes, the output is continuously monitored to ensure that we have said what we intended to say in the manner we intended to say it. Amazingly, we are able to transform this intent into action at a rate of approximately 150 words per minute, or 2-3 words per second, with relative ease (Mcclay & Osgood, 1959).

Spoken language production has been modeled as a series of stages, across which the representation, or unit of processing, may vary (Garrett, 1980; Levelt, 1999a; Levelt, 1989; Rogers & Spencer, 2001). Most conservatively, the stages thought to be required for spoken language production include: conceptualization, formulation, and motor planning and execution. During conceptual preparation, the speaker's non-linguistic intent, or *lexical concept*, is generated. Although little is understood about how this intent is represented, it appears that a one-to-one mapping of lexical concept to message is absent; that is, speakers may approach the same message from a variety of different perspectives, combining internal cognitive input and external, environmental stimuli to formulate the intent for output (Levelt, 1989). This abstract intent is then converted into a linguistic code during formulation, at which time words are selected, syntactic frames are constructed, and phonological forms are specified. Formulation can be further subdivided: during grammatical encoding, word meaning and syntactic form are processed; the unit of representation at this level is referred to as the *lemma*. During phonological encoding, the abstract representation of the word form is retrieved; the representational units, or *lexemes*, of the sound, syllable, and

segment are built from stored morphological and phonological information. During phonetic encoding the allophonic and contextual phonetic adjustments are specified (e.g., aspiration), accounting for the fact that words are produced in a contextually sensitive manner. The output of formulation is then translated into a code suitable for motor execution. During motor planning, motor goals for each phoneme are specified and sequentially organized. During motor programming, neuromuscular, and, perhaps, aerodynamic parameters are set to determine movement direction, force, velocity, range, and rate for each speech subsystem (i.e., respiration, phonation, resonance, articulation and prosody). Finally, motor execution takes place and the linguistic form is uttered.

Although the functional characteristics of the levels or stages of processing necessary for spoken language production are generally agreed upon across researchers and to some degree across various models of spoken language production (Dell & O'Seaghdha, 1992), the temporal nature is less well understood. Additionally, the interaction between these stages, or lack thereof, remains highly debated. Discrete serial models propose that processing occurs one stage at a time (i.e., they are temporally and functionally distinct) with the absence of feedback loops (Fromkin, 1971; Garrett, 1975; Levelt, 1989). Such models consider the various stages of processing as modular in nature; for example, phonologic encoding is not initiated until semantic encoding has been completed, and so forth. In this regard, stages models necessarily assume feed forward processing¹⁵.

Interactive spreading activation models, on the other hand, contend that processing occurs in an overlapping or parallel manner, and feedback mechanisms allow for the bidirectional flow of information between all adjacent functional stages (Dell, 1986; Dell, 1988; Dell & O'Seaghdha, 1992; Dell, 1991); as such, interactive activation models have been conceptualized as "globally modular, but locally interactive" (Dell & O'Seaghdha, 1992, p. 604). Interactive spreading activation models propose that as a conceptual item is activated from external input, it projects simultaneous activation both to semantic and phonological units for encoding. The most activated units are then selected and inserted into assembly frames which are then encoded for motor programming and motor execution. Subsequent to

¹⁵ Levelt's 1989 model assumes bidirectional flow between the lexical concept and lemma but nowhere else during the stages of spoken language production.

selection of the most activated units, post-selection inhibition momentarily resets the units' activation levels to zero to prevent reselection of the same unit.

Most studies of spoken language production in individuals with aphasia appear to assume either a feed-forward stage model that permits cascading processing (e.g., Rogers, Jones-Redmond, & Alarcon, 1999) or an interactive activation model that relies less on the sequential activation of the various levels of processing and more on the interconnectivity between levels of processing (e.g., Martin, Fink, Laine, & Ayala, 2004). Regardless of the nature of interaction between the stages, the onset of semantic encoding has been reliably shown to occur prior to the onset of phonologic encoding in both healthy controls and individuals with aphasia (e.g., Rogers, Jones-Redmond, & Alarcon, 1999). Evidence to support this two-stage model stems from several lines of research including speech errors studies (Brown & McNeil, 1966; Fromkin, 1971; Garrett, 1980; Vitevitch, 1997), behavioral studies (Calkins, 2003; Levelt, 1991; Rogers, Jones-Redmond, & Alarcon, 1999; Rogers & Storkel, 1999; Schriefers, Meyer, & Levelt, 1990); and functional imaging studies (Chertkow, 1997; Demonet & Thierry, 2001; Indefrey & Levelt, 2000; Levelt, 1998; Savoy, 2001; Schmitt, Munte, & Kutas, 2000; Schmitt, Schiltz, Zaake, Kutas, & Munte, 2001; van Turennout, Hagoort, & Brown, 1997; van Turennout, Hagoort, & Brown, 1998).

Methods of Investigating Lexical Retrieval

Off-Line Methods

Historically, psycholinguistic research relied upon offline methods to test hypotheses and models of lexicalization (e.g., Fromkin, 1971). By definition, offline methods collect data on the end products of language and cognitive processing, not the real-time processes. Consequently, this line of research is unable to probe the temporal features of processing, resulting in limited and possibly confounded information about the origin/locus of various processing mechanisms. The most prevalent offline tasks include *speech error studies* and examinations of the tip-of-the-tongue phenomenon. Offline observations have generated a significant corpus of information about post-lexical processing, but data about the time course of processing is lacking. Furthermore, it has been argued that theories of normal

language production should not be solely based upon these occasional aberrations (Levelt, 1999b; Levelt, 1991; Meyer, 1992). Accordingly, offline methods do not provide a complete picture of the processes underlying spoken language production.

On-Line Methods

Reaction Time Studies Reaction time (RT) studies are used to approximate real-time processing of spoken language production across a variety of language-related tasks. Although RT studies cannot continuously record temporal processing mechanisms for spoken language production from start to finish, they are instead used to systematically probe the stages of lexicalization at short intervals (on the order of milliseconds). Reaction time studies include but are not limited to pronunciation tasks, picture-naming tasks, word-reading tasks, lexical-decision tasks, repetition priming tasks, form-based priming tasks, dual-task paradigms, and cross-modal interference studies.

Neuroimaging Studies Online methods afford researchers the opportunity to probe the process of lexicalization at systematic time intervals (on the order of milliseconds) during the act of spoken language production (Shapiro, Swinney, & Borsky, 1998). The most noteworthy examples of online methods include electroencephalography (EEG) and magnetoencephalography (MEG). Electroencephalography records the electrical currents of the brain, or event-related potentials (ERPs), during a cognitive task by positioning electrodes upon the scalp. Event related potentials directly reflect the electrical activity of the outer cortex (presumably from cortical pyramidal cells), thereby revealing a real-time temporal record of neural processing. Magnetoencephalography also provides real-time assessment by measuring electromagnetic energy that emanates from the cortex.

While these methods result in detailed temporal information about neural processes (on the order of milliseconds), the spatial resolution and source information is relatively poor; that is, it is difficult to identify the exact source or groups of cells that produces the electrical activity. Some laboratories, however, continue to investigate the feasibility and accuracy of co-registration techniques used to merge structural imaging procedures (e.g., MRI, CT) with these temporally

resolute techniques (e.g., EEG, MEG) to gain more specific information about the cortical location of neural activity at specific temporal intervals (Clark, Moores, Weber, Fitzgibbon, Greenblatt, Brown, & Taylor, 2001). Quasi-online functional imaging techniques include functional magnetic imaging (fMRI) and positron emission tomography (PET). While fMRI is typically used to glean information about the neural location of various processing mechanisms, it may eventually prove to be somewhat sensitive to the temporal domain as well. Positron emission tomography, however, provides only an examination of neural mechanisms in respect to location; the temporal window is on the order of minutes.

Generally, aphasiologists employ off-line methods, most frequently of which include error analyses (e.g., type of errors, frequency of errors, and relative proportion of errors). Although psycholinguistic researchers frequently use reaction time in addition to error analyses to assess the underlying linguistic impairment in individuals with aphasia (Wilshire, Keall, Stuart, & O'Donnell, 2005; Wilshire, Scott, & Stuart, 2006), few treatment protocols rely on reaction time data as a dependent measure, and even fewer clinicians are technologically equipped to collect reaction time data during treatment probes. More recently, neuropsychological researchers have begun to use fMRI, ERP, and MEG studies to examine both the underlying neural characteristics associated with impairment and the neural characteristics associated with treatment. Collectively, these offline and online methodologies reflect dependent measures currently used in the studies that will be reviewed in the forthcoming sections of this dissertation. The project discussed in forthcoming sections employed reaction time methodology.

Anomia: Lexical Retrieval in Impaired Linguistic Systems

Anomia, the most ubiquitous characteristic of aphasia (Benson, 1988; Maher & Raymer, 2004), is considered to be a disorder of lexical retrieval, not a loss of lexical representations (Avila, Lambon-Ralph, Parcet, Geffner, & Gonzalez-Darder, 2001). Currently, stage models of spoken language production (e.g., Dell, 1986; Garrett, 1980; e.g., Levelt, Roelofs, & Meyer, 1999) motivate investigations designed to better understand the underlying nature of naming impairments produced by aphasic individuals. Both discrete serial and interactive-activation

models suggest that anomia stems from an impairment(s) of: (1) conceptual preparation (also referred to as a general semantic impairment), (2) semantic encoding (also referred to as an impairment of lexical selection), and/or (3) phonological encoding. Anomia is not predicted to manifest as a result of impairment at the levels of phonetic encoding, motor planning and/or motor programming (e.g., apraxia of speech) or motor execution (i.e., dysarthria). More recently, aphasiologists have hypothesized that processing mechanisms existing between semantic encoding and phonological encoding may also bring about naming deficits (Chiarelli, Menichelli, & Semenza, 2006; Wheeldon & Monsell, 1992). In light of these theories of linguistic impairment in individuals with aphasia, anomia treatment protocols are designed, in general, to improve spoken language production in brain-injured individuals by increasing the likelihood that lemmas and lexemes within the lexicon are successfully retrieved for production purposes (e.g., Nickels, 1995a).

Behavioral Characteristics of Anomia

Historically, aphasiologists have described and investigated two primary manifestations of anomia: (1) general semantic anomia resulting from impairments at the level of conceptual preparation, and (2) output anomia resulting from impairment at the levels of semantic and/or phonological encoding (Benson, 1988; Geschwind, 1967; Hillis, Chaudhry, Davis, Kleinman, Newhart, & Heidler-Gary, 2006; Maher & Raymer, 2004; Wilshire & Coslett, 2000). Individuals with general semantic anomia produce errors across all output modalities including spoken language and writing, in addition to corresponding receptive (i.e., comprehension) deficits.

Output anomia, on the other hand, reflects impairments at the level of lexical retrieval or phonological encoding, manifesting solely during spoken language production tasks, in the absence of accompanying comprehension deficits (Maher & Raymer, 2004; Nickels, 1995b; Wilshire & Coslett, 2000). That is, individuals with output anomia have complete access to the lexical concept and many of its semantic associations and connections to semantic encoding, but these patients demonstrate deficits at the level of the lemma or lexeme, and/or may have

impairments that disrupt the processes involved between retrieval of the lemma and retrieval of the lexeme. To narrow the scope of the anomia literature reviewed in this paper, and to focus solely on disorders of spoken language without accompanying receptive deficits, only output anomia will be explored in further detail.

Two primary types of output anomia have been proposed in the context of stage models of spoken language production: (1) impairment at the level of semantic encoding, and (2) impairment of phonological encoding (Lambon-Ralph, Moriarty, & Sage, 2002; Maher & Raymer, 2004; Nickels, 1995b; Wilshire & Coslett, 2000). Furthermore, several researchers have suggested that lexical retrieval deficits may also stem from an impairment of the processing mechanism(s) that occur between lemma retrieval and lexeme retrieval. For example, although a patient may demonstrate intact semantic knowledge (i.e., they can describe the semantic features of the word in question including grammatical class, etc.) and may have intact phonological knowledge about the word (i.e., they can tell you what the word rhymes with and may be able to repeat the word when given a model) they cannot name the item. Authors who assume stage models of spoken language production have hypothesized that impairment at the level of semantic encoding should predict the elicitation of semantic errors, (i.e., literal or semantic paraphasias¹⁶), and impairment at the level of phonological encoding should predict phonological errors (i.e., formal or phonemic paraphasias¹⁷). However, the type of errors made by individuals with anomia does not appear to directly correlate with the underlying linguistic impairment. Abel and colleagues have suggested that the lack of predictable errors may result from (1) mixed impairments, (2) a potentially interactive nature of processing between hypothesized levels of impairment, and/or (3) methodological issues including incorrectly diagnosing the underlying linguistic impairment, or that the stimuli used may inherently contain both semantic and phonological aspects (Abel, Grande, Huber, Willmes, & Dell, 2005).

¹⁶ Semantic paraphasias typically manifest as lexical items that relate to the target word as follows: (1) coordinate of the target word (e.g., "cat" for "dog"), (2) superordinate to the target word (e.g., "animal" for "dog"), (3) subordinate to the target word (e.g., "Labrador" for "dog"), or (4) some other semantic relationship (e.g., "leash" for "dog").

¹⁷ Phonological naming errors are typically referred to as formal or phonemic paraphasias, and typically manifest as an off-target form of the intended word (e.g., "dilib" for "dog").

To better understand anomic errors in relation to the underlying linguistic impairment, Lambon-Ralph and colleagues conducted a well-designed case-series study investigating 21 individuals with mild to severe anomia (Lambon-Ralph, Moriarty, & Sage, 2002). Of these 21 individuals, 16 demonstrated mild semantic deficits, and all but one demonstrated some degree of phonological deficit, as demonstrated by a detailed language battery. Lambon-Ralph and colleagues coded naming errors, based on a 100-item picture-naming task; errors were coded as: (1) omissions, (2) semantic errors (i.e., coordinates, superordinates, and associates), (3) circumlocutions, (4) phonological errors (i.e., phonologically related words or nonwords), or (5) other (e.g., visually related errors or gestures). The authors found significant correlations between the following errors and underlying linguistic impairments:

- (1) omissions significantly correlated with the degree of semantic impairment (i.e., aphasics demonstrated an increased number of omissions with increased severity of the semantic impairment);
- (2) phonologically and unrelated nonword responses significantly correlated with the degree of phonological impairment (i.e., aphasics demonstrated increased number of nonword errors with increased severity of the phonological impairment); and
- (3) semantic errors significantly negatively correlate with phonological ability (i.e., as the phonological impairment becomes more severe, the number of semantic errors decreases).¹⁸

Lambon-Ralph and colleagues concluded that these anomic individuals were primarily characterized by phonological deficits, with accompanying mild semantic deficits; however, others report primary deficits of semantic encoding with more semantic than phonological errors (Davis, Farias, & Baynes, 2005; Howard, Patterson, Franklin, Morton, & Orchard-Lisle, 1984). While the discrepancies observed across studies may result from variability of diagnostic procedures, it is more likely that subject variability is at the heart of the matter.

¹⁸ The authors posit that as the phonological impairment becomes more severe, fewer semantic errors are observed because phonological errors become so prevalent that they mask the semantic errors. Phonological errors and unrelated errors were too few to analyze relative to underlying linguistic impairments.

Lexical retrieval by anomic individuals is behaviorally characterized by reduced accuracy and increased latency, relative to healthy participants (Moreno, Buchanan, & Van Orden, 2002; Wilshire, Keall, Stuart, & O'Donnell, 2005). As was discussed above, individuals with anomia produce a variety of errors including omissions, perseverations, semantic paraphasias, unrelated lexical errors, phonological paraphasias, and neologisms. However, individuals with anomia make qualitatively similar errors as compared with healthy participants (Silkes, McNeil, & Drton, 2004); that is, the proportion of error types made by individuals with aphasia is comparable to that of healthy participants¹⁹. Furthermore, anomic individuals are likely to produce approximately the same proportion of errors across naming tasks, despite the fact that they demonstrate significant individual variability across trials (Howard, Patterson, Franklin, Morton, & Orchard-Lisle, 1984; Moreno, Buchanan, & Van Orden, 2002). Howard and colleagues further detail the error performance of individuals with anomia:

- (1) Group data revealed that, collectively, individuals with anomia are highly variable in regards to proportions of error types; that is, each anomic individual displays his/her own pattern of error type;
- (2) Group data revealed that proportions of each error type are not "systematically related to the diagnostic categories or the severity of the naming impairment" (p.270);
- (3) Group data revealed that, collectively, individuals with anomia are more likely to name a picture accurately on successive trials if they named it correctly the first time, relative to items they failed on the first attempt;
- (4) Individual anomic errors are not influenced by sequences of easy or hard-to-name items (i.e., there is no effect of success or failure on one item to the one that follows);
- (5) Individual subjects demonstrate significant variability in regards to their naming accuracy across trials (i.e., that is, anomic individuals may not always name the same item correctly); and

¹⁹ Silkes et al. found a higher proportion of semantic errors for both individuals with aphasia (55%) and healthy participants (70%).

- (6) Individual subjects are likely to maintain the relative proportion of correct/incorrect responses across naming tasks.

Collectively, these studies demonstrate that anomie errors are not particularly reliable indicators of the underlying linguistic impairment. Therefore, investigations that examine lexical retrieval of individuals with aphasia need to include a detailed assessment battery that includes general aphasia tests in addition to a multitude of lexical retrieval tasks that are designed to tease apart semantic encoding deficits from phonological encoding deficits. Please see table 1.2 for a sample of informal tasks and formal assessments that address the respective stages of processing (Boyle, 2004; Martin, Fink, Laine, & Ayala, 2004; Morrow & Fridriksson, in press)²⁰.

Table 1.2 Informal Tasks and Formal Measures of Encoding Deficits

General Aphasia Batteries	Semantic Encoding Tasks	Phonological Encoding Tasks
Western Aphasia Battery (WAB) (Kertesz, 1982)	Peabody Picture Vocabulary Test (PPVT) (Dunn & Dunn, 1981)	Oral Picture Naming: Boston Naming Test (Kaplan, Goodglass, & Weintraub, 2001) Philadelphia Naming Test (Roach, Schwartz, Linebarger, Martin, & Bochetto, 1988)
Boston Diagnostic Aphasia Battery (BDAE) (Goodglass & Kaplan, 1972)	Word-Picture Matching w/Semantic Distractors (both spoken and written word to picture matching)	Written Picture Naming
Reading Comprehension Battery for Aphasia (RCBA) (LaPointe & Horner, 1979)	Synonymy Judgments	Oral Word Reading
	Semantic Category Sorting	Writing Words to Dictation
	Pyramids and Palm Trees (Howard & Patterson, 1992)	Repetition of Single Words
		Phoneme Discrimination
		Auditory Rhyme Judgments

Neural Substrates of Lexical Access

Currently, aphasiologists use one of the following methodologies to identify the neural substrates of lexical retrieval during spoken language production: (1) lesion data in combination with behavioral data obtained from individuals with aphasia (e.g., Geschwind, 1967), (2) neuroimaging data in combination with

²⁰ Please note: many of these informal tasks have been formalized within the Psycholinguistic Assessments of Language Processing in aphasia (PALPA) (Kay, Lesser, & Coltheart, 1992).

descriptions of cognitive-linguistic impairments or behavioral treatment data obtained from individuals with aphasia, or (3) neuroimaging data in combination with descriptions of cognitive-linguistic behavioral from healthy participants²¹. Collectively, lesion and neuroimaging studies have provided significant detail about the spread of activation from more posterior regions (i.e., occipital activation) during perceptual and visual object recognition processes, to more anterior regions (i.e., left parietal and temporal lobes) during semantic encoding through motor execution (for a detailed review, see Whatmough & Chertkow, 2002)²². For example, in a MEG study, Levelt and colleagues mapped out the time course and cortical regions associated with processes associated with picture-naming in healthy controls (Levelt, 1998). The authors found occipital, parietal (both right and left) and infrequently, temporal activation during lemma selection. During phonological encoding the authors found activation near the left posterior third of the superior temporal gyrus and the left temporo-parietal junction (i.e., Wernicke's area). Finally, during phonetic encoding and motor execution, the authors found widespread activation with the largest magnitude of activation in the motor cortex and in the parietal and temporal lobes.

Most recently, using MRI with diffusion-weighted images (DWI) and perfusion-weighted images (PWI), Hillis and colleagues found that general semantic errors spanning both naming and comprehension were correlated with hypoperfusion or infarct of Wernicke's area and the anterior inferior temporal cortex, while output anomia was highly associated with hypoperfusion or infarct of the posterior middle inferior temporal and fusiform gyrus (Hillis, Chaudhry, Davis et al., 2006).

Recent neuroimaging studies examining the neural correlates associated with anomia recovery and/or rehabilitation provide yet another approach to better

²¹ A discussion of the strengths and limitations of these methodologies is beyond the scope of this paper; however it should be noted that neither offers a complete picture of the underlying neural networks and/or mechanisms necessary to characterize cognitive processes.

²² Whatmough & Chertkow (2002) found the following areas of interest: semantic processing = posterior left temporal lobe (lesion data) vs. left frontal activation (imaging data); phonological encoding = middle gyrus of the left temporal lobe, left posterior inferior frontal lobe, anterior inferior parietal area. The authors provide evidence that the variability of activation within these stages of processing stems from slightly different types of impairments.

understanding the neural structures associated with lexical retrieval. Using a multiple baseline fMRI study (i.e., 3 scans pre-treatment and 3 scans post-treatment) that examined the effects of an intense phonological naming treatment, Fridriksson and colleagues found increased activation in the left temporal and parietal lobes after treatment, whereas the left inferior frontal lobe, the right temporal lobe and the right motor cortex were activated prior to treatment (Fridriksson, Morrow-Odom, Moser, Fridriksson, & Baylis, 2006). These findings support the above lesion and neuroimaging data revealing left temporal and parietal activation during lexical retrieval tasks (see also Cornelissen, Laine, Tarkiainen et al., 2003 who found left inferior parietal lobe activation (perilesional) following treatment).

Variables to Consider: Investigating Individuals with Aphasia

Subject Variables

Subject variables thought to influence aphasia recovery have had a long history of investigation, with a significant amount of attention given to the examination of the relationship between the location and/or size of the lesion relative to aphasia severity and subsequent recovery from aphasia. For example, Kertesz and colleagues examined 70 individuals with aphasia and correlated their aphasia severity, language performance, and language recovery as measured by the Western Aphasia Battery (WAB) Aphasia Quotient (AQ), to lesion location and lesion size using computed tomography (CT) scans (Kertesz, Harlock, & Coates, 1979). These authors grouped 70 stroke survivors based upon their aphasia type and severity, as determined by the WAB classification of aphasia and WAB AQ, respectively. These subgroups included: chronic global aphasia; chronic Broca's aphasia; acute Broca's aphasia; chronic anomic aphasia – recovered from initial Broca's aphasia; acute Wernicke's aphasia; chronic Wernicke's aphasia; acute transcortical sensory aphasia; chronic conduction aphasia; acute anomic aphasia; chronic anomic aphasia; non-dominant lesions with constructional apraxia; and non-dominant lesions without constructional apraxia. Collectively across these subgroups, Kertesz and colleagues found a significant negative correlation ($r = -0.57$), indicating that the larger the lesion, the more severe the aphasia. Of the participants diagnosed with acute anomic aphasia ($n=8$), CT scans revealed that

several participants had small lesions in the frontal operculum and third frontal convolution (i.e., Broca's area), while others had small temporal lesions. Computed tomography scans for the participants with chronic anomic aphasia (n=13) revealed both anterior and poster lesions in the left hemisphere. Collectively, individuals with acute and chronic anomic aphasia had the highest degree of correlation between the WAB AQ and lesion size. That is, for individuals with anomic aphasia, the lesion size appeared to be more important in regards to the severity of the impairment than the lesion location itself. Kertesz and colleagues also found that for individuals with naming impairments, larger lesions resulted in a smaller degree of recovery as measured by the WAB AQ 1 year after the initial CT scan. As these findings suggest, it is of utmost importance to adequately describe the lesion size, location, and severity of aphasia when investigating individuals with anomic aphasia and their expected recovery.²³

Naeser and Palumbo agree that the size of the lesion is important, but also suggest that lesions other than very large lesions or very small lesions are difficult to reliably correlate to prognosis or aphasia severity (Naeser & Palumbo, 1994). Instead, the authors strongly suggest that aphasia studies should provide detailed descriptions of both the size and location of the lesion by using CT scans or structural magnetic resonance imaging (MRI). In addition to providing information about the lesion size and location for their participants, aphasiologists should provide an explicit description of the spared tissue surrounding the lesion in known left hemisphere language areas, as this intact tissue is a primary candidate for reorganization of function during stroke recovery (Cramer & Bastings, 2000; Herholz & Heiss, 2000; Naeser & Palumbo, 1994). For further examples of studies that correlate a large lesion to increased severity of aphasia and poor recovery see also (Goldenberg & Spatt, 1994; Naeser, Helm-Estabrooks, Haas, Auerbach, & Srinivasan, 1987; Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1995;

²³ Please Note: Values for lesion size were not provided in the Kertesz et al article; i.e., "small vs. large" lesions are not adequately defined. Goldenburg and Spatt (1994) determine lesion size by "...the number of pixels covered by the redrawn lesion on the standard template. It is expressed as the percentage of the number of pixels covered by all left hemisphere templates together" (Goldenberg & Spatt, 1994, p.686). Naeser and Palumbo (1994) define lesion size by percentage of tissue in the left-hemisphere that is damaged.

Selnes, Niccum, Knopman, & Rubens, 1984). For an additional early study examining size and, to a greater extent, site of lesion using CT scans see Mazzocchi and Vignolo (Mazzocchi & Vignolo, 1979); individuals with naming impairments were not specifically reported in this study, thus results are not discussed in this paper.

Lexical Variables

In addition the underlying impairment and associated neuroanatomical deficits in individuals with anomia, lexical variables are likely to influence naming performance (Maher & Raymer, 2004; Nickels, 1995b). For impairments at the level of semantic encoding, the semantic category (e.g., animals, tools, fruits, vegetables) and/or grammatical class (e.g., nouns vs. verbs) may influence response time, accuracy, and response stability (Pashek & Tompkins, 2002). For impairments at the level of phonological encoding, lexical variables such as word frequency and/or familiarity, and word and/or syllable length may influence response time, accuracy, and response stability (Moreno, Buchanan, & Van Orden, 2002). Moreno and colleagues suggest that variability of reaction time is, in fact, a hallmark of anomic performance. Lexical variables including word frequency, word and/or syllable length, and the relative abstractness or concreteness of nameable pictures have been shown to differentially influence naming accuracy and reaction time for both healthy control subjects and individuals with anomia. Consequently, these variables are either controlled or actively manipulated in anomia studies, but are rarely considered in daily clinical practice. A discussion of how lexical variables are thought to influence anomic naming performance is provided in the following sections²⁴.

Word Frequency *Word frequency* is the most commonly manipulated variable for both psycholinguistic studies involving healthy participants and

²⁴ Repetition priming effects are thought to arise during phonological encoding. The same has been hypothesized for the word frequency effect and the word length effect (Barry, Hirsch, Johnston, & Williams, 2001; Nickels, 1995a). Furthermore, repetition priming may interact with word frequency. As such two lexical variables will be discussed: word frequency and word length. Variables such as imageability, thought to influence processing during semantic encoding will not be addressed in this paper. Notably, Raposo and colleagues did not find a differential influence of repetition priming for abstract vs. concrete nouns (Raposo, Moss, Stamatakis, & Tyler, 2006).

individuals with lexical retrieval deficits. Initially documented by Oldfield & Wingfield (Oldfield & Wingfield, 1965), this robust effect demonstrates that frequently used words or high frequency (HF) words are responded to faster and more efficiently than infrequently used or low frequency (LF) words (Oldfield & Wingfield, 1965), with measurable frequency effects ranging from three milliseconds (Levelt, 1998) to 60 milliseconds (Jescheniak & Levelt, 1994) in spoken language production protocols including healthy participants²⁵. Although the *Word Frequency Effect (WFE)*²⁶ has been substantiated across experimental tasks including *picture naming* (Alario, Ferrand, Laganaro, New, Frauenfelder, & Segui, 2004; Dell, 1990; Vitevitch, 1997), lexical decision and word recognition (Andrews & Heathcote, 2001; Bowers, 2000), and pronunciation (Grainger, 2000) for healthy participants, some debate exists about how robust the effect is for individuals with aphasia (Nickels & Howard, 1995). Low frequency words have been shown to be more susceptible to error than HF words for both healthy participants (Dell, 1990; Vitevitch, 1997), and individuals with aphasia (Nickels & Howard, 1995). Furthermore, Moreno and colleagues found that the variability of response times is greater for LF words than it is for HF words in individuals with aphasia (Moreno, Buchanan, & Van Orden, 2002). However, Nickels and Howard conducted two experiments (the second of which was a replication study with a new set of participants and a new set of stimuli) in which they assessed picture-naming in individuals with aphasia (n=12; n=15, respectively), controlling the stimuli for word frequency, familiarity, age of acquisition, and word length (Nickels & Howard, 1995). Surprisingly, the authors only obtained a significant word frequency effect for two of these 27 participants. That is, generally speaking, LF and HF words were responded to equally well. The same results, however, did not hold true for word *familiarity*²⁷; that is, more familiar words were responded to faster than less familiar words for most participants. However, the

²⁵ The most common measure of word frequency comes from the Francis and Kucera database that obtained word counts from newspapers (Francis & Kucera, 1982).

²⁶ Various cognitive models have been put forth to explain the locus of the WFE, but a discussion of these hypotheses is beyond the scope of this paper. A common hypothesis is that the WFE arises during phonological encoding (Jescheniak & Levelt, 1994; Monsell, 1991).

²⁷ Familiarity is a subjective measure purported to be a better reflection of true spoken word frequency than is the Francis and Kucera database; notably, however, familiarity was not found to be a significant predictor of naming speed in a recent study of 46 healthy participants (Alario, Ferrand, Laganaro et al., 2004).

authors found that when they entered age of acquisition into the statistical model, neither word frequency nor word familiarity were accurate predictors of naming success. Word length was also not predictive. The authors concluded that the age at which an individual acquires the target word is most predictive of naming accuracy for these two heterogeneous groups of individuals with aphasia. Recent evidence supports this claim, suggesting that age of acquisition can predict picture-naming accuracy and latency for both individuals with aphasia and healthy participants (Alario, Ferrand, Laganaro et al., 2004), and many investigators argue that the WFE is rooted in the age at which a word is required (Hirsh & Ellis, 1994; Morrison, Ellis, & Quinlan, 1992). At present, it appears that a significant interconnectivity exists between word frequency, word familiarity and age of acquisition; therefore each variable should be controlled for in studies of lexical access and retrieval.

Word Length/Number of Syllables For both individuals with aphasia (for a review, see Nickels, 1997) and healthy participants (e.g., Alario, Ferrand, Laganaro et al., 2004), increasing word length is tightly coupled with slower response times and a larger number of errors. For example, Nickels asked 15 individuals with aphasia to name 130 pictures that varied across word frequency and syllable length (Nickels, 1995a; Nickels & Howard, 1995). Nickels found length effects for phonological errors but not semantic errors, thus supporting her hypothesis that word and/or syllable length influences production at the level of phonological encoding. Notably, the influence of word frequency on naming performance was inconsistent across subjects. Nickels also proposed that impairments originating somewhere between phonological and phonetic encoding may manifest as difficulties with words of increasing length and phonetic complexity (Nickels, 1995b).

Although manipulation of these two lexical variables is undoubtedly important for the systematic study of lexical access and retrieval for individuals with anomia, individuals with aphasia appear to be extremely variable in regards to the influence that these lexical variables have on their own naming performance. For example, Howard and Gatehouse found significant variability across subjects: for one subject imageability and familiarity influenced naming performance, for a second frequency and familiarity influenced naming performance and for a third

subject only imageability influenced naming performance (Howard & Gatehouse, 2006).

Anomia Treatment Research

Anomia treatment efficacy research is composed primarily of phase I and II investigations (Robey, 2004; Robey & Schultz, 1998), characterized by single-subject studies or group designs including fewer than 20 participants, and most of which, by definition, lack control, blinding, and/or randomization procedures (Robey, 1998; Robey & Beeson, 2005). Anomia efficacy studies, however, consistently indicate that treatment improves picture-naming abilities. Robey and Beeson's recent systematic review of 19 qualifying lexical retrieval treatment studies indicates that treatment brings about large improvements (i.e., an average effect size of 7.27), whereas spontaneous recovery results in an average effect size of 0.6 (Robey & Beeson, 2005).²⁸ Given such large and consistent indicators of behavioral change, investigators are safe to assume that anomia treatments are, by and large, efficacious; however, many of the variables considered in Phase II research have yet to be systematically investigated. These treatment variables must be detailed before the field can move forward into Phase III clinical trials.

Anomia treatment can be classified as restitutive, substitutive, or compensatory in nature (Maher & Raymer, 2004). Restitutive approaches attempt to restore the process of lexicalization, as close as possible, to its original state by using (re)learning techniques. Substitutive approaches attempt to train the individual with anomia to use an alternative process(s) to gain access to, and subsequent retrieval of, lexical items. Finally, compensatory approaches train individuals with anomia to use alternative communication modalities (e.g., writing, drawing, gesturing) to express themselves when they experience word-finding difficulties. As one of the goals of this paper is to better understand the mechanisms underlying (re)learning in individuals with anomia, only restitutive approaches will be considered.

²⁸ Based on this systematic review, Robey and Beeson provide reference standard effect sizes for future lexical retrieval treatment protocols: small (4.0), medium (7.0), and large (10.0).

Many clinicians and researchers involved in restitutive anomia rehabilitation assume a stage model of spoken language production and hypothesize that targeting the appropriate level of impairment will result in faster, more accurate, and less variable production of inconsistently produced or unused lexical items (Best, Howard, Bruce, & Gatehouse, 1997; Boyle & Coelho, 1995; Martin, Fink, Laine, & Ayala, 2004). As discussed above, linguistic impairments resulting from anomia are thought to vary depending on the stage or stages of processing that are disrupted. As such, anomia treatments have largely focused on various protocols to improve semantic processing, phonological processing, or to improve the efficiency of the processes that occur between lemma and lexeme retrieval, ostensibly by initiating the automatic spread of feed-forward activation to the level or levels of processing that are thought to be impaired (e.g., Wible, Han, Spencer, Kubicki, Niznikiewicz, Jolesz, McCarley, & Nestor, 2006).

Numerous treatment approaches have been proposed to improve output anomia.²⁹ Treatment approaches that hope to improve semantic encoding are more widely varied than those that seek to improve phonological encoding. Collectively, across both semantic and phonological treatment paradigms, the single most common method of facilitating improved naming performance involves some form of cueing, which is thought to prime the target word and subsequently increase access to and subsequent retrieval of the hypothesized level of impairment or encoding mechanism. Specifically, these include *semantic cueing* (Boyle & Coelho, 1995; Drew & Thompson, 1999; Kiran & Thompson, 2003; Nickels & Best, 1996), and *phonologic cueing* (Best, Howard, Bruce, & Gatehouse, 1997) (Best, Howard, Bruce, & Gatehouse, 1997; Hickin, Best, Herbert, Howard, & Osborne, 2002).

Although these semantic and phonological cueing methodologies were originally developed to prime the stage of processing thought to be impaired, recent evidence indicates that both semantic and phonologically-based treatments elicit positive change, regardless of the hypothesized level of impairment (Hillis & Caramazza, 1994; Howard, 2000; Howard, Patterson, Franklin, Orchard-Lisle, &

²⁹ To further narrow the focus of the review of the anomia literature, treatment approaches given by non-professionals or paraprofessionals have been excluded from this review, as were computer-based treatments. Only treatment studies that reported reaction time, and/or response accuracy (or error type) during spoken language production tasks have been included in this review of anomia treatments.

Morton, 1985b; Nickels, 2002; Rochon, Leonard, Laird, Burianova, Soros, Graham, & Grady, 2006; Wambaugh, 2003; Wambaugh, Linebaugh, Doyle, Martinez, Kalinyak-Fliszar, & Spencer, 2001)³⁰. Consequently, many clinicians and clinical researchers have approached the treatment of lexical retrieval deficits by employing a *combination of semantic and phonologic cuing* (Cameron, Wambaugh, Wright, & Nessler, 2006; Linebaugh & Lehner, 1977; Martin, Fink, Laine, & Ayala, 2004; McNeil, Doyle, Spencer, Jackson Goda, Flores, & Small, 1998; Nettleton & Lesser, 1991; Wambaugh, 2003). To date, aphasiologists remain unclear as to why semantic and phonological cuing/priming paradigms both lead to improved naming. Abel and colleagues provide three possible accounts to explain this phenomenon: (1) the patient may have a mixed as opposed to pure deficit; (2) both types of cuing may inherently contain aspects of semantic and phonological processing; and (3) lexical retrieval may be inherently interactive, thus predicting that both semantic and phonological cuing methodologies will improve lexical retrieval as a result of the bidirectional flow of spreading activation between lemma and lexeme (Abel, Grande, Huber, Willmes, & Dell, 2005).

To better understand how these cuing paradigms influence lexical retrieval in an impaired system, the following sections will relate behavioral outcomes during anomia priming protocols with the behavioral correlates of priming for healthy participants. As discussed in the previous chapter, priming has been shown to be persistent, sensitive to the number of stimulus repetitions, and item-specific (i.e., priming is unlikely to generalize to untrained items) in healthy participants. Such comparisons may elucidate the characteristics of learning that are similar across healthy participants and individuals with aphasia to help develop a theory of learning for individuals with aphasia.

Is Priming Persistent for Individuals with Anomia?

While repetition priming has been shown to be very long lasting in healthy participants (i.e., up to 48 weeks), the literature examining individuals with anomia is

³⁰ Although participants often demonstrate greater and more stable gains with semantic cuing paradigms (Howard, Patterson, Franklin, Orchard-Lisle, & Morton, 1985a; Wambaugh, 2003). The issue of stability/maintenance will be discussed in the following section, with specific emphasis on repetition priming effects.

less consistent; that is, some investigators have found long lasting effects of priming (Rochon, Leonard, Laird et al., 2006; Wambaugh, 2003) while others have only found immediate priming effects for picture naming (Howard, Patterson, Franklin, Orchard-Lisle, & Morton, 1985a, , 1985b)³¹. The earliest repetition priming study with individuals with aphasia was conducted by Patterson and colleagues in 1983 (Patterson, Purell, & Morton, 1983). In this seminal study, 14 individuals with anomia underwent repetition priming and phonological priming protocols. Based on the naming performance of 265 black and white drawings, ten items were selected for the repetition priming experiment. Items that were named correctly within five seconds became fillers or untrained items. Those that were not named correctly were used as targets; five items were selected to be repeated and five were selected for naming purposes only. The subjects participated in three experimental sessions during which the subject either named the picture or was asked to repeat the name of the picture after the experimenter. The trials were designed so that 0, 10, 20, 30, or 40 items intervened between the repetition trials and the naming trials. The authors found strong immediate repetition priming effects, but did not find delayed priming effects.

Best and colleagues investigated what they consider immediate and delayed priming effects using both semantic and phonological primes (Best, Herbert, Hickin, Osborne, & Howard, 2002). Eleven individuals with anomia were asked to name 164 black and white line drawings of single syllable words. Those pictures that were named correctly within five seconds became filler items, while those that were named incorrectly were distributed into one of three experimental naming conditions: (1) extra time to name the picture; (2) a single prime, or (3) a choice of two primes where one of the primes is correctly associated with the target word. Primes were randomly selected from one of four types (1) whole word repetition, (2) a rhyme prime, (3) a phonological prime including the onset and nucleus, or (4) a written prime including the initial consonant and vowel of the target word. After the prime condition, the participant was asked to name the picture. The picture was then presented again after a ten minute delay. The number of intervening stimuli between the first and second presentation ranged from 36-134 items.

³¹ For a recent review, please refer to Best and colleagues (Best, Herbert, Hickin, Osborne, & Howard, 2002).

Best and colleagues found that all four types of primes resulted in significant immediate and delayed priming effects, with repetition primes eliciting the largest priming effects. Furthermore, the authors found that the long-lasting priming effects were greatest for participants who demonstrated impairments involving mapping from semantic to phonological processing. Best and colleagues did not provide concrete conclusions about why they found persistent priming effects, but hypothesized that the delayed priming resulted from longer exposure to the picture during the initial presentation; the picture remained in front of the participant during the period of time when the cue was given. This procedural difference is unlike traditional priming studies with healthy participants and the 1983 Patterson study. Although Best et al. found delayed priming effects (i.e., 10 minutes delay), they did not demonstrate that the individuals with anomia maintained these priming effects after the training protocol had ended; that is, the authors did not conduct a follow-up probe to assess long-lasting maintenance effects.

In summary, although priming effects have been conclusively demonstrated to be long-lasting in healthy participants, both short-lived and long-lasting priming effects have been found in individuals with aphasia. Further investigation of maintenance effects in the context of repetition priming protocols for individuals with aphasia is thus warranted.

Is Priming Sensitive to Stimulus Dosage for Individuals with Anomia?

To the best of my knowledge, the repetition priming experiment conducted by Patterson and colleagues remains the single repetition priming study using picture naming (as opposed to a paradigm like lexical retrieval that does not require overt production) that has explicitly compared repetition priming effects across multiple repetitions in individuals with anomia (Patterson, Purell, & Morton, 1983)³². That is, while all semantic and phonological priming studies necessarily require the participants to repeatedly name or repeatedly repeat the names of multiple pictures, investigators have not systematically manipulated or controlled for these repetition effects. Patterson and colleagues manipulated the number of repetitions (i.e.,

³² Martin and colleagues employed a repetition priming paradigm with many repetitions; however, the authors did not report results on incremental priming effects for each successive repetition (Martin, Fink, Laine, & Ayala, 2004).

repeated 1-5 times) and found that participants were no more likely to name a target correctly after five repetitions than they were with only one repetition of the item. This lack of sensitivity to the number of repetitions may stem from the lack of relevance to the participant; simply repeating after a clinician may not make the task meaningful enough for the participant. Furthermore, response accuracy may not be a sensitive enough measure for priming protocols. A repeated theme discussed by many authors presenting at the recent Academy of Aphasia conference (October 14-17, 2006), was that for those patients who are at or near ceiling on picture-naming tasks in regards to accuracy, reaction time measures can be used to reveal continuing improvements. As such, a systematic reaction time investigation of picture naming with incremental repetitions is warranted.

Is Priming Item-Specific for Individuals with Anomia?

The question asked here is whether or not trained targets generalize to untrained items during picture-naming priming protocols. Repetition priming experiments examining healthy control participants have indicated that repetition priming is item-specific; that is, priming effects do not generalize to untrained items. For clinical aphasiologists, generalization is potentially one of the most frustrating components of anomia treatment. Although clinicians and aphasiologists regularly document significant treatment effects in regards to acquisition and quite often for maintenance as well, very few find that their approach resulted in coinciding improvements of untrained items. For example, despite successfully training participants to name six sets of word lists composed of multiple classes of single words (e.g., nouns, verbs, adjectives, and preposition), little generalization to untrained items, regardless of word class, was found (McNeil, Doyle, Spencer et al., 1998)³³.

The study discussed relative to repetition priming (Best, Herbert, Hickin, Osborne, & Howard, 2002; Patterson, Purell, & Morton, 1983; Wambaugh, 2003) did not report data for untrained items. In an anomia treatment protocol (i.e., not a

³³ A small generalization effect was found for one of six sets of stimuli in the antonym condition. The authors were unable to provide an explanation for this occurrence given that the stimuli were randomly divided across sets. The authors dismiss the possibility of an accumulation effect as coinciding generalization effects were not observed for the synonyms condition.

traditional psycholinguistic repetition priming protocol), Howard found a small (i.e., not statistically significant) generalization effect from untrained to trained items, with slightly more generalization observed for semantic cuing than phonological cueing (Howard, Patterson, Franklin, Orchard-Lisle, & Morton, 1985b). Using a semantic-based anomic treatment protocol, Kiran and colleagues have also found significant generalization from trained to untrained items when the trained targets include atypical members of a category; that is, training “emu” in the category of birds is more likely to result in generalization to untrained items than training “robin” is (Kiran & Thompson, 2003). Additionally, Nadeau and Kendall found generalization effects for three out ten participants after a semantic treatment similar to the Semantic Features Analysis protocol developed by Boyle and colleagues (Nadeau & Kendall, 2006). In a single-subject multiple baseline study of four individuals with severe anomia (resulting from various levels of impairment), Raymer and colleagues (1993) administered a phonological treatment protocol during which the participants attempted to name pictures (see description provided in table 5). Results based on response accuracy demonstrate significant acquisition and maintenance effects during training and at two months post-treatment. Even more noteworthy was the consistent generalization from trained to untrained items for all four subjects.

As such, the results are mixed and investigators have yet to systematically study the myriad subject and treatment variables that may influence the potential for generalization. More importantly, the traditional psycholinguistic repetition priming paradigm has not been systematically and incrementally employed for individuals with anomia; therefore, details about the effects of repetition priming, including generalization to untrained items, are absent from the anomia literature base. A significant variable that has not been studied in any of these treatment protocols is the number of times the patient named the target (i.e., the number of repetitions). It is possible that the variability in regards to acquisition, maintenance and generalization reflects the amount of practice, regardless of the underlying impairment³⁴. However, given that repetition priming is item-specific for healthy

³⁴ For example, both Raymer and colleagues (Raymer, Thompson, Jacobs, & Le Grand, 1993) and Martin and colleagues (Martin, Fink, Laine, & Ayala, 2004) require the participant to repeat the target more times than most treatments. In fact, Martin et al. suggest that

control participants, generalization from trained to untrained items is not expected in anomia protocols that simply employ a repetition priming paradigm³⁵.

Is Repetition Priming Reflected as Neural Repetition Suppression for Individuals with Anomia?

In healthy participants, repetition priming is reflected at the neural level primarily as repetition suppression. That is, relative to the initial picture-naming trial, neuroimaging results have revealed decreased neural activity in healthy participants after repetition. To date, no aphasiologists have investigated the effect of repetition priming at the neural level for individuals with anomia. Recently, a few studies have begun to investigate changes in the hemodynamic response via fMRI as a result of intensive treatment protocols (for a recent MEG study, see Meinzer, Elbert, Wienbruch, Djundja, Barthel, & Rockstroh, 2004). One can infer that repetition has taken place in these treatment protocols; however, it is impossible to tease apart the treatment effects to specifically analyze repetition suppression or enhancement. The continuously expanding body of literature examining the laterality of aphasia recovery often finds activation in some cortical regions and deactivation in others; however, these changes in hemodynamic response have been correlated only to language function, not repetition priming.

A comparison of repetition priming effects across anomia studies remains nearly impossible as a result of the heterogeneity of subject and training variables. For example, although the studies conducted by Patterson and colleagues (Patterson, Purell, & Morton, 1983) and Best and colleagues (Best, Herbert, Hickin, Osborne, & Howard, 2002) more closely adhere to traditional psycholinguistic priming paradigms, those conducted by Wambaugh and colleagues and Rochon and colleagues more closely adhere to traditional anomia treatment protocols. The studies reported here have investigated the underlying impairment, or the effect of a

repetition priming was responsible for the large immediate (and unexpected) facilitation effects that they documented.

³⁵ However, in a repetition priming protocol used to improve oral reading for an individual with phonological text alexia, Sperling and colleagues found generalization from trained to untrained sets of sentences (Sperling, Lott, Watson, & Friedman, 2006). It is noteworthy that successful acquisition and generalization were observed, given that the subjects only repeated the sentences once.

type of prime on naming, but few have systematically addressed priming as a learning paradigm. To date, no repetition priming studies have been conducted that reflect traditional psycholinguistic repetition priming paradigms that (i.e., healthy controls typically name the picture once, and then name it again at some later time); instead, individuals with aphasia are cued to repeat the word after clinician and then attempt to name the picture. That is, few studies have attempted to systematically describe the nature of priming effects for individuals with aphasia. Furthermore, most of the studies discussed here employed a variety of primes, in addition to repetition, but none of them have examined the cumulative effects of multiple prime types.

Nearly all anomia treatment protocols used in research and by clinicians require the individual to name pictures repeatedly. As such, a systematic investigation of how repetition alone (i.e., in the absence of other components of traditional treatment protocols) influences the acquisition, maintenance and generalization of trained to untrained target stimuli can provide information about the nature and persistence of repetition priming in an impaired linguistic system³⁶. The following variables are likely to influence both the acquisition and the maintenance of repetition priming effects and should be considered in future repetition priming paradigms:

- total number of target items (i.e., set size);
- the number of intervening stimuli between repeated presentations³⁷;
- the duration between the repeated presentations;

³⁶Repetition priming is likely to differentially influence lexical retrieval based upon the linguistic impairment (i.e., semantic vs. phonological encoding). Ferrand and colleagues have suggested that repetition priming is more attributable to phonological encoding as opposed to semantic encoding (Ferrand, Grainger, & Segui, 1994). To the contrary, Wheeldon and Monsell found evidence to indicate that repetition priming stems from the connection between semantic and phonological encoding (Wheeldon & Monsell, 1992). Based upon these two studies it is likely that repetition priming is more likely to influence deficits of phonological encoding or some processes immediately preceding phonological encoding.

³⁷ Although see Blumstein et al. for a repetition priming study in the context of lexical decision tasks (Blumstein, Milberg, Brown et al., 2000). The authors found that individuals with Broca's and Wernicke's aphasia demonstrated significant repetition priming effects for words (as opposed to nonwords) only for the 0 lag condition (i.e., no intervening stimuli).

- the type of repetition (i.e., anomia studies require the participant to repeat after the clinician; traditional repetition priming studies simply require the participant to rename the picture at a later time); and
- the type of outcome measure (i.e., response accuracy vs. reaction time).

ANOMIA TREATMENT DOSAGE AND INTENSITY

In a recent review of anomia management, Maher & Raymer state that 30% of aphasia intervention research from 1946 to 2001 focused on naming (the most of any type of aphasia intervention); however, “despite this proliferation of case reports and small group studies, there is still no clear agreement on how best to manage these deficits” (Maher & Raymer, 2004, p. 13). As was mentioned in earlier chapters, the inconsistency of acquisition, maintenance, and generalization effects observed across participants and types of treatment protocols is likely to stem from an inadequate knowledge base about how subject and treatment variables influence (re)learning. One treatment variable that has received intermittent attention is *dosage* or *treatment intensity* (Basso, 2005; Basso, Capitani, & Vignolo, 1979; Bhogal, Teasell, & Speechley, 2003; Bhogal, Teasell, Foley, & Speechley, 2003; Brindley, Copeland, Demain, & Martyn, 1989; de Pedro-Cuesta, Widen-Holmqvist, & Bach-y-Rita, 1992; Denes, Perazzolo, Piani, & Piccione, 1996; Hinckley & Craig, 1998; Pulvermuller, Neininger, Elbert et al., 2001; Robey, 1998). Specifically, Bhogal, Teasell & Speechley (2003) suggest that intensity of treatment is likely to emerge as the variable that contributes most to the inconsistency of acquisition, maintenance and generalization effects across studies. As such, this section will further explore the learning affects stemming from treatment variables including treatment intensity (i.e., the number of sessions per week and number of total sessions), and stimulus dosage (i.e., the number of repetitions of individual lexical items).

Treatment Intensity

In the motor rehabilitation literature, treatment intensity broadly refers to the amount of time that is dedicated to practice (Kwakkel, 2006). More specifically, treatment intensity or duration is defined as the length of treatment for one session

or the total amount of time dedicated to treatment across sessions. This concept of duration includes aspects of intensity including the number of minutes or hours per session, the number of sessions per day or week, and the number of weeks or months of total treatment.

For aphasia rehabilitation, participants who receive a greater number of treatment sessions improve to a larger degree than those who receive fewer treatment sessions (Basso, 2005; Bhogal, Teasell, & Speechley, 2003; Bhogal, Teasell, Foley, & Speechley, 2003; Robey, 1998). Specifically, Robey's (1998) meta-analysis indicated that treatment should include a minimum of two hours of training per week, with five or more hours per week resulting in the greatest degree of change. More recently, in a review of the aphasia literature, Bhogal and colleagues demonstrated that treatment studies that elicited improved linguistic performance required participants to engage in therapy for an average of eight hours per week, while those that did not result in improved linguistic performance required participants to engage in an average of two hours per week. Finally, both Pulvermuller and colleagues (2001), and Meinzer et al. (2005) demonstrated that three to four hours per day of treatment for ten consecutive days resulted in significant and stable linguistic improvement for individuals with chronic aphasia. For a summary of the aphasia literature that has directly or indirectly provided evidence about treatment intensity³⁸, please refer to table 1.3.

Table 1.3 Summary of Intensity Literature for General Aphasia Rehabilitation

Reference	Number of Aphasic Subjects	Conclusions about Intensity
(Basso, Capitani, & Vignolo, 1979)	162	No less than 3 individual sessions/wk for no less than 6 months
(Bhogal, Teasell, & Speechley, 2003; Bhogal, Teasell, Foley, & Speechley, 2003)	10 studies reviewed (n=864 across studies)	Positive intervention results were found in shorter treatment protocols with greater intensity (more hours/week)
(Brindley, Copeland, Demain, & Martyn, 1989)	10	Improvement of speech and syntax w/intensive tx (25 hours per week for 12 weeks)
(David, Enderby, & Bainton, 1982)	96	2 hours/wk shows improvement

³⁸ Please note: the studies reported in table 1.3 investigated or reported on intensity for general aphasia treatment, not lexical retrieval in particular. Review articles examining intensity have also been included.

Reference	Number of Aphasic Subjects	Conclusions about Intensity
(Denes, Perazzolo, Piani, & Piccione, 1996)	17	Intensive tx is better than regular tx in global aphasics (5 sessions/wk vs. 2.5 sessions/wk)
(de Pedro-Cuesta, Widen-Holmqvist, & Bach-y-Rita, 1992)	20 studies reviewed	2hrs/wk (of any tx or counseling) not effective; 8-10 hrs/wk of tx show sig improvements
(Hartman & Landau, 1987)	60	Cannot comment on efficacy of intensive tx
(Hinckley & Craig, 1998)	40 across three studies	No experimental control - unable to draw conclusions - authors conclude that intensive tx is better than no tx or little tx
(Lincoln, McGuirk, Mulley, Lendrem, Jones, & Mitchell, 1984)	327	Cannot comment on efficacy of intensive tx; 2hrs/wk does not show efficacy
(Marshall, Wertz, Weiss, Aten, Brookshire, Garcia-Bunuel, Holland, Kurtzke, LaPointe, & Milianti, 1989)	121	8-10 hrs of tx shows improvement; unable to compare to less intense tx
(Pulvermuller, Neining, Elbert et al., 2001)	17	Intensive treatment (3-4 hours per day for 10 consecutive days) can lead to improvement in chronic aphasics
(Robey, 1998)	55 studies reviewed	The more intense the tx, the greater the change; Tx length in excess of 2 hrs/wk brings about gains exceeding those that result from shorter durations. Two hrs/wk should constitute a minimum length for patients who can withstand the rigors of receiving treatment (pp. 184).
(Shewan & Kertesz, 1984)	100	3hrs/wk shows sig gains; no comparison to less intense txs
(Wertz, Weiss, Aten, Brookshire, Garcia-Bunuel, Holland, Kurtzke, LaPointe, Milianti, Brannegan, & et al., 1986)	121	8-10 hrs of tx shows improvement; unable to compare to less intense tx

Stimulus Dosage

Principles of neurobiological learning across both animal (Squire, 1992) and human research (Poldrack & Gabrieli, 2001) suggest that the intensity of treatment is a significant factor for learning; further research addressing neural plasticity involved in memory and learning indicates that a large number of trials are required to elicit change (Squire, 1992). The number of trials per session refers to the concepts of treatment frequency or stimulus dosage (Kwakkel, 2006). Although a renewed interest in treatment intensity has emerged in aphasia research, and a considerable number of researchers are currently exploring the effects of constraint-induced aphasia treatment (CIAT), no studies have systematically investigated the incremental effects of treatment intensity separate from other subject and/or treatment variables and no studies to date have manipulated stimulus dosage to determine a dose-response curve in individuals with aphasia.

Specifically, the number of repetitions of a given stimulus required to yield consistent improvement of naming accuracy and latency has not been investigated, despite the fact that repeated verbal practice of picture-naming is inherent to nearly all anomia treatment protocols. Lexical retrieval studies typically report the characteristics of their participants, details of the treatment approach, and the overall intensity (i.e., duration) the protocol. Rarely do investigators provide the exact number of times the picture was presented to the participant³⁹. For example, using a treatment protocol that purported to use principles of massed practice, spaced retrieval, and errorless learning, Fridriksson and colleagues attempted to train three words per day for three individuals with anomia (Fridriksson, Morrow-Odom, Moser, Fridriksson, & Baylis, 2006). Although the authors provide a detailed description of the hierarchy of training procedures they used, the criteria for the participant to move on to the next level in the hierarchy of cuing was based upon three consecutive errorless productions of the target picture. The authors do not report the details for each participant in regards to how many times they required each level of cuing before being able to name the picture three times in a row. As such, the exact number of times that the participant attempted production or actually produced the target's name correctly cannot be determined. For a review of anomia treatment protocols that have purported to be intensive, please refer to table 1.4.

³⁹ The only exception is a repetition priming study conducted by Martin and colleagues in which an exact number of repetitions was provided (Martin, Fink, Laine, & Ayala, 2004). However, the authors were not able to report on the isolated effects of repetition priming as the protocol also employed contextual priming. Additionally, the authors did not provide data about the incremental effects of each repetition on naming.

Table 1.4 Summary of Stimulus Dosage in Anomia Studies

Reference	Number of Aphasic Subjects	Treatment Description	Type and Number of Stimuli	Number of Repetitions	Duration of Each Session	Total Length of Treatment
(Fridriksson, Morrow-Odom, Moser, Fridriksson, & Baylis, 2006)	3	Spaced retrieval, massed practice, errorless learning	15 nouns selected by patient	3 items/day (at least 27 repetitions per day) – no way of determining how many attempts were made	4 hr/day	2 weeks
(Breitenstein, Kamping, Jansen, Schomacher, & Knecht, 2004)	2	Implicit associative learning	50 drawings and 50 pseudo-words	Correct pairings: 20/session; incorrect: 2/session	Not reported	1 day
(Martin, Fink, Laine, & Ayala, 2004)	11	Contextual priming: semantic, phonological, or unrelated primes	10 pictures	At least 32 repetitions at least per session	Not reported	3 days
(Meinzer, Elbert, Wienbruch et al., 2004)	28	CIAT or model-based	Not reported	Not reported	3 hrs/day	10 consecutive days
(Cornelissen, Laine, Tarkiainen et al., 2003)	3	Contextual priming technique	50 trained black and white drawings	5 repetitions of each picture	1 hr/session	3 times/week for approx. 3 weeks (until 70% correct)
(Pulvermuller, Neininger, Elbert et al., 2001)	17	CIAT vs. "conventional treatment"	16 pictures	Not reported	CIAT: 3-4 hours/day	Conventional: 3-5 weeks (20-54 hours) CIAT: 10 days (23-33 hours)
(Patterson, Purell, & Morton, 1983)	14	Repetition Priming	10 pictures that participants had difficulty naming	1 vs. 5 repetitions	Not reported	One day

As additional motivation for understanding the relationship between repetition and naming performance, a brief examination of those who are investigating CIAT (e.g., Meinzer, Elbert, Wienbruch et al., 2004; e.g., Pulvermuller, Neininger, Elbert et al., 2001) is warranted. These researchers have adapted their protocol from the motor learning literature (Kunkel, Kopp, Muller et al., 1999), which

necessarily requires intensive or massed practice (i.e., repetition) as one of its three primary components of the therapy protocol⁴⁰. Although studies that have investigated the effects of CIAT have shown promising and significant effects on the acquisition and maintenance of naming performance for individuals with chronic aphasia, the three components of CIAT (i.e., intensive practice, constraint, and shaping) have yet to be investigated in isolation, and more importantly, aphasiologists do not have data that illustrates the influence of repetition (i.e., one component of intensive practice) on naming performance. Without incremental repetition priming data, investigators will be unable to parse the accumulative effects of treatment type, treatment variables, and overall treatment intensity.

⁴⁰ CIAT is based upon three fundamental principles of learning: (1) intensive practice; (2) shaping, and (3) constraint of the unimpaired function to forced use of the impaired function (Meinzer, Elbert, Wienbruch et al., 2004; Pulvermuller, Neiningger, Elbert et al., 2001).

CHAPTER II: GOALS AND RESEARCH QUESTIONS

Picture-naming tasks are inherent to nearly all anomia treatments and closely approximate the linguistic processes used during spontaneous language production (Indefrey & Levelt, 2004). Pictures are named, or attempts are made to name them, multiple times within and/or across sessions. Although anomia treatments have been demonstrated to be largely effective regardless of the targeted level of impairment, many treatment variables including stimulus dosage have not been systematically manipulated to document their influence on the acquisition and maintenance of trained items or generalization to untrained items (response generalization) or alternate exemplars (stimulus generalization). Despite the considerable amount of literature examining the overall intensity of (i.e., the total length of treatment in hours, days, or weeks), data are not available regarding the duration (i.e., the number of minutes or hours per session) or frequency (i.e., the number of repetitions of each stimulus item per session) of treatment at which stroke survivors will maximally benefit. Thus, systematic dosage manipulations are necessary to provide evidence for optimal intervention rates for patients with anomia. Additionally, although repeated picture-naming is built into most anomia treatments, and repetition priming as a learning process is likely to provide a foundation for any other type of impairment-based lexical retrieval treatment procedure applied during the course of stroke recovery, a systematic investigation of stimulus dosage has been unexamined in individuals with anomia.

Finally, repetition is also likely to interact with lexical variables associated with the items that are being trained. For example, the word frequency effect (WFE) has been shown to be susceptible to repetition for healthy participants⁴¹. That is, the relative magnitude of the WFE (i.e., the difference between response times for high frequency (HF) and low frequency (LF) words) has been shown to change over the course of multiple repetitions. The most frequently observed phenomenon in healthy adults is what is known as a frequency attenuation effect during which LF

⁴¹ Repetition effects have been proposed to arise during retrieval of the phonological word form (La Heij, Puerta-Melguizo, van Oostrum, & Starreveld, 1999), as are effects of word frequency (Balota, 1984; Hino & Lupker, 1996; Humphreys, 1988; Huttenlocher & Kubicek, 1983; Jescheniak & Levelt, 1994). Data also suggests that the word frequency effect arises during phonological encoding. Not surprisingly, repetition priming has been observed to interact with word frequency (La Heij, Puerta-Melguizo, van Oostrum, & Starreveld, 1999).

words benefit to a greater extent than do HF words (Scarborough, Cortese, & Scarborough, 1977; Versace & Nevers, 2003)⁴². As such, repetition priming may allow infrequently used words to be processed more like frequently used words (La Heij, Puerta-Melguizo, van Oostrum, & Starreveld, 1999; Whiteside & Varley, 1998). Assuming that the WFE does in fact interact with repetition for healthy participants, individuals with aphasia may demonstrate differential learning (i.e., repetition priming) effects across lexical items.

Repetition priming is a learning paradigm that can be used to investigate behavioral changes associated with manipulations of stimulus dosage during picture-naming for individuals with anomia. Dosage and intensity manipulations can be made easily, detailing the stimulus set size, the number of presentations, the duration between stimulus presentations, the duration between training sessions, and the overall duration of treatment. Understanding repetition priming effects in isolation of other training variables (i.e., treatment intensity, type of treatment, etc.) is important if we want to examine multiple conditions using the same target stimuli. Furthermore, because the repetition priming paradigm is relatively implicit in nature and requires no controlled attentional processes, it is an ideal tool by which to incrementally investigate acquisition, generalization, and maintenance of trained items during spoken language production.

The following two experiments were designed to document the behavioral effects of repetition priming on naming performance (response accuracy and response time) among individuals with aphasia who have lexical retrieval impairments (i.e., anomia). The first experiment is a pilot study developed to assess inclusionary criteria, protocol procedures, and feasibility of this type of protocol with individuals with aphasia. The second experiment applies these refined procedures to document the influence of repetition priming on picture naming for individuals with aphasia. Manipulation of various independent variables have been made in order to compare the influence of repetition priming on learning in individuals with aphasia with those processes associated with repetition priming in healthy non-brain injured participants.

⁴² Although, Forster and Davis found equal effects of repetition on LF and HF words when the prime was masked (Forster & Davis, 1984).

Please refer to table 2.1 for a summary of the specific questions pertaining to repetition priming that are addressed, hypotheses stemming from these questions, and the independent variables that have been selected to be manipulated to explore these questions.

Table 2.1 Experimental Questions and Independent Variables

Experimental Question	Independent Variables Manipulated
<p><i>Is repetition priming persistent across time for individuals with aphasia?</i></p> <p>H0: Repetition priming is not persistent across time for individuals with aphasia. During the acquisition phase, decreased response time and increased response accuracy will be observed for immediate but not delayed probes. Furthermore, decreased response time and/or increased response accuracy observed during the acquisition phase of the protocol will not be observed after training has been terminated.</p> <p>H1: Repetition priming is persistent across time for individuals with aphasia. During the acquisition phase, decreased response time and increased response accuracy will be observed across immediate and delayed probes. Furthermore, decreased response time and increased response accuracy will be observed during all maintenance probe sessions.</p>	<p>Immediate vs. Delayed Probes</p> <p>Training Phase vs. Maintenance Phase</p>
<p><i>Is repetition priming sensitive to the number of trials/session?</i></p> <p>H0: Stimulus dosage will not influence response time or response accuracy. That is, no difference in response time or response accuracy for 1 vs. 4 trials per session will be observed during acquisition or maintenance phases.</p> <p>H1: Larger number of trials per session (i.e., 4 trials per session vs. 1 trial per session) will result in larger decreases in response time and increases in response accuracy across both acquisition and maintenance phases.</p>	<p>1 vs. 4 Trials per Session</p>
<p><i>Is repetition priming item specific?</i></p> <p>H0: Repetition priming is item specific. That is, trained, but not untrained items will be responded to more quickly and more accurately as a result of the repetition priming</p>	<p>Trained vs. Untrained Items</p> <p>Alternate Exemplars</p>

Experimental Question	Independent Variables Manipulated
<p>protocol. Alternate exemplars will not demonstrate decreased response time or increased accuracy as a result of the repetition priming protocol.</p> <p>H1: Repetition priming is item specific; however, alternate exemplars will be responded to more quickly and more accurately as a result of the repetition priming protocol.</p>	

CHAPTER III: PILOT STUDY

The pilot study was designed to explore the feasibility of a picture-naming repetition priming protocol with individuals with aphasia. Specifically, this pilot investigation sought to initially explore the influence of repetition on picture-naming in individuals with aphasia. Secondary goals included the refinement of inclusionary criteria, technical procedures and stimuli. A brief summary of the pilot study will be provided below. For more detailed information about this study, please refer to the Master's theses written by Abigail Potts (Potts, 2006) and Ann Kenny (Kenny, 2006).

RESEARCH DESIGN AND METHODS

A single-subject A-B design was used to investigate the influence of repetition priming on the acquisition, maintenance and generalization of lexical retrieval for individuals with aphasia. Dependent measures included response accuracy and response time; independent variables included word frequency and syllable length.

Participants

One individual with aphasia and two non-brain injured healthy controls participated in the pilot study. Individuals with chronic aphasia (i.e., greater than six months post CVA) were recruited to participate in this study. For inclusion into this study, individuals with aphasia had normal to corrected hearing and vision, were pre-morbidly right-handed, did not demonstrate visual agnosia, and met a variety of specific language modality criteria as determined by an extensive cognitive-linguistic battery (refer to table 3.1 for a summary of language modality-specific criteria). Participants with abnormal structural-functional abilities, severe dysarthria, severe dementia, and/or a prior history of speech, language and/or neurological deficits were excluded from this study. Two measures administered during the cognitive-linguistic battery were re-administered following completion of the training protocol including the portions of the Western Aphasia Battery (WAB) required to compute

the Aphasia Quotient (AQ) and the Picture Naming by Frequency subtest of the Psycholinguistic Assessment of Language Processes in Aphasia (PALPA).

Table 3.1 Pilot Study Inclusionary Criteria/Cognitive-Linguistic Battery

Test	Description/Purpose	Inclusionary/Exclusionary criteria
Vision Screening	Snellen chart.	Pass = 20/30 at 2.3 feet with or without glasses/contacts Fail = referral to optometrist
Hearing Screening	Portable audiometer. Tested best ear at 500, 1000, 2000, & 4000 Hz.	Pass = 35 dB Fail = referral to audiologist
Edinburgh Handedness Test <i>Oldfield (1971)</i>	To determine hand dominance for future fMRI studies.	Right-hand dominance
Beck Depression Inventory (BDI-II) <i>Beck (1978)</i>	To rule out significant depression that may influence response time or participation in the protocol.	Pass = 0-20
Structural/Functional Exam	To examine oral structures and their functions.	Descriptive only
Informal Assessment of Visual Agnosia	To rule out visual/perceptual deficits.	Pass = raw score > 8/10 Fail = excluded from study
Western Aphasia Battery (WAB) <i>Kertesz (1982)</i>	To assess language across modalities. Administered only those subtests needed to calculate Aphasia Quotient(AQ)	Pass = AQ 25-75/100; fluency 4-8 Fail = excluded from study
Boston Naming Test (BNT) <i>Goodglass & Kaplan (1983)</i>	To assess word-finding abilities.	Descriptive only Raw score = ___/60
Raven's Coloured Progressive Matrices <i>Raven (1976)</i>	To assess non-verbal problem solving (non-linguistic cognitive abilities).	Pass = >12/36 Fail = excluded from study
Apraxia Battery for Adults (ABA) <i>Dabul (1979)</i>	To assess motor planning/programming.	Pass = no scores in "Severe to Profound" range on "Profile Score Sheet". No more than 3 items on "Checklist of Apraxic Features" Fail = excluded from study
Subtests 1-3 of the Reading Comprehension Battery for Aphasia (RCBA) <i>LaPoint & Homer (1979)</i>	To assess single word reading ability.	Descriptive only Raw score = ___/30
Pyramids and Palm Trees Test <i>Howard & Patterson (1992)</i>	A test of semantic access. To assess the participant's ability to access detailed semantic representations from words and pictures.	Descriptive only Raw score = ___/52
Subtests of the Psycholinguistic Assessment of Language Processes in Aphasia (PALPA) <i>Kay, Lesser, & Coltheart (1992)</i>	To assess confrontational picture naming with high and low frequency stimuli; written synonym judgment; rhyme judgment.	Pass = 10-40/60 Naming Pass = >36/60 Synonym Judgment Pass = >5/16 Rhyme Judgment
Revised Token Test (RTT) <i>McNeil & Prescott (1978)</i>	To assess auditory comprehension with increasing length and complexity.	Pass = no worse than moderate auditory comprehension

For inclusion into this study, healthy non-brain injured control participants were required to pass a vision screening with or without glasses and/or contacts; demonstrate a score of 34/60 on the Picture Naming by Frequency subtest of the PALPA or 50/60 on the Boston Naming Test; achieve a raw score greater than 23 on the Raven's Coloured Progressive Matrices; score within the normal range for auditory comprehension on the Revised Token Test; and be right-handed as demonstrated by positive values for right handedness on the Edinburgh Handedness Inventory. Control participants were excluded from the study if they had a history of current or past speech, language, hearing or neurological impairments, or were not native speakers of American English. Refer to table 3.2 for a summary of the participants' profiles.

Table 3.2 Pilot Study Participant Profiles

	Individual with Aphasia		Control 1	Control 2
Age	80		74	50
Gender	Female		Male	Female
Cognitive-Linguistic Battery	Pre	Post		
WAB AQ	29.7	33.1	DNT	DNT
PALPA (Naming)	23/60	30/60	DNT	59/60
BNT	DNT		54/60	DNT
PALPA (Synonym Judgment)	10/60		DNT	DNT
PALPA (Rhyme Judgment)	44/60		DNT	DNT
RTT	11.5/36		30/36	33/36
ABA	Minimal Verbal Apraxia; Moderate Limb Apraxia; Mild-Moderate Oral Apraxia		DNT	DNT
RCBA	24/30		DNT	DNT
Visual Agnosia	9/10		DNT	DNT
Raven's	24/36		34/36	36/36
BDI-II	4/60		DNT	DNT
Edinburgh Handedness Inventory	Left Hand: 0/10 Right Hand: 10/10		Left Hand: 2/10 Right Hand: 10/10	Left Hand: 1/10 Right Hand: 9/10

DNT = Did Not Test

Procedures

Stimuli

Stimulus items were drawn from a previously collected corpus of 220 picturable, concrete nouns depicted by colored photographs. Half of these words were high frequency (i.e., greater than 100 instances per million) and half were low frequency (i.e., less than seven instances/million) based upon frequency counts made by Francis & Kucera (Francis & Kucera, 1982). These stimulus items were also evenly balanced for one- and two-syllables.

Of these 220 words, 40 items were selected as trained items and 180 were selected as untrained items. Audio files of the verbally-produced names for all of these items were previously recorded with wavelength durations between 400 and 600 ms.

Protocol – Individual with Aphasia

Following enrollment, the individual with aphasia was administered five baseline probes to assess pre-training picture-naming performance. Each baseline probe session consisted of the complete set of all 40 trained items and 20 untrained items. The participant was exposed to the picture and the verbally-produced name of the picture prior to each baseline probe session. During the probe session, the participant was asked to name the picture.

Following completion of the baseline phase, the participant began the training sessions. Training sessions consisted of the participant seeing a picture, hearing its name through the speakers, and then naming the picture. This sequence occurred twice for each of the 40 trained items during each training session. Training probes were administered after every two training sessions. Training probes consisted of the 40 trained items and 20 novel/untrained items. Participant A002 completed 16 training sessions, with each session lasting approximately one hour.

Following completion of the training phase, the participant returned to complete three maintenance probes. The target stimuli lists from baseline sessions 1-3 were repeated during the three maintenance probes.

Protocol – Healthy Control Participants

Similar to the individuals with aphasia, two healthy control participants completed five baseline probe sessions and four training probe sessions (after every two training sessions). Three maintenance probes were completed after the last training session was completed.

The first control participant completed an “extended” 12 week protocol. Baseline and probe sessions were held twice per week over an eight week period. Maintenance probes were administered one week, two weeks, and one month after completion of the last training session. The second control participant completed a “compressed” two-week protocol. Baseline line probes 1-2 were administered on one day. Baseline probes 3-5 and training probes 1-4. The two sessions were spaced one week apart. This participant did not complete training sessions or the maintenance probes.

SUMMARY OF RESULTS

Average response accuracy and response time were calculated for baseline, training, and maintenance phases of the protocol. Response accuracy increased for both trained and untrained items from baseline to training phases, with trained items (54% increase) increasing more than untrained items (32% increase). From training to maintenance, trained items increased an additional 3% while untrained items decreased in response accuracy 16%. No increase in response accuracy was observed during the baseline phase, despite the fact that each baseline probe included the 40 trained items. Response time also increased (i.e., participants named items more slowly) during the training phase for both trained (72 ms) and untrained (94 ms) items. Response times returned to baseline levels during the maintenance phase. Collectively, these data suggest a response time for response accuracy trade-off. That is, as participants became more accurate during the training phases of the protocol their response times increased. However, once these items had been acquired (as demonstrated by a persistent increase in response accuracy during the maintenance phase), response times returned to baseline levels during the maintenance phase.

The effects of word frequency as they interacted with repetition priming were also assessed in this pilot study. During baseline, high frequency words were responded to 20 ms faster than low frequency words although high frequency words were responded to less accurately than low frequency words. No systematic effects of word frequency on response time were observed during the training; however, high frequency words were more accurately responded to during training than low frequency words. Although no systematic effects of word frequency on response time were observed during the maintenance phase of the investigation, high frequency words were responded to more accurately during the maintenance phase than low frequency words. These initial findings suggest that more practice may be required for low frequency words in order to achieve long lasting improvement. That is, maintenance appears to be superior for high frequency words. No systematic effects of word frequency were observed in regards to generalization of trained to untrained words.

Results from the healthy control pilot data indicated that trained items were responded to faster than untrained items by the final probe session (approximately 109-279 ms decrease). Session by session analysis revealed that repetition priming effects on reaction time occurred during the first three probe sessions. A plateau was observed from that point on. The small amount of data stemming from only two participants did not provide clear patterns for word frequency or syllable length as they related to repetition priming influences on reaction time. However, a non-significant trend was observed for low frequency words being responded to faster with additional repetitions relative to high frequency words.

This pilot study demonstrated that a repetition priming experiment is feasible for individuals with aphasia. Individuals with aphasia are able to tolerate multiple lengthy sessions per week over the course of several weeks. These initial results indicate that repetition priming does occur in individuals with aphasia; however, it appears that individuals with aphasia require significantly more repetitions than healthy control participants to demonstrate priming effects that are present after only one exposure in healthy adults. As a result of these findings, several modifications were made prior to initiating the dissertation project.

MODIFICATIONS STEMMING FROM PILOT DATA

As a result of some difficulties encountered during the pilot study, several technical adjustments were made to instrumentation and stimulus delivery: (1) the throat microphone used during the pilot study was found to provide inconsistent response times for the individual participant. For this reason, a head-mounted microphone was used for the dissertation project; and (2) ambient noise within and outside of the lab created auditory distractions. For this reason, high quality sound-canceling headphones were used in the dissertation project to deliver the auditory stimuli.

The lack of word-frequency related effects on picture naming in the pilot study was worrisome. For this reason, the corpus of pictures was reassessed for name agreement and to pull out words that neared the cutoff for the word frequency count. New pictures were developed for items that appeared to result in naming agreement confusion. Several targets for which reliable names could not be assigned were thrown out.

The inclusionary criteria for enrollment into the study was also revised. In general, inclusionary criteria were made more permissible to increase enrollment by making many of the tests descriptive in nature rather than have an inclusionary criteria cut-off. The Edinburgh Handedness Inventory was dropped as no imaging studies were planned for the future. The Revised Token Test was also dropped as the WAB sections assessing comprehension provided enough information about the participants' comprehension abilities in regards to participating in the study. The Boston Naming Test and Pyramids and Palm Trees test were added to the protocol as a pre/post measure of naming performance in order to be able to compare our results with those of others publishing in the field. Two of the subtests of the PALPA were dropped as they did not provide information that proved to be useful for inclusion into the study. Finally, a Trial Run Probe was added to the inclusionary criteria to ensure that participants could successfully participate in the computer-based naming protocol. During the pilot study some participants who met all our other criteria were not able to adapt to the computer-based task.

The pilot study also indicated that the individual with aphasia required significantly more repetitions before priming effects were observed, despite the fact

that she had five baseline probes during which the 40 trained items were named. For this reason, an additional manipulation was constructed for the dissertation project: the number of trials per session (1 vs. 4) was added as an independent variable. As a result of this added manipulation, the delivery of the stimuli was also modified. During the pilot study stimuli were pseudo-randomized; that is, attention was paid to minimize successive words with similar initial consonants or similar semantic categories. With the added variable of number of trials pseudo-randomization was no longer possible. As such, stimuli in the dissertation project were delivered randomly with no control over initial consonant, semantic category, or number of trials intervening between repetitions.

CHAPTER IV: METHODOLOGY

RESERCH DESIGN

A single subject A-B design with replication across four participants with chronic aphasia and 1 healthy, non-brain injured control participant was used to investigate the acquisition and maintenance of trained stimuli and generalization to untrained stimuli using a repetition priming protocol. This experiment followed the participants through a training protocol that involved repeated exposure to pictures and their names, along with repeated attempts to name those pictures, to determine the effect of repetition priming on picture naming performance (response accuracy and response time).

Independent variables included stimulus dosage (1 vs. 4 trials per session); generalization variables (trained vs. untrained stimuli; alternate exemplars of trained stimuli), and lexical variables (word frequency; syllable length). Dependent variables included response/reaction time and response accuracy.

PARTICIPANTS

Four individuals with chronic aphasia and one gender-matched healthy non-brain injured control participated in the investigation.

Recruitment

Participants with aphasia and the healthy non-brain injured control participant were recruited from eleven medical facilities located throughout the greater Seattle-Tacoma Metropolitan area. Once a letter of cooperation was obtained from the participating facility, flyers were sent to and circulated by the contact person associated with the facility. The participants were not directly contacted by the PI; instead, potential participants contacted the PI after reading the flyer. Additionally, the investigator was invited to speak about the study at Northwest Hospital's Young Survivor's Stroke Group. Flyers provided general inclusionary/exclusionary criteria, general experimental procedures and information about compensation. Participants were reimbursed for travel expenses including parking and/or bus fare.

Demographics

Persons under the age of 18 were excluded as the experiment was designed to investigate spoken language production of adults with neurological disorders resulting from stroke. No exclusions were made according to gender. No exclusions were made according to race and/or ethnicity. Ethnic and minority populations were recruited according to Seattle, WA demographics obtained from the 2000 US Census. Despite efforts to provide a balanced gender and race/ethnicity distribution, no participants from diverse families chose to participate in the investigation. All of the participants were female and all of the participants were of Northern European descent. Attempts were also made to recruit age- and gender-matched healthy non-brain injured control participants for each of the individuals with aphasia. Despite these efforts, only one healthy age- and gender-matched non-brain injured control was recruited. The primary difficulty with enrollment appeared to stem from the time commitments associated with the protocol.

Consent

Consent documents were sent to the home of the potential participant with aphasia prior to their initial cognitive-linguistic assessment session so that they had sufficient time to review the documents and could ask their caregiver/spouse to assist them with reading if needed. On the first day of cognitive-linguistic evaluation, the principle investigator (PI) presented the consent forms (see appendices A and B). Informed consent procedures were followed in accordance with the approved guidelines of the Human Subjects Division (HSD) at the University of Washington (#05-7338-B03; new 2008 HSD code #28283). Approved consent forms were reviewed with the participants prior to any research activities. The PI explained the purpose and procedures of the study; no deception procedures were conducted. The consent forms emphasized that participation in the study was completely voluntary. Exceptional care was taken by the PI to ensure that participants with aphasia completely understood all components of the study's purpose and procedures; multimodality support was used as needed to ensure complete understanding. In addition to the consent documents, participants reviewed and

signed an audio/video-recording release, medical release of information, and HIPPA agreement forms (see appendices C-E).

Recordings

All cognitive-linguistic evaluation sessions involving participants with aphasia were video-recorded to ensure accurate scoring of the cognitive-linguistic tests and for procedural reliability purposes. The brief cognitive-linguistic battery administered to the healthy non-brain injured control was not video-recorded.

All experimental sessions involving both participants with aphasia and healthy non-brain injured control participants were audio-recorded using an Olympus Digital Voice Recorder (VN-24-PC). Audio recordings were used to verify accuracy and response/reaction time data and to evaluate inter-judge reliability for response accuracy. All transcriptions and accuracy judgments were made using the audio recordings.

Individuals with aphasia

Subject Selection/Enrollment

For enrollment into the study, individuals with aphasia met the following inclusionary criteria:

- native speaker of American English,
- between the age of 21-95,
- demonstrated a medically-documented, single cardiovascular accident to the left hemisphere of the brain (previous transient ischemic attacks were permitted),
- presented as medically stable and at least six months post-cerebral vascular accident (CVA) prior to enrolling in the study, with no subsequent decline,
- mild-to-moderate symptoms of expressive language impairment (with evidence of anomia),
- no other previous or concomitant neurological, psychiatric, or substance abuse disorders, per self report and medical records,
- corrected to normal hearing and vision

Individuals with chronic aphasia were selected for this study because of their stable neurological status.

An initial telephone interview was conducted with either the individual with aphasia or their caregiver to screen for stroke history, time post onset, age, and native language (see appendix F). If these inclusionary criteria were met, individuals with aphasia were scheduled to undergo a comprehensive cognitive-linguistic evaluation. At this time participants were sent a welcome letter, directions to the clinic, and a copy of the consent documents for review.

Subsequent to obtaining consent, personal, medical, and social history was collected from participants (see appendix G). For participants who met the cognitive-linguistic inclusionary criteria listed below, medical records were obtained to confirm medical history pertaining to their CVAs including neurology exam reports, computed tomography (CT) and/or magnetic resonance imaging (MRI) reports and/or scans, speech and language diagnostic reports. Medical information pertinent to the study was recorded on a data entry sheet (see appendix H); medical records were then destroyed.

Cognitive-linguistic evaluations took place in the University of Washington Speech and Hearing Clinic and were conducted by, or under the direct supervision (minimum 50%) of the primary investigator, a certified speech-language pathologist. If the participant had been administered any of the tests within three months of the evaluation (and reports were obtainable) those scores were used in lieu of re-administering the particular test. Refer to table 4.1 for a summary of the tests administered. A sub-set of the cognitive-linguistic battery was re-administered to participants with aphasia at the completion of the study to assess general language change across modalities. This subset included the portions of the WAB required to calculate the Aphasia Quotient, the BNT, and subtest 54 of the PALPA.

Table 4.1 Cognitive-Linguistic Battery and Inclusionary Criteria

Test	Description/Purpose	Inclusionary/Exclusionary criteria
Vision Screening	Snellen chart.	Pass = 20/30 at 2.3 feet with or without glasses/contacts Fail = referral to optometrist prior to enrollment
Hearing Screening	Portable audiometer. Tested best ear at 500, 1000, 2000, & 4000 Hz.	Pass = 35 dB Fail = referral to audiologist prior to enrollment
Structural/Functional Exam	To examine oral structures and their functions. To document possible peripheral contributors of dysarthria.	Descriptive only
Informal Assessment of Visual Agnosia	To rule out visual/perceptual deficits. Participant presented with 10 common objects and asked to demonstrate use of objects using hands.	Pass = raw score > 8/10 Fail = excluded from study
Western Aphasia Battery (WAB) <i>Kertesz (1982)</i>	To assess language across modalities. Administered only those subtests needed to calculate Aphasia Quotient (AQ).	Pass = AQ > 25/100 Fail = excluded from study
Boston Naming Test (BNT) <i>Goodglass & Kaplan (1983)</i>	To assess word-finding abilities.	Descriptive only Raw score = ___/60
Raven's Coloured Progressive Matrices <i>Raven (1976)</i>	To assess non-verbal problem solving (non-linguistic cognitive abilities).	Pass = >12/36 Fail = excluded from study
Apraxia Battery for Adults (ABA) <i>Dabul (1979)</i>	To assess motor planning/programming.	Pass = no scores in "Severe to Profound" range on "Profile Score Sheet". No more than 3 items on "Checklist of Apraxic Features" Fail = excluded from study
Subtests 1-3 of the Reading Comprehension Battery for Aphasia (RCBA) <i>LaPoint & Homer (1979)</i>	To assess single word reading ability.	Descriptive only Raw score = ___/30
Pyramids and Palm Trees Test <i>Howard & Patterson (1992)</i>	A test of semantic access. To assess the participant's ability to access detailed semantic representations from words and pictures.	Descriptive only Raw score = ___/52
Subtest 54 of the Psycholinguistic Assessment of Language Processes in Aphasia (PALPA) <i>Kay, Lesser, & Coltheart (1992)</i>	To assess confrontational picture naming with high and low frequency stimuli.	Descriptive only Raw score = ___/60
Trial Run of Training Protocol	To assess the participant's ability to participate in the experimental protocol (computer-based). Twenty-five pictures presented. Participants asked to name pictures.	Pass = >5/25 Fail = re-test to see if participant can learn task; multiple failures results in exclusion from study

Exclusionary Criteria

Individuals with aphasia were excluded if they demonstrated: (1) a severe-to-profound expressive language impairment that precluded them from participating in the training protocol (e.g., unable to produce single words), (2) a severe-to-profound receptive language impairment that interfered with protocol completion, (3) a severe-to-profound concomitant apraxia of speech, (4) a moderate-to-profound concomitant dysarthria, (5) a pronounced cognitive and/or memory impairment. Individuals with aphasia who could not repeat single words to some degree when given an auditory presentation of a word were also excluded.

Case #1: A102

Participant A102 is a 90 year-old female who presented with a left middle cerebral artery (MCA) embolic CVA that had occurred six months prior to the initial cognitive-linguistic testing session. According to the speech-language pathology assessment conducted while the participant was hospitalized, A102 demonstrated moderate-severe fluent aphasia impacting all modalities. At the time of enrollment into this study, A102 presented with moderately severe expressive aphasia and a minimal to mild receptive language impairment. She was able to repeat single words and short sentences with no errors; her ability to repeat broke down with more difficult sentences, containing less familiar words (e.g., "The pastry cook was elated"). Confrontational picture-naming was moderately to severely impaired, typically characterized by non responses and phonemic paraphasias. A102's spontaneous expressive language was characterized by short grammatically correct sentences composed of occasional correct concrete noun production accompanied by multiple phonemic paraphasias most frequently resulting in nonwords (e.g., /kIlg/ for "shrimp") and infrequent semantic paraphasias (e.g., "rat" for "mouse").

Case #2: A103

Participant A103 is a 47 year-old female who presented with a hemorrhagic left temporal lobe CVA that had occurred three and a half years prior to the initial cognitive-linguistic testing session. She presented with moderate expressive aphasia and severe receptive aphasia (in the absence of written cues). As a result

of her severe receptive aphasia, A103's ability to repeat was severely impaired. A103's confrontational picture-naming was also severely impaired, most frequently characterized by non-responses. Her spontaneous expressive language was characterized by complete, grammatically correct, sentences composed of a minimal to moderate number of naming errors characterized by semantic paraphasias (e.g., "broom" for "mop"), circumlocutions or picture descriptions (e.g., "I say law" for "judge"), and non-responses (e.g., "I forgot what it's called").

Case #3: A104

Participant A104 is a 76 year-old female who presented with a left basal ganglia CVA that had occurred approximately one and a half years prior to the initial cognitive-linguistic testing session. She presented with moderate to severe expressive aphasia, minimal receptive aphasia, and minimal dysarthria. A minimal right droop of A104's lips was observed during a structural-functional exam. No other structural-functional abnormalities were observed at the time of enrollment. A104's ability to repeat was intact, with only occasional errors during repetition of longer, more syntactically complex sentences. Her confrontational picture-naming varied from minimal to moderately-severe depending on the familiarity of the target; she performed at ceiling levels for naming items presented during completion of the WAB, but performed at chance for naming items presented during completion of the Boston Naming Test, which is composed of increasingly less familiar target items. A104's spontaneous expressive language was characterized by medium length grammatically correct sentences composed of a minimal to moderate number of naming errors characterized by semantic paraphasias (e.g., "ant" for "cricket") and non-responses (e.g., "um, oh gosh").

Case #4: A106

Participant A106 is a 78 year-old female who presented with an embolic/thrombotic (exact nature not reported in medical records) left MCA CVA that had occurred eight months prior to the initial cognitive-linguistic testing session. The participant's CT report indicated a large left hemisphere lesion occurring in the left

frontal lobe including the anterior insular region and left frontal operculum. A106 presented with severe expressive aphasia and minimal receptive aphasia. A106 was able to repeat single words and portions of short phrases and sentences. Her confrontational picture naming was at chance levels across all assessments administered. Her spontaneous expressive language was characterized by single words and short phrases (approximately 2-3 words per phrase) attempts, characterized by a multiple phonemic paraphasias resulting in non-words (e.g., /pɹɪmp/ for “shrimp”). Although A106 often used written language (single words or short phrases) as a compensatory strategy during conversational speech, the same phonemic paraphasias observed during verbal production were observed during written dictation tasks as well. She was also able to produce short sentences with a written model (e.g., “Turn up the heat”). Please refer to table 4.2 for a summary of the participants’ profiles and scores on the cognitive-linguistic batteries.

Table 4.2 Profiles of Individuals with Aphasia

	A102		A103		A104		A106	
Age	90		47		76		78	
Gender	Female		Female		Female		Female	
Time Post CVA	6 months		3.5 yrs		1.5 yrs		8 months	
Type of CVA	Embolic		Hemorrhagic		Hemorrhagic		Embolic/Thrombotic	
Lesion Location	L MCA		L Temporal lobe		L Basal Ganglia		L MCA; L frontal lobe, anterior insular region, L frontal operculum	
Cognitive-Linguistic Battery	Pre	Post	Pre	Post	Pre	Post	Pre	Post
WAB AQ	73.9	61.4	60.2	70.8	82.8	81	54.1	Pending
WAB Aphasia Classification	Anomic		Wernicke's/ Anomic		Anomic		Conduction	
BNT	10/60	14/60	5/60	10/60	27/60	24/60	12/60	14/60
PALPA	25/60	43/60	26/60	38/60	50/60	52/60	35/60	37/60
Cognitive-Linguistic Battery	Administered only once at study onset. Data used for inclusionary & descriptive purposes.							
Pyramids & Palms	38/52		48/52		48/52		49/52	
ABA	No apraxia		No apraxia		No apraxia		DNT - Complicated by Conduction Aphasia/possible AOS	
RCBA	29/30		30/30		30/30		30/30	
Visual Agnosia	10/10		10/10		10/10		10/10	
Raven's	18/36		36/36		20/36		18/36	
BDI-II	3/63		10/63		18/63		7/63	

Non-brain injured, healthy control participant

Subject Selection/Enrollment

A single non-brain injured, healthy control participant was recruited to match the gender and race/ethnicity of the individuals with aphasia. For enrollment into the study, the control participant met the following inclusionary criteria:

- native speaker of American English,
- no known history of neurologic, speech, or language deficits (per self-report),

- no known psychiatric condition or substance abuse condition that might interfere with protocol completion (per self report),
- corrected to normal hearing and vision

An initial phone interview (see appendix I) was conducted with the potential participant to screen for age, ethnic background, and native language. If these inclusionary criteria were met, the non-brain injured healthy control participant was scheduled to undergo a brief cognitive-linguistic evaluation. Subsequent to obtaining consent, a brief personal, medical, and social history was collected from the participant (see appendix J).

The cognitive-linguistic evaluation took place in the University of Washington Speech and Hearing Clinic and was conducted by, or under the direct supervision (minimum 50%) of the researcher, a certified speech-language pathologist. Refer to table 4.3 for a summary of the tests administered. The non-brain injured healthy control participant did not undergo post-experimental re-assessment.

Table 4.3 Summary of Assessments Administered to Control Participant

Test	Description/Purpose	Inclusionary/Exclusionary Criteria
Vision Screening	Snellen chart.	Pass = 20/30 at 2.3 feet with or without glasses/contacts Fail = referral to optometrist prior to enrollment
Hearing Screening	Portable audiometer. Tested best ear at 500, 1000, 2000, & 4000 Hz	Pass = 35 dB Fail = referral to audiologist prior to enrollment
Edinburgh Handedness Test <i>Oldfield (1971)</i>	To assess handedness (language lateralization)	Descriptive only
Raven's Coloured Progressive Matrices <i>Raven (1971)</i>	To assess non-verbal problem solving (non-linguistic cognitive abilities)	Pass = >12/36 Fail = excluded from study

Participant C102 is a 53 year-old female with Northern European (Norwegian) ancestry who was selected as a gender-matched control for the participants with aphasia. She has no history of neurological, speech, language, or hearing disorders or substance abuse. Her hearing and vision were within normal limits. C102 scored within normal limits on the Raven's Coloured Progressive

Matrices (35/36) and scored as right-hand dominant on the Edinburgh Handedness Test.

PROCEDURES

Stimuli & instrumentation

Stimuli

Target stimuli were randomly selected from a previously developed corpus of 240 color photographs depicting 1- and 2-syllable concrete nouns. This corpus was developed and refined across three studies including the pilot study described in detail in the previous chapter (Kenny, 2006; Krohn, 2005; Potts, 2006). Digitized color photographs, assessed for easy recognition, depict the target word on a yellow background (see figure 4.1 for a sample item). The corpus is composed of 130 high-frequency words and 110 low-frequency words. High-frequency words are defined as greater than or equal to 150 instances per million words; low-frequency words are defined as less than or equal to 20 instances per million words (Francis & Kucera, 1982).

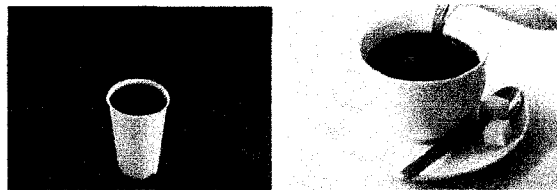


Figure 4.1 Sample Stimulus Item and Alternate Exemplar, "Coffee"

From this corpus, 40 words were randomly selected as trained stimuli and 100 pictures were randomly selected as untrained stimuli. Trained and untrained stimuli were balanced across word frequency and syllable length. Additionally, in order to assess participants' responses to alternate exemplars of trained stimuli (i.e., stimulus generalization), different photographs were selected for each of the 40 trained items (refer to figure 4.1 for a sample alternate exemplar). Refer to appendix K for a list of trained and untrained items. During training sessions visual target stimuli were accompanied by the spoken and written name of the picture. Auditory

stimuli were previously recorded and edited for duration using Computerized Speech Lab 410.

Instrumentation

Cognitive-linguistic batteries for individuals with aphasia were video-recorded in the University of Washington Speech and Hearing Clinic using video cameras that are built into the ceiling and mounted on the walls of the clinic room and a Panasonic DVD Recorder, Model DMR-T6070 that is located in the observation room adjacent to the clinical treatment room.

Experimental sessions were carried out using E-Prime (Schneider, Eschman & Zuccolotto, 2002) on a Micron Millennia computer. Participants were seated comfortably in front of the computer monitor. A head-mounted microphone (AKG Acoustics, MicroMic Series III, model C 420^{III} PP) was used to record response/reaction time. The microphone was routed through a TubeMP Project Series pre-amplifier to allow for individually-based calibration of voice onset detection. Microphone calibration took place immediately prior to every probe and training session. The pre-amplifier was then connected to a serial response box (Psychology Software Tools, Pittsburgh, PA) which was then interfaced with the computer. Response/reaction time, in milliseconds, was collected by E-Prime based on the time between the onset of the visual stimulus and the initiation of voicing of the response.

Audio files containing the names of the pictures were imported into the delivery software (E-Prime) and were presented through Bose QuietComfort 2 Acoustic Noise-Canceling headphones. These files were presented at a level audible to each participant. Calibration of the headphone volume delivery occurred immediately prior to each probe and/or training session.

To verify accuracy and reaction time data, participants' responses were recorded using an Olympus Digital Voice Recorder (VN-24-PC). These audio files were also used to evaluate reliability for response accuracy, as discussed below.

Delivery Schedule & Protocol Details

Baseline Probe Sessions

Following completion of cognitive-linguistic testing and subsequent enrollment into the study, participants were administered four baseline probes to assess pre-training picture-naming performance. For participants with aphasia, each of the four baseline probe sessions took place on a separate day within a two-week period. For the control participant, the baseline probes were administered at the convenience of the participant as long as all four were completed within a two-week period. Multiple baseline probes were permitted to occur within single day for control participants. Please refer to figure 4.2 for a visual depiction of a sample delivery schedule.

SESSION #	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Baseline Phase	█																
Training Sessions					█												
Training Probes							█			█			█				
Stimulus Generalization Maintenance							█			█			█	█			

Figure 4.2 Delivery Schedule

During each baseline probe session, participants were instructed to name 60 target pictures (40 "trained" pictures and 20 randomly selected "untrained" pictures) aloud once as quickly as possible while maintaining accuracy; participants were discouraged from self-correcting errors, coughing, and/or clearing their throats during probe sessions. Target pictures were presented randomly. Each trial proceeded as follows: a black fixation mark (*) appeared at the center of a white computer screen; the target picture then appeared at the center of the computer screen during which time the participant attempted to name the picture. A red "X" then appeared in the middle of a white screen to indicate that the participant must stop attempting to name the picture. The black fixation point then reappeared to prepare the participant for the subsequent target. A completion message was presented on the computer screen to indicate the end of the session. Please refer to

appendix L for a list of the timing parameters associated with the delivery of the fixation marks, targets, and red "Xs" for all probe and training sessions.

Training Sessions

Training sessions were initiated no more than two weeks following baseline testing. Participants with aphasia attended training sessions 2-3 times per week until they reached 80% accuracy, or for a maximum of 15 training sessions. The control participant was administered a total of nine training sessions within a two-week period. Multiple training sessions were permitted within a single day. As such, the training delivery schedule varied from participant to participant. Participant-specific delivery schedules are discussed in detail in the results section.

Forty target pictures were randomly selected as trained stimuli; these forty items were balanced across word frequency and syllable length. Furthermore, trained target stimuli were repeated either 1 time or 4 times during each training session to assess differential effects of stimulus dosage. These 100 target pictures (20 1-trial/session targets; 20 4-trials/session targets) were presented randomly; intervals between repetitions were not controlled.

During each training session, participants were instructed to name pictures aloud as quickly as possible while maintaining accuracy; they were discouraged from self-correcting errors, coughing and/or clearing their throat. Each trial proceeded as follows: a black fixation mark (*) appeared at the center of a white computer screen; the target picture appeared at the center of the computer screen, during which time the participant attempted to name the picture. A red "X" then appeared in the middle of a white screen to indicate to the participant that they must stop attempting to name the picture. The picture then reappeared, accompanied by both the auditory presentation of the name of the target and the orthographic form. A red "X" then appeared in the middle of the white screen to indicate to the participant that they must stop attempting to repeat the name of the picture. The black fixation mark then reappeared to prepare the participants for the next target. The 100 target pictures were divided equally into five runs (20 targets per run) with breaks provided as needed between runs. A completion message was presented on

the computer screen to indicate the end of each run. Refer to figure 4.3 for a visual depiction of this protocol.

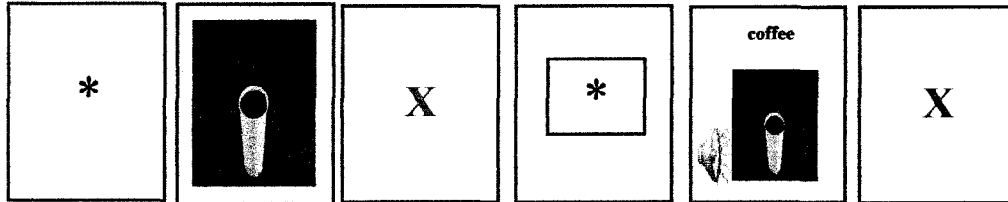


Figure 4.3 Training Session Depiction

Training Probe Sessions

Training probes were administered immediately after every third training session and immediately prior to every fourth training session to assess both immediate and delayed effects of training on response accuracy and response time. Each training probe assessed response accuracy and response/reaction time of all 40 trained items in addition to 20 randomly selected untrained pictures to assess generalization to untrained stimuli.

During each training probe session, participants were instructed to name the 60 target pictures aloud once as quickly as possible while maintaining accuracy; participants were discouraged from self-correcting errors, coughing, and/or clearing their throats during probe sessions. Target pictures were presented randomly. Each trial proceeded as follows: a black fixation mark (*) appeared at the center of a white computer screen; the target picture then appeared at the center of the computer screen during which time the participant attempted to name the picture. A red "X" then appeared in the middle of a white screen to indicate that the participant must stop attempting to name the picture. The black fixation point then reappeared to prepare the participant for the subsequent target. A completion message was presented on the computer screen to indicate the end of the session.

Generalization Probe Sessions

Stimulus generalization probes, during which participants were asked to name alternate exemplars of the trained stimuli, were administered immediately after every third training session and immediately prior to every fourth training

session to assess both immediate and delayed effects of training on response accuracy and response time. Each stimulus generalization probe assessed response accuracy and response/reaction time of 20 randomly selected alternate exemplars of the trained items.

During each stimulus generalization probe session, participants were instructed to name the 20 target pictures aloud once as quickly as possible while maintaining accuracy; participants were discouraged from self-correcting errors, coughing, and/or clearing their throats during probe sessions. Target pictures were presented randomly. Each trial proceeded as follows: a black fixation mark (*) appeared at the center of a white computer screen; the target picture then appeared at the center of the computer screen during which time the participant attempted to name the picture. A red "X" then appeared in the middle of a white screen to indicate that the participant must stop attempting to name the picture. The black fixation point then reappeared to prepare the participant for the subsequent target. A completion message was presented on the computer screen to indicate the end of the session.

Maintenance Probe Sessions

Participants returned three times beginning six weeks following the last training session to assess behavioral performance after training had been withdrawn. Each maintenance probe assessed response accuracy and response/reaction time of all 40 trained items in addition to 20 randomly selected untrained pictures to assess generalization to untrained stimuli.

During each maintenance probe session, participants were instructed to name the 60 target pictures aloud once as quickly as possible while maintaining accuracy; participants were discouraged from self-correcting errors, coughing, and/or clearing their throats during probe sessions. Target pictures were presented randomly. Each trial proceeded as follows: a black fixation mark (*) appeared at the center of a white computer screen; the target picture then appeared at the center of the computer screen during which time the participant attempted to name the picture. A red "X" then appeared in the middle of a white screen to indicate that the participant must stop attempting to name the picture. The black fixation point then

reappeared to prepare the participant for the subsequent target. A completion message was presented on the computer screen to indicate the end of the session.

Data Collection and Analysis

Response Accuracy Data

The experimenter transcribed all responses verbatim during all training and probe sessions. The experimenter subsequently reviewed 100% of the recorded data of probes sessions to ensure accurate transcription of participants' responses. The experimenter then coded the transcribed responses. Initially, responses were coded using a binary +/- coding system. Accurate (+) responses reserved for analysis included: (1) the exact production of the target (e.g., "coffee"); (2) the target plus a filler (e.g., "um/the/a coffee"); (3) multiple correct productions (e.g., "coffee...coffee"); or (4) multiple productions/production attempts with the first attempt being correct (e.g., "coffee...croffee"). Erred responses were subsequently assigned an error code according to an error code taxonomy adapted from the Philadelphia Naming Test (Roach, Schwartz, Linebarger, Martin, & Bochetto, 1988). The error code taxonomy can be found in appendix M. Error data was used for analysis in a separate investigation (Kavalier, 2008) and will not be discussed in this paper.

Descriptive statistics including means, ranges, and standard deviations for response accuracy were calculated for each participant, for each phase of the experimental protocol relative to independent variables. Line graphs were produced for each participant depicting performance across phases of the experimental protocol (i.e., baseline phase, training phase, and maintenance phase) for trained vs. untrained items, 1-trial vs. 4-trials/session items, and for the stimulus generalization probes. Visual analysis of the line graphs was used to interpret level, trend, variability, onset of training effects, and the magnitude of change relative to baseline performance.

Response Time/Reaction Time Data

Response times⁴³ for individuals with aphasia and reaction times for the control participant were detected and recorded by E-Prime during all sessions; the digital voice recorder also tracked time by millisecond for those responses not detected by E-Prime. Response times from all erred responses were removed prior to data analysis. Response times less than 250 ms were removed from the data set for each participant. Furthermore, as latencies for individuals with aphasia are characterized by variability, outliers four standard deviations or greater from the individual's mean performance were removed from the data set for each probe session (Moreno, Buchanan, & Van Orden, 2002).

Descriptive statistics including means, ranges, and standard deviations of response/reaction time were calculated for the remaining latencies for each participant for each phase of the experimental protocol relative to independent variables. Line graphs were produced for each participant depicting performance across phases of the experimental protocol (i.e., baseline phase, training phase, and maintenance phase) for trained vs. untrained items, 1-trial vs. 4-trials/session items, and for the stimulus generalization probes. Visual analysis of the line graphs was used to interpret level, trend, variability, onset of training effects, and the magnitude of change relative to baseline performance.

Reliability Procedures

One judge, uninvolved in data collection, transcribed (i.e., glossed) 100% of the audio-recorded probe data for all individuals with aphasia and the healthy control participant. When phonemic errors were made by the participant, resulting in nonwords, the reliability judge transcribed the utterance using the International Phonetic Alphabet (IPA). After transcribing and recording the participants' responses, the reliability judge made a binary +/- accuracy judgment for each trial, following the accuracy rules described above. A second judge, also uninvolved in data collection, reviewed the first reliability judge's transcriptions and assigned error

⁴³ Response times differ from reaction times in that individuals with aphasia were not encouraged to respond as quickly as possible. Instead, they were simply asked to name the picture. The term "reaction time" was reserved for the control participant, who was asked to name the picture as quickly as possible without making an error.

codes according to the error coding taxonomy presented in appendix M. Both judges were blind to the original transcriber's transcriptions and accuracy judgments. Judges were trained to transcribe and assign error codes prior to the onset of examining data. Judges completed all necessary HIPPA and Human Subject's training prior to viewing participant data. Cohen's Kappa was used to calculate inter-judge reliability for the binary accuracy judgment between the experimenter and reliability judge for each subject (see table 4.4); collectively, across all subjects, inter-judge reliability was 0.88. Error code reliability will not be presented in this paper.

Table 4.4 Inter-Judge Reliability (Cohen's Kappa)

Participant	Kappa Statistic
A102	0.89
A103	0.86
A104	0.95
A106	0.67
Overall	0.88

Some difficulties were encountered during transcription from the digital audio recorder for participants whose errors largely consisted of phonemic paraphasias. The sensitivity of the microphone was not ideal for detailed transcription. This poor sensitivity, however, did not influence the binary +/- judgment for either the experimenter or the reliability judge; as such, calculating inter-judge reliability was not influenced by this technical difficulty. Reliability difficulties for error coding analysis stemming from this reduced audibility are discussed in detail in the investigation being completed by Kavalier (2008).

CHAPTER V: RESULTS

PARTICIPANT DELIVERY SCHEDULES & STIMULUS DOSAGE

A102 participated in 2-3 training sessions per week, as her schedule permitted, for a total of 15 training sessions. Over the course of the training protocol, A102 was exposed to the trained items a total of 600 times; she attempted to name the trained 1-trial/session pictures 600 times and the 4-trials/session pictures 2400 times across the training protocol. She returned for three maintenance probes at six weeks, seven weeks and eight weeks following her last training session. Please refer to table 5.1 for a summary of the participants' stimulus dosage.

A103 participated in two training sessions per week for a total of six training sessions. Over the course of the training protocol, A103 was exposed to the trained items a total of 240 times; she attempted to name the trained 1-trial/session pictures 240 times and the 4-trials/session pictures 960 times across the training protocol. She returned for the first of three maintenance probes beginning six weeks following her last training probe. The second two maintenance probes were completed during the seventh week following her last training probe.

A104 participated in two training sessions per week for a total of 12 training sessions. Over the course of the training protocol, A104 was exposed to the trained items a total of 480 times; she attempted to name the trained 1-trial/session pictures 480 times and the 4-trials/session pictures 1920 times across the training protocol. She returned for three maintenance probes, all of which occurred during the sixth week following her last training probe.

A106 participated in two training sessions per week for a total of 15 training sessions. Over the course of the training protocol, A106 was exposed to the trained items a total of 600 times; she attempted to name the trained 1-trial/session pictures 600 times and the 4-trials/session pictures 2400 times across the training protocol. Maintenance probes are pending. A106 returned for three maintenance probes, the first of which occurred during the sixth week following her last training probe; the second and third maintenance probes occurred during the seventh week following her last training probe.

C102 participated in two training sessions per day for three days for a total of nine training sessions. Breaks lasting between ten minutes and two hours were given between the training sessions that occurred on the same day. Over the course of the training protocol, C102 was exposed to the trained items a total of 160 times; she named the 1-trial/session pictures 360 times and the 4-trials/session pictures 1440 times across the training protocol. C102 returned for three maintenance probes, the first of which occurred during the sixth week following her last training probe; the second and third maintenance probes occurred during the seventh week following her last training probe.

Table 5.1 Stimulus Dosage by Participant

	A102	A103	A104	A106	CONTROL
Total Training Sessions	15	6	12	15	9
Total Exposures	600	240	480	600	160
Total Naming Attempts Without support	300;1200	120;480	240;960	300;1200	180;720
Total Naming Attempts With Support	300;1200	120;480	240;960	300;1200	180;720
Total Naming Attempts	600;2400	240;960	480;1920	600;2400	360;1440

ACCURACY DATA

Trained vs. Untrained Items

Descriptive Statistics

Descriptive statistics including means and standard deviations were calculated for each participant across each phase of the experimental protocol for trained and untrained items relative to response accuracy. Refer to table 5.2 for a summary of means and standard deviations.

Table 5.2. Mean Response Accuracy for Trained vs. Untrained Items

		A102	A103	A104	A106	CONTROL
Baseline Phase	Untrained	35% (11)	46% (13)	83% (12)	38% (4)	98% (3)
	Trained	41% (5)	60% (8)	90% (5)	47% (6)	99% (1)
Training Phase	Untrained	34% (5)	35% (21)	79% (8)	34% (14)	94% (5)
	Trained	70% (8)	93% (10)	95% (4)	54% (9)	99% (1)
Maintenance Phase	Untrained	32% (12)	43% (13)	70% (8)	40% (13)	93% (0.06)
	Trained	78% (1)	94% (4)	90% (5)	60% (3)	100% (0)

()=standard deviation

Visual Analysis

Line graphs were produced for each participant to depict percent response accuracy across phases of the experimental protocol for trained and untrained item (see figures 5.1-5.4). A line graph was not produced for the control participant as she was at near-ceiling performance in terms of response accuracy for the entire protocol. However, trained items were consistently named more accurately than untrained items for the control participant across all probe sessions⁴⁴.

A102. Across the four baseline probes, A102 averaged 38% accuracy (range=20-45%; SD=8.4); no visual differences between trained (mean=41%; range=38-45%; SD=5.2) and untrained (mean=33%; range= 20-45%; SD=10.8) items were observed during the baseline phase except for the first probe at which time trained items (38%) were responded to more accurately than untrained items (20%). During the training phase, a large split between trained and untrained items is observable by visual inspection, with trained items being responded to more accurately than untrained items. A102 demonstrated a 22% increase, relative to baseline, for response accuracy of trained items at the time of the first training probe (onset). A positive but low magnitude slope for was observed for trained items ($y=1.75x+61.25$; $R^2=0.35$) relative to untrained items ($y=0.33x+32.22$; $R^2=0.0353$) throughout the training phase. Occasional dips in accuracy were observed for the

⁴⁴ The items missed by the control participant were consistent across phases of the protocol. That is, the items responded to inaccurately during baseline were also responded to inaccurately during the training and maintenance phases of the protocol. No changes in accuracy were observed as a result of training.

delayed training probes relative to the immediate training probes. Towards the end of the training protocol, however, response accuracy was no longer influenced by the length of time between training sessions and probes (i.e., immediate vs. delayed). By the end of the training phase (i.e., the last two training probes), A102 averaged 79% accuracy on trained items, reflecting a 38% increase in response accuracy relative to baseline performance. This increase in response accuracy for trained items persisted throughout the maintenance phase; she averaged 78% for trained items (range= 78-80%; SD=1) and 32% for untrained items (range=25-45%; SD=12) across the three maintenance probes. Response accuracy for untrained items during the training and maintenance phases remained within the participant's baseline performance rate. Refer to figure 5.1 for A102's naming performance for trained vs. untrained items across experimental phases.

A103. Across the four baseline probes, A103 averaged 53% accuracy (range=35-65%; SD= 12). Trained items (mean=60%; range=48-65%; SD=8) were responded to slightly more accurately than untrained items (mean=46%; range=35-65%; SD=13) during the baseline phase. With the onset of the training phase, however, an immediate and marked split occurred between trained and untrained items. Relative to the baseline phase mean response accuracy, A103 demonstrated a 38% increase in response accuracy for trained items at the time of the first training probe; however, her response accuracy for trained items did not continue increase as a result of further training ($y=2x+87.5$; $R^2=0.0667$). Untrained items were characterized by a moderate negative slope ($y=-12x+65$; $R^2=0.5442$). By the end of the training phase (i.e., the last two training probes), A103 averaged 98% accuracy for the trained items and 18% accuracy for the untrained items. A dip in response accuracy for trained items was observed for the first delayed probe during the training phase. A103's response accuracy was not influenced by the time from training session to training probe (i.e., immediate vs. delayed probe) for the rest of the training phase. A103's increased accuracy for trained items relative to untrained items persisted throughout the maintenance phase; she averaged 94% for trained items (range=90-98%; SD=4) and 43% for untrained items (range=30-55%; SD= 13) across the maintenance probes. A103 demonstrated an observable dip in response accuracy for the untrained items during the training phase of the experiment (15-

20%); however, her response accuracy for untrained items returned to baseline performance range (35-65%) during the maintenance phase. Refer to figure 5.2 for A103's naming performance (response accuracy) for trained vs. untrained items across experimental phases.

A104. Across the four baseline probes, A104 averaged 86% accuracy (range=75-100%; SD=9). No consistent visual differences were observed between trained (mean=90%; range=85-95%; SD=5) and untrained (mean=83%; range=75-100%; SD= 12) items during the baseline phase. During the first half of the training phase a split can be observed between trained and untrained items as trained items are characterized by a minimally positive slope ($y=0.2381x+93.93$; $R^2=0.0238$) as they reach near ceiling to ceiling levels; however, this gap narrows as untrained items become more reliably produced by the end of the repetition priming protocol ($y=2.381x+68.036$; $R^2=0.5442$). This gap appears to represent a decrease in response accuracy for untrained items as opposed to an increase in accuracy for trained items. The onset of this split, however, was immediate. By the end of training (i.e., the last two training probes), A104 averaged 98% accuracy for trained items and 83% accuracy for untrained items. The split between trained items relative to untrained items persisted through the maintenance phase; she averaged 90% for trained items (range=85-95%; SD= 5) and 70% for untrained items (range= 65-80%; SD=8). Refer to figure 5.3 for A104's naming performance (response accuracy) for trained vs. untrained items across experimental phases.

A106. Across the four baseline probes, A106 averaged 42% accuracy (range=30-53%; SD= 7). Trained items were responded to ten percent more accurately (mean=47%, range=43-53%; SD=4) than untrained items (mean= 38%; range=30-45%; SD=7) during the baseline phase; this difference appears to be a result of less stable production of untrained items relative to trained items. During the training phase, a steady increase in response accuracy was observed for trained items ($y=3.006x+38.661$; $R^2=0.7698$), while untrained items were responded to much less consistently with no observable change in response accuracy ($y=1.7857x+26.964$; $R^2=0.103$). Approximately five percent drops in response accuracy were observed for delayed vs. immediate probes for the first half of the training phase. The influence time of probe following training disappears by the

fourth probe session. By the end of the training phase (i.e., the last two training probes), A106 averaged 63% accuracy for trained items and 30% accuracy for untrained items. Although A106 demonstrated a steady increase in response accuracy for trained relative to untrained items, improvement relative to baseline did not occur until the third training probe (i.e., after nine training sessions). A106 demonstrated an increase in response accuracy for untrained items at the third probe session (both immediate and delayed); however, her performance returned to within baseline performance rate at the final probes (both immediate and delayed). The split between trained items relative to untrained items persisted through the maintenance phase; she averaged 60% for trained items (range=58-63%; SD= 3) and 40% for untrained items (range= 30-55%; SD=13). Refer to figure 5.4 for A106's naming performance (response accuracy) for trained vs. untrained items across experimental phases.

C102. Participant C102 performed at near-ceiling levels for response accuracy (93-100%) for all phases of the experimental protocol. As such, her data will not be presented in this section of results.

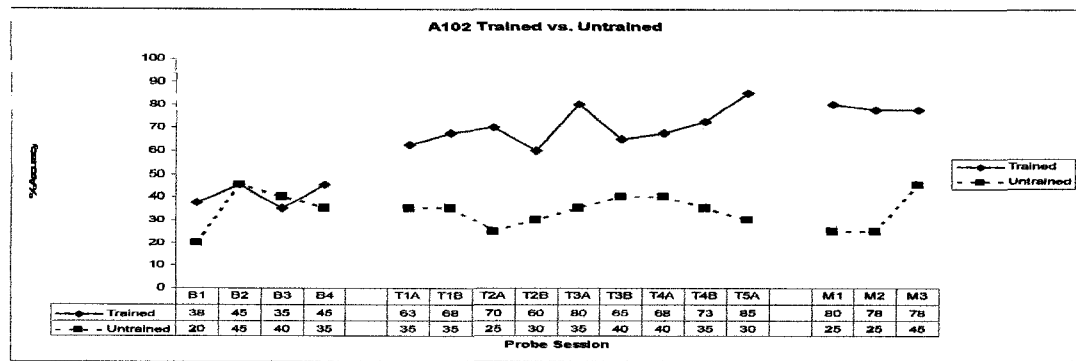


Figure 5.1 A102 Response Accuracy for Trained vs. Untrained Items

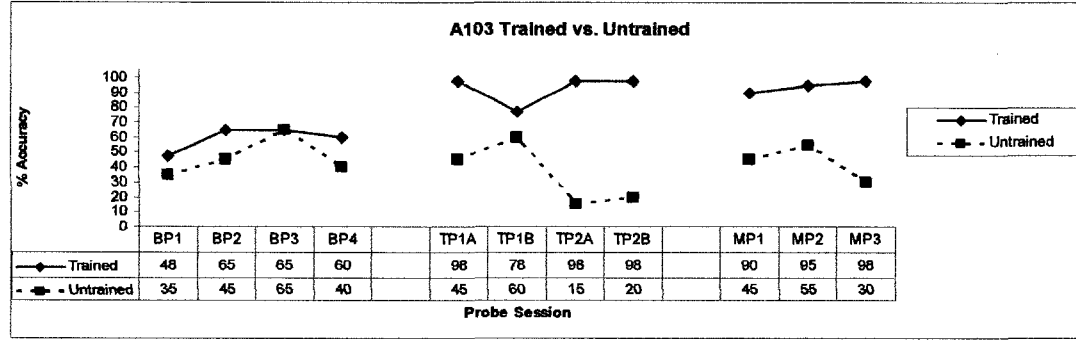


Figure 5.2 A103 Response Accuracy for Trained vs. Untrained Items

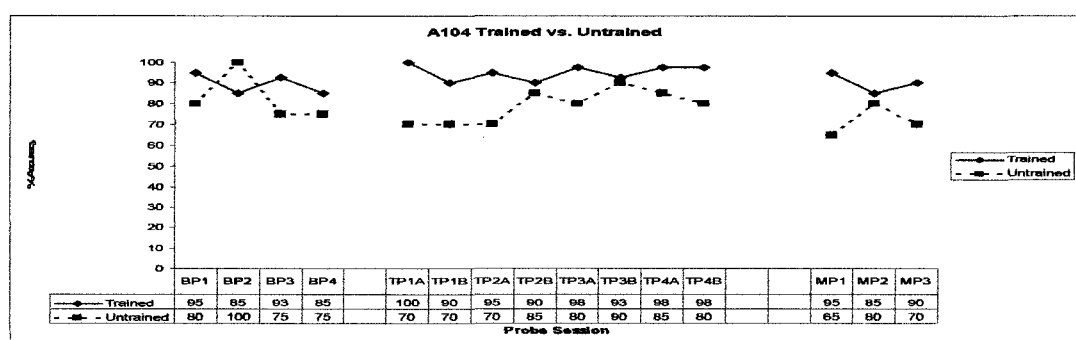


Figure 5.3 A104 Response Accuracy for Trained vs. Untrained Items

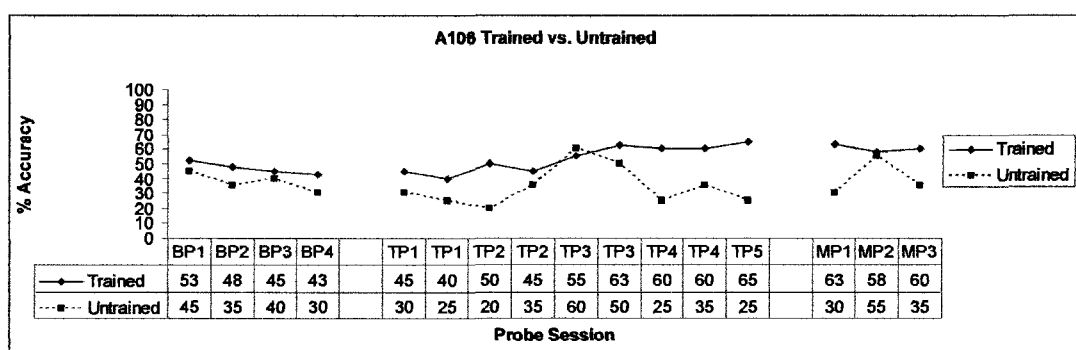


Figure 5.4 A106 Response Accuracy for Trained vs. Untrained Items

1-Trial vs. 4-Trials per Session

Descriptive Statistics

Descriptive statistics including means and standard deviations were calculated for each participant across each phase of the experimental protocol for 1-trial/session and 4-trials/session items relative to response accuracy. Please refer to table 5.3 for a summary of these means and standard deviations. The control participant responded to 100% of the pictures accurately. As such, standard deviations are not provided for her data.

Table 5.3 Mean Response Accuracy for 1- vs. 4-Trials/Session Items

		A102	A103	A104	A106	CONTROL
Baseline Phase	Untrained	35% (11)	46% (13)	83% (12)	38% (4)	98% (3)
	Trained	41% (5)	60% (8)	90% (5)	47% (6)	99% (1)
Training Phase	1 Trial	66% (13)	94% (6)	96% (4)	61% (14)	99% (0.02)
	4 Trials	74% (10)	91% (14)	94% (6)	46%(9)	100% (0)
Maintenance Phase	1 Trial	70% (10)	92.7% (8)	90% (0)	65% (0)	100% (0)
	4 Trials	86.7% (8)	96.7% (3)	90% (10)	55% (5)	100% (0)

Visual Analysis

Line graphs were produced for each participant to depict response accuracy across phases of the experimental protocol for 1-trial and 4-trials/session items (see figures 5.1-5.4). A line graph was not produced for the control participant as she was at ceiling performance in terms of response accuracy for the entire protocol.

A102. Across the four baseline probes, A102 averaged 41% response accuracy for trained items (range=30-50%). Items selected to be 1-trial/session (mean=43%; range=30-50%; SD=10) appeared to be less stable than 4-trials/session (mean=39%; range=35-40%; SD=3) during the baseline phase. During the training phase, both 1- and 4-trials/session items demonstrated an immediate increase in response accuracy. However, 4-trials/session items demonstrated a steady increase in response accuracy ($y=3.1667x+58.056$; $R^2=0.8143$) while 1-trial/session items were responded to much less reliably, with no noticeable change in response accuracy during the course of the training phase ($y=0.333x+64.4$; $R^2=0.0048$). By the end of training (i.e., the last two training probes), A102 averaged

70% accuracy for 1-trial/session items and 88% accuracy for 4-trials/session items. However, both 1- and 4-trials/session items were responded to with 85% accuracy at the time of the final training probe. This overall 33% increase of response accuracy for 4-trials/session items and 25% increase in response accuracy for 1-trial/session items (relative to baseline) persisted through the maintenance phase of the protocol; A102 averaged 70% accuracy for 1-trial/session items (range=60-80%; SD=10) and 87% accuracy for 4-trials/session items (range=80-95%; SD=8) during the maintenance phase. Refer to figure 5.5 for A102's response accuracy for 1 vs. 4 trials per session across experimental phases.

A103. Across the four baseline probes, A103 averaged 60% response accuracy for trained items (range=45-70%). No differences were observed between items selected to be 1-trial/session (mean=61%; range=50-70%; SD=9) vs. 4-trials/session (mean=58%; range=45-65%; SD=9) during the baseline phase. During the training phase, no noticeable changes in response accuracy were observed for either items selected to be 1-trial/session ($y=2.5x+85$; $R^2=0.051$) or 4-trials/session items ($y=1.5x+90$; $R^2=0.0947$). By the end of the training phase (i.e., the last two training probes), A103 averaged 98% accuracy for both 1-trial/session items and 4-trials/session items. A slight difference was observed between 1- and 4-trials/session items during the maintenance phase; 1-trial/session items were responded to with 93% accuracy (range=85-100%; SD=8) and 4-trials/session items were responded to with 97% accuracy (range=95-100%; SD=3). Refer to figure 5.6 for A103's naming performance for 1 vs. 4 trials per session across experimental phases.

A104. Across the four baseline probes, A104 averaged 90% response accuracy for trained items (range=75-95%). During the baseline phase, items selected to be 1-trial/session (mean=94%; range=90-95%; SD=3) were responded to more accurately and more consistently than those selected to be 4-trials/session (mean=85%; range= 75-95%; SD=9). No observable changes were observed for response accuracy during the course of the training phase for either 1-trial/session items ($y=5357x+93.214$; $R^2=0.0989$) or 4-trial/session items ($y=-0.0595x+94.64$; $R^2=0.007$). As a result of the apparent instability of 4-trials/ession items, 4-trials/session items appeared more susceptible to immediate vs. delayed probes

than the 1-trial/session items until the fourth training probe. By the end of the training phase (i.e., the last two training probes), A104 averaged 100% accuracy for 1-trial/session items and 95% accuracy for 4-trials/session items. During the maintenance phase, A104 averaged 90% response accuracy for both 1-trial/session items (range=90-90%; SD=0) and 4-trials/session items (range=90-100%; SD=10). Her performance across the three maintenance probes was completely stable for the 1-trial/ session items, while her performance for the 4-trials/session items fluctuated across the three maintenance probes. Refer to figure 5.7 for A104's naming performance for 1 vs. 4 trials per session across experimental phases.

A106. Across the four baseline probes, A106 averaged 47% response accuracy for trained items (range=40-55%). During the baseline phase, a 10 percent difference in mean performance was observed between items selected to be 1-trial/session (mean=50%; range=45-55%; SD=6) and items selected to be 4-trials/session (mean=41%; range=40-50%; SD=45). This difference, however, appears to stem from a single 15 percent difference between 1-trial and 4-trials/session items at the second baseline probe. All other baseline probes range between a zero and five percent difference between 1-trial and 4-trials/session items. During the training phase, both 1-trial/session items ($y=3.4524x+43.214$; $R^2=0.4216$) and 4-trials/session ($y=2.5595x+34.107$; $R^2=0.4095$) items demonstrate a slight increase in response accuracy. By the end of the training phase (i.e., the last two training probes), A106 averaged 68% accuracy for 1-trial/session items and 53% accuracy for 4-trials/session items. During the maintenance phase, A106 averaged 65% response accuracy for 1-trial/session items (range=65-65%; SD=0) and 55% response accuracy for 4-trials/session items (range=50-60%; SD=5). Her performance across the three maintenance probes was completely stable for the 1-trial/ session items, while her performance for the 4-trials/session items fluctuated across the three maintenance probes. Refer to figure 5.8 for A106's naming performance for 1-trial/session vs. 4-trials/session items across experimental phases.

C102. As anticipated, C102 performed at ceiling levels for response accuracy (100%) for all phases of the experimental protocol. As such, her data will not be presented in this section of results.

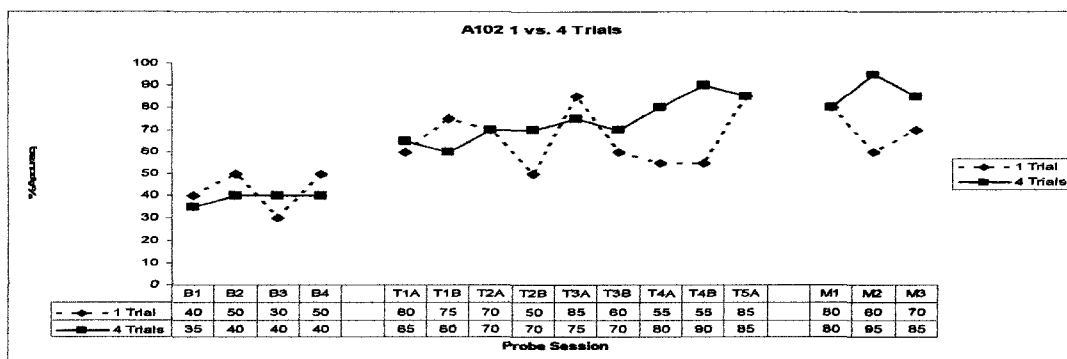


Figure 5.5 A102 Response Accuracy for 1- vs. 4-Trials/Session Items

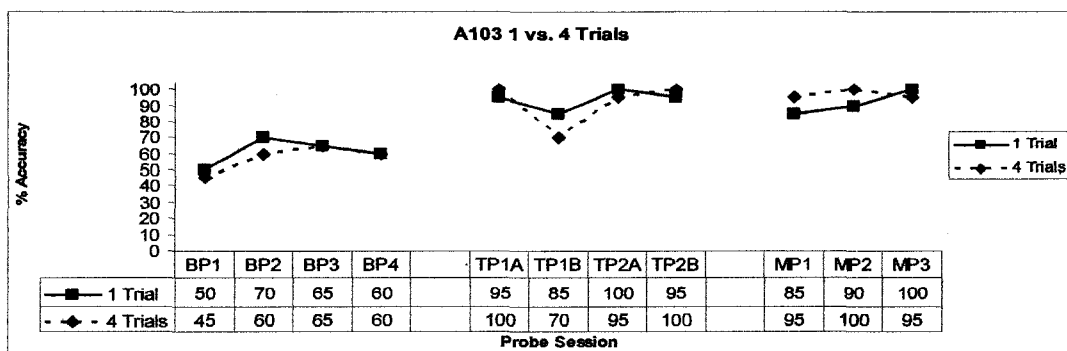


Figure 5.6 A103 Response Accuracy for 1- vs. 4-Trials/Session

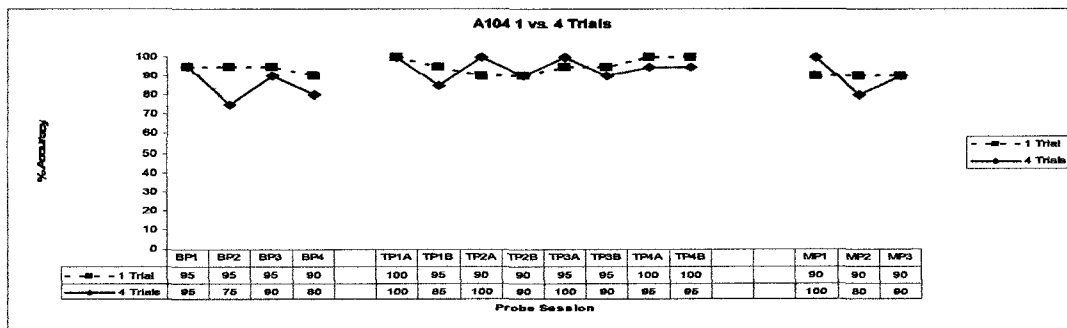


Figure 5.7 A104 Response Accuracy for 1- vs. 4-Trials/Session

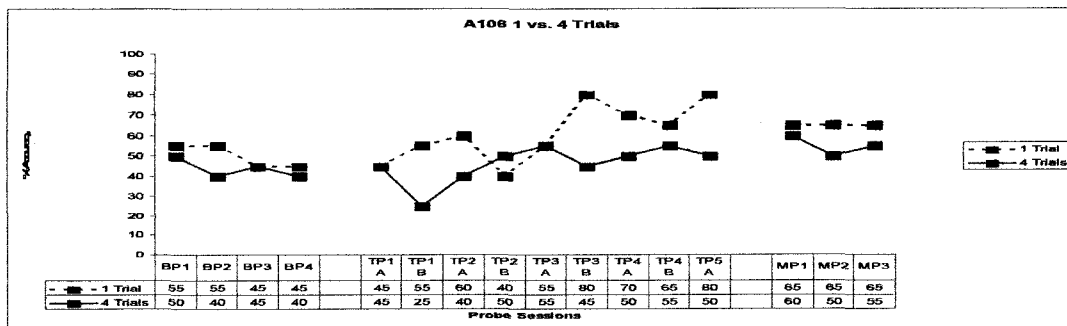


Figure 5.8 A106 Response Accuracy for 1- vs. 4-Trials/Session Items

Effect Sizes for Response Accuracy

To determine the amount of change in response accuracy observed as a result of the repetition priming protocol, effect sizes were calculated for trained and untrained items, and 1-trial/session and 4-trials/session for each participant. Busk and Serlin's d^{45} was used, which compares mean performance during the maintenance phase to the mean performance during the baseline phase, relative to the variance observed during the baseline phase. This effect size calculation assumes that the variance observed during baseline is the variance inherent to each participant prior to treatment (Beeson & Robey, 2008). Busk and Serlin's d does not take into consideration performance during the training phase. Traditional benchmarks for effect sizes in the sciences have been provided by Jacob Cohen (Cohen, 1969) as follows: small (0.20), medium (0.50), and large (0.80). Recently, aphasiologists Beeson and Robey synthesized data from treatment studies involving individuals with aphasia to provide benchmarks for effect sizes relative to single subject design studies investigating lexical retrieval (see table 5.4).

Table 5.4 Benchmarks for Effect Sizes Relative to Aphasia Research

	Small	Medium	Large
Lexical Retrieval	6.5	8.0	9.5

These benchmarks will be used as a reference point for upcoming discussions about the effect sizes calculated for the current investigation. In an earlier study, Robey found that spontaneous recovery produces an average effect size of 0.6 (Robey, 1998). From a theoretical standpoint, however, repetition priming is not expected to produce effect sizes as large as those produced by impairment-based, linguistically-motivated treatments designed to improve spoken language production for individuals with aphasia. That is, this repetition priming protocol was not designed to be a treatment study; instead, the current investigation sought to observe the learning behavior of individuals with aphasia in the context of repetition priming and to determine how stimulus dosage influences such priming in individuals with aphasia. Refer to tables 5.5-5.8 for a summary of the effect sizes

⁴⁵ Busk & Serlin's $d = \text{mean}(\text{post-treatment}) - \text{mean}(\text{pre-treatment}) / \text{standard deviation}(\text{pre-treatment})$

calculated for each participant in regards to response accuracy as influenced by stimulus dosage (i.e., trained, untrained, 1-trial/session, 4-trials/session).

Table 5.5 Effect Sizes for Response Accuracy of Trained Items

	A102	A103	A104	A106
Trained	7.30	4.19	0.12	2.31
Direction of Effect Size	Positive	Positive	Positive	Positive
Size Relative to Benchmark	Small-Medium	Small	No change	Small

Table 5.6 Effect Sizes for Response Accuracy of Untrained Items

	A102	A103	A104	A106
Untrained	-0.13	-0.22	-0.92	0.38
Direction of Effect Size	Negative	Negative	Negative	Positive
Size Relative to Benchmark	No change	No change	No change	No change

Table 5.7 Effect Sizes for Response Accuracy of 1-Trial/Session Items

	A102	A103	A104	A106
1 Trial/Session	2.89	3.54	-1.5	1.83
Direction of Effect Size	Positive	Positive	Negative	Positive
Size Relative to Benchmark	Small	Small	Small	Small

Table 5.8 Effect Sizes for Response Accuracy of 4-Trials/Session Items

	A102	A103	A104	A106
4 Trials/Session	19.1	4.53	0.56	2.34
Direction of Effect Size	Positive	Positive	Positive	Positive
Size Relative to Benchmark	Large	Small	No Change	Small

Stimulus Generalization

Descriptive Statistics

Stimulus generalization probes were administered immediately following each training and maintenance probe to document the effects of repetition priming on naming response accuracy of alternate exemplars of the trained items. Descriptive statistics including means and standard deviations for each stimulus generalization probe were calculated for each participant across the training and maintenance phases of the experimental protocol for 1-trial/session and 4-trials/session items relative to response accuracy. Alternate exemplars were not probed during the baseline phase of the protocol. Please refer to table 5.9 for a summary of these means and standard deviations. The control participant responded to 95-100% of the alternate exemplars accurately. As such, standard deviations are not provided for her data.

Table 5.9. Mean Response Accuracy for Stimulus Generalization Probes

		A102	A103	A104	A106	CONTROL
Training Phase	1 Trial	60%(19)	88%(18)	95%(6)	47% (17)	100% (0)
	4 Trials	50%(17)	84%(4)	91%(7)	41% (15)	93% (3)
Maintenance Phase	1 Trial	56%(9)	93%(6)	83%(8)	59% (10)	100% (0)
	4 Trials	59%(16)	87%(11)	90%(17)	51% (12)	97% (3)

Visual Analysis

Line graphs were produced for each participant to depict response accuracy across training and maintenance phases of the experimental protocol for 1-trial and 4-trials/session items (see figures 5.9-5.12). A line graph was not produced for the control participant as she was at near-ceiling performance in terms of response accuracy for the entire protocol. Consistent effects of repetition priming on generalization to alternate exemplars were not observed for any of the participants.

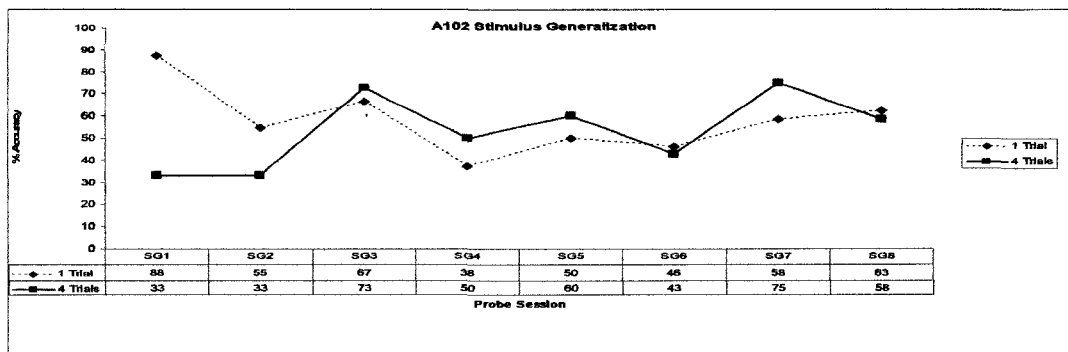


Figure 5.9 A102 Response Accuracy for Stimulus Generalization

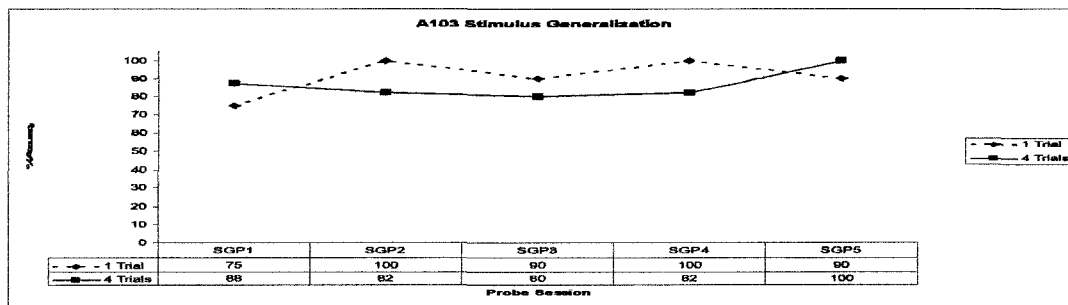


Figure 5.10 A103 Response Accuracy for Stimulus Generalization

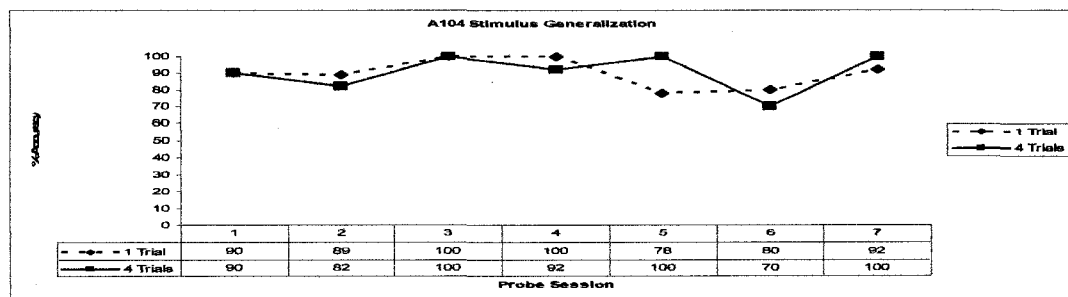


Figure 5.11 A104 Response Accuracy for Stimulus Generalization

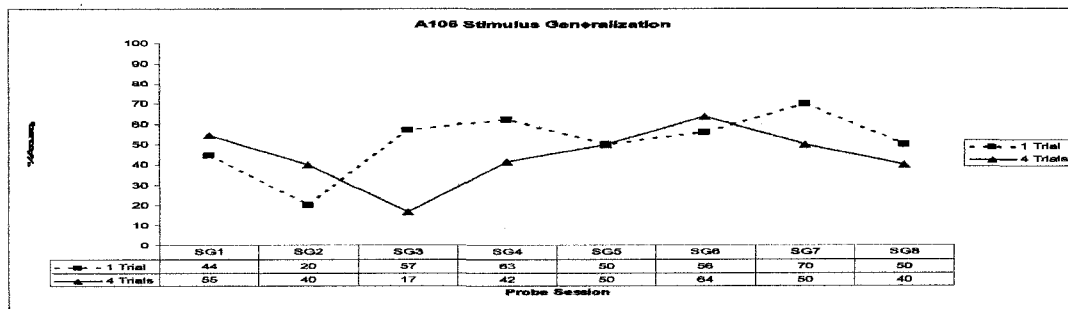


Figure 5.12 A106 Response Accuracy for Stimulus Generalization

RESPONSE TIME/REACTION TIME DATA

Response time data for individuals with aphasia is provided below, with some caveats. With the exception of A104, who began the protocol with at a near-ceiling level, a significant amount of data was trimmed prior to calculating descriptive statistics for each probe session. For each participant, erred responses were removed from the data set, along with outliers as described in earlier sections. As such, the response time data presented below often depicts only a handful of response time data points. Please see table 5.10 for a description of the amount of data trimmed for each participant for each probe. Reaction time data for the control participant is also presented below. Data reduction for the control participant was minimal, resulting from occasional technical difficulties; this data reduction is also presented in table 5.10.

Table 5.10 Response/Reaction Time Data Trimmed by Participant

	Baseline Probe Data Trimmed		Training Probe Data Trimmed		Maintenance Probe Data Trimmed		Stimulus Generalization Probe Data Trimmed		Total Data Trimmed	
	#	%	#	%	#	%	#	%	#	%
A102	177	74%	213	44%	75	42%	74	46%	539	51%
A103	120	50%	72	30%	44	24%	18	18%	254	33%
A104	42	18%	59	13%	33	18%	17	12%	151	15%
A106	138	58%	287	53%	85	47%	83	52%	593	53%
C102	3	1%	11	3%	4	2%	3	0.8%	21	2%

Trained vs. Untrained Items

Descriptive Statistics

Descriptive statistics including means and standard deviations were calculated for each participant across each phase of the experimental protocol for trained and untrained items relative to response/reaction time. Please refer to table 5.11 for a summary of means and standard deviations.

Table 5.11 Mean Response/Reaction Time (ms) for Trained vs. Untrained Items

		A102	A103	A104	A106	CONTROL
Baseline Phase	Untrained	1080 (143)	1996 (881)	1240 (200)	734 (191)	731 (197)
	Trained	1134 (70)	1797 (151)	1061 (65)	719 (180)	672 (148)
Training Phase	Untrained	1451 (193)	1888 (990)	1077 (145)	909 (430)	777 (200)
	Trained	1159 (127)	1878 (577)	1023 (64)	804 (289)	613 (142)
Maintenance Phase	Untrained	1092 (175)	1800 (68)	1144 (48)	1184 (516)	749 (174)
	Trained	1585 (77)	1887 (470)	1042 (43)	883 (404)	619 (169)

()=standard deviation

Visual Analysis

Line graphs were produced for each participant to depict response/reaction time across phases of the experimental protocol for trained and untrained item (see figures 5.13-5.17).

A102. Across the four baseline probes, A102 averaged 1107 ms (range=986-1244 ms; SD=105 ms). Upon visual inspection, small differences between trained (mean=1134 ms; range= 1053-1178 ms; SD=70 ms) and untrained items (mean=1080; range=986-1244; SD=143 ms) were observed during the baseline phase. During the training phase, A102 responded to trained items somewhat faster than untrained items. A102 demonstrated a moderate, but consistent decrease in response time for trained items ($y=-38.23x+1330.6$; $R^2=0.5469$) compared to untrained items ($y=-6.7077x+1481.3$; $R^2=0.0072$) as the training protocol progressed. By the end of the training protocol (i.e., the last two training probes), she responded to trained items approximately 116 ms faster than she did during the baseline phase. Untrained items were responded to much less consistently and no visual decrease was observed as the training protocol progressed. By the end of the training protocol she responded to untrained items 339 ms slower than she did during the baseline phase. During the maintenance phase, A102 responded to trained items (mean=1092 ms; range=1026-1176 ms; SD=77 ms) faster and more consistently than to untrained items (mean=1585 ms; range=1401-1749 ms; SD=175 ms). Furthermore, during the maintenance phase, she responded to trained items 42 ms faster than during the baseline phase; she responded to untrained

items 505 ms slower during the maintenance phase than she did during the baseline phase. Refer to figure 5.13 for a visual depiction of A102's response time across phases of the protocol. Note: response time probe data from the second baseline probe and first training probe were lost as a result of technical difficulties. Response time logging did not occur within E-Prime as a result of undetermined technical difficulties.

A103. Across the four baseline probes, A103 averaged 1897 ms (range=1350-3304 ms; SD=304 ms). Upon visual inspection, small differences between trained (mean=1797 ms; range=1690-2021 ms; SD=151 ms) and untrained items (mean=1996; range=1350-3304; SD=889 ms) were observed during the baseline phase. During the training phase, A103 responded similarly to both trained and untrained items. A large, consistent decrease of response time occurred for both trained items ($y=-422.66x+2934.5$; $R^2=0.8935$) and untrained items ($y=-759.97x+3787.4$; $R^2=0.9813$) as the training protocol progressed. By the end of the training protocol she responded to trained items 388 ms faster than during the baseline phase; she responded to untrained items 904 ms faster than during the baseline phase. By the end of the training phase (i.e., the last two training probes), A103 responded to untrained items (mean=1092 ms) faster than to trained items (mean=1409 ms). During the maintenance phase, A103 responded to trained items (mean=1800 ms; range=1386-2311) ms; SD=470 ms) faster but less consistently than she did to untrained items (mean=1887 ms; range=1815-1951 ms; SD=175 ms). Furthermore, during the maintenance phase, she responded to trained items 3 ms slower than during the baseline phase; she responded to untrained items 109 ms faster during the maintenance phase than she did during the baseline phase. Refer to figure 5.14 for a visual depiction of A103's response time across phases of the protocol.

A104. Across the four baseline probes, A104 averaged 1151 ms (range=982-1459 ms; SD=156 ms). Upon visual inspection, large differences between trained (mean=1061 ms; range=982-1140 ms; SD=65 ms) and untrained items (mean=1240; range=987-1459; SD=200 ms) were observed during the baseline phase beginning with the second baseline probe. During the training phase, A104 responded more consistently and somewhat faster for trained items than she did for

untrained items. Neither trained ($y=3.3302x+1008.1$; $R^2=0.0164$) nor untrained items ($y=-6.6996x+1107.3$; $R^2=0.0128$) demonstrated a trend in terms of a decrease or increase in response time as the training protocol progressed. By the end of the training protocol A104 responded to trained items 76 ms faster than during the baseline phase; she responded to untrained items 141 ms faster than during the baseline phase. By the end of the training phase (i.e., the last two training probes), A104 responded to trained items (mean=1041 ms) faster than to untrained items (mean=1118 ms). During the maintenance phase, A104 responded to trained items (mean=1042 ms; range=1010-1090) ms; SD=43 ms) faster than she did to untrained items (mean=1145 ms; range=1102-1196 ms; SD=48 ms). Furthermore, during the maintenance phase, she responded to trained items 19 ms faster than during the baseline phase; she responded to untrained items 95 ms faster during the maintenance phase than she did during the baseline phase. Refer to figure 5.15 for a visual depiction of A104's response time across phases of the protocol.

A106. Across the four baseline probes, A106 averaged 727 ms (range=660-840 ms; SD=59 ms). Visual inspection indicates a very stable baseline phase with no observable differences between trained items (mean=719 ms; range=664-759 ms; SD=43 ms) and untrained items (mean=734 ms; range=664-840 ms; SD=75 ms). During the training phase, A106 responded more consistently and much faster for trained items than she did for untrained items. Both trained ($y=-24.702x+888.43$; $R^2=0.3972$) and untrained items ($y=-51.188x+1373.4$; $R^2=0.4028$) demonstrated a small decrease in response time as the training protocol progressed; however, untrained items were responded to much slower than baseline performance at the start of the training phase. By the end of the training protocol A106 responded to trained items 26 ms faster than during the baseline phase; she responded to untrained items 222 ms slower by the end of the training phase than during the baseline phase. By the end of the training phase (i.e., the last two training probes), A106 responded to trained items (mean=912 ms) faster than to untrained items (mean=1902 ms). A106 responded to trained items 164 ms slower during the maintenance phase than during the baseline phase; she responded to untrained items 450 ms slower during the maintenance phase than she did during the baseline

phase. Refer to figure 5.16 for a visual depiction of A104's response time across phases of the protocol.

C102. Across the four baseline probes, the control averaged 702 ms (range=637-795 ms; SD=173 ms). Visual inspection indicates a stable baseline phase with observable differences between trained items (mean=672 ms; range=637-719 ms; SD=148 ms) and untrained items (mean=731 ms; range=683-795 ms; SD=197 ms). Both trained items ($y=-13.677x+660.04$; $R^2=0.4908$) and untrained items ($y=-31.349x+874.92$; $R^2=0.6209$) demonstrated a small to moderate decrease in reaction time over the course of the four baseline probes. During the training phase, C102 responded more consistently and much faster for trained items than she did for untrained items. Both trained and untrained items demonstrated a decrease in reaction time as the training protocol progressed, although untrained items were responded to much slower than baseline performance at the start of the training phase. By the end of the training protocol C102 responded to trained items 59 ms faster than during the baseline phase; she responded to untrained items 46 ms faster by the end of the training phase than during the baseline phase. C102 responded to trained items 53 ms faster during the maintenance phase than during the baseline phase; she responded to untrained items 18 ms faster during the maintenance phase than she did during the baseline phase. Refer to figure 5.17 for a visual depiction of C102's response time across phases of the protocol.

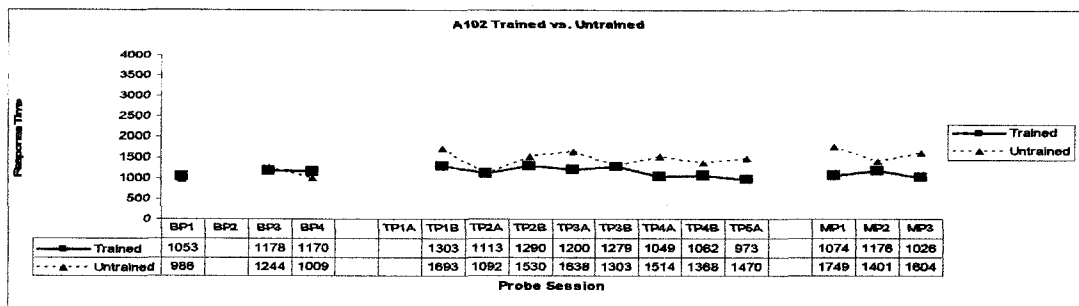


Figure 5.13 A102 Response Time for Trained vs. Untrained Items

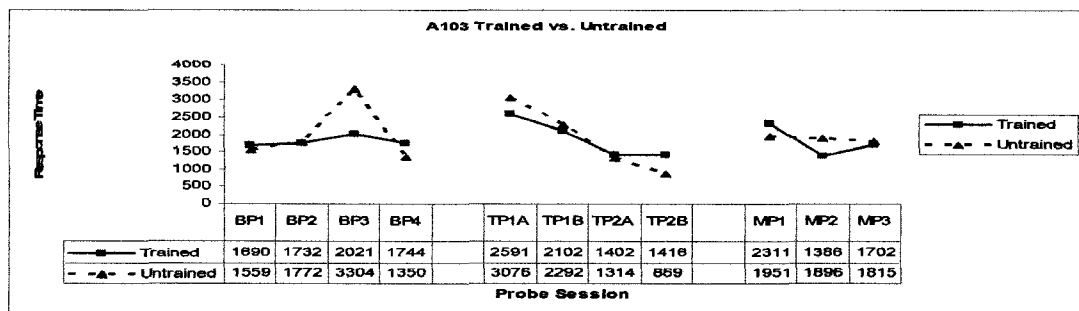


Figure 5.14 A103 Response Time for Trained vs. Untrained Items

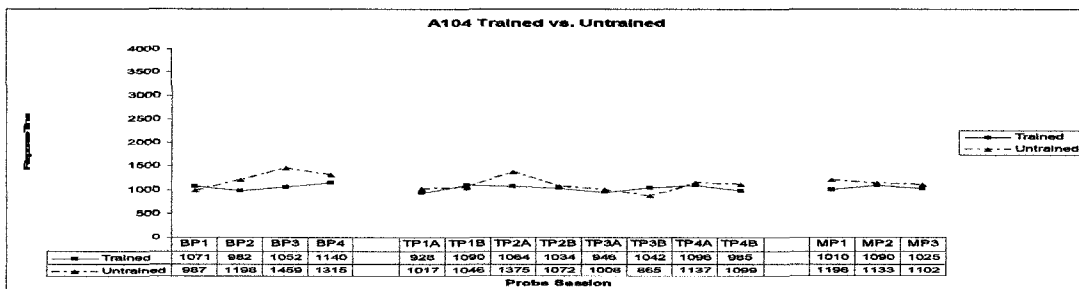


Figure 5.15 A104 Response Time for Trained vs. Untrained Items

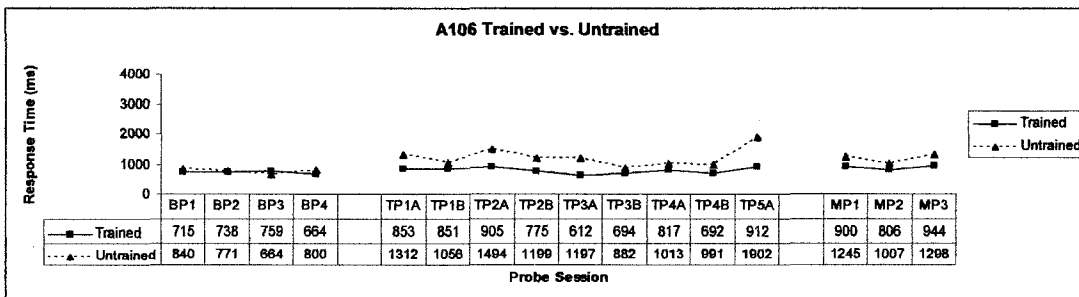


Figure 5.16 A106 Response Time for Trained vs. Untrained Items

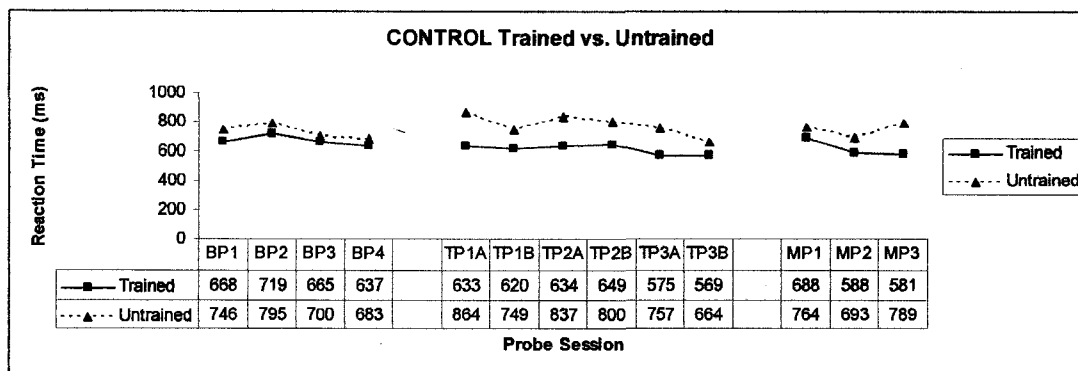


Figure 5.17 C102 Reaction Time for Trained vs. Untrained Items

1-Trial/Session vs. 4-Trials/Session

Descriptive Statistics

Descriptive statistics including means and standard deviations were calculated for each participant across each phase of the experimental protocol for 1- and 4-trials/session items relative to response/reaction time. Please refer to table 5.12 for a summary of these means and standard deviations.

Table 5.12 Mean Response/Reaction Time (ms) for 1- vs. 4-Trials/Session Items

		A102	A103	A104	A106	CONTROL
Baseline Phase	1 Trial	1136 (45)	1850(282)	1050 (111)	698 (179)	698 (161)
	4 Trials	1102 (229)	1735(268)	1077 (35)	746 (189)	648 (122)
Training Phase	1 Trial	1194 (161)	1860 (692)	996 (97)	812 (240)	632 (177)
	4 Trials	1124 (132)	1902 (509)	1056 (101)	784 (309)	595 (125)
Maintenance Phase	1 Trial	1106 (122)	1765 (413)	1021 (38)	926 (340)	640 (197)
	4 Trials	1086 (53)	1806 (534)	1065 (62)	815 (289)	598 (131)

()=standard deviation

Visual Analysis

Line graphs were produced for each participant to depict response/reaction time across phases of the experimental protocol for 1-trial and 4-trials/session items (see figures 5.18-5.22).

A102. Across the four baseline probes, A102 averaged 1118 ms response time for trained items (range=838-1239 ms). Items selected to be 1-trial/session

(mean=1136; range=1111-1188 ms; SD=45 ms) appeared to be responded to more consistently than 4-trials/session (mean=1102 ms; range=838-1239; SD=229 ms) during the baseline phase. During the first part of the training phase, 4-trial/session items were responded to more quickly than 1-trial/session items; however, by the end of the training phase (i.e., the last two training probes), 1-trial (mean=1014 ms) and 4-trials/session (mean=1017 ms) items were responded to with nearly identical response times. Over the course of the training phase, 1-trial/session items demonstrated a slight decrease in response time ($y=-21.712x+1221.6$; $R^2=0.1637$) while 4-trials/session items demonstrated a moderate decrease in response time ($y=-54.696x+1440.7$; $R^2=0.6921$). During the maintenance phase, 4-trial/session items (mean=1086 ms; range=1048-1146 ms; SD=53 ms) were responded to slightly faster and more consistently than 1-trial/session items (mean=1106 ms; range=996-1237 ms; SD=122 ms). Refer to figure 5.18 for A102's response times for 1- vs. 4-trials/session across experimental phases. NOTE: response time data for the second baseline probe and the first training probe are missing as a result of undetermined technical difficulties.

A103. Across the four baseline probes, A103 averaged 1792 ms response time for trained items (range=1362-2082 ms; SD=262 ms). Items selected to be 1-trial/session (mean=1850; range=1487-2082 ms; SD=282 ms) were not responded to differently than items selected to be 4-trials/session (mean=1735 ms; range=1362-1960; SD=266 ms) during the baseline phase. After an initial increase in response time during the first part of the training phase, relative to baseline, both 1-trial/session items ($y=-484.76x+3071.6$; $R^2=0.8189$) and 4-trials/session items ($y=-359.37x+2800.4$; $R^2=0.8304$) demonstrated a large decrease in response time during the course of the training phase. By the end of the training phase (i.e., the last two training probes), 1-trial/session items (mean=1360 ms) were responded to slightly faster than 4-trials/session (mean=1462 ms); collapsing 1- and 4-trials/session items, a 381 ms decrease in response time was observed by the end of the training session relative to baseline. During the maintenance phase, both 1-trial/session items (mean=1786 ms; range=1375-2201 ms; SD=413 ms) and 4-trials/session items (mean=1806 ms; range=1396-2410 ms; SD=534 ms) were responded to in nearly identical manner a similar fashion; both 1- and 4-

trials/session items returned to baseline mean performance. Refer to figure 5.19 for A103's response times for 1- vs. 4-trials/session across experimental phases.

A104. Across the four baseline probes, A104 averaged 1063 ms response time for trained items (range=924-1173 ms; SD=73 ms). Items selected to be 1-trial/session (mean=1050; range=924-1173 ms; SD=111 ms) were responded to much less consistently than items selected to be 4-trials/session (mean=1077 ms; range=1038-1113; SD=35 ms) during the baseline phase. During the first half of the training phase, both 1-trial and 4-trials/session items were responded to similarly, with no observable change in response time relative to baseline. During the second half of the training phase, however, A104 responded much more quickly to 1-trial/session items relative to 4-trials/session items, and somewhat faster than 4-trials/session items during baseline. Overall, neither 1-trial/session items ($y = -2.8889x + 1008.7$; $R^2 = 0.0054$) nor 4-trials/session items ($y = 10.732x + 1007.4$; $R^2 = 0.0685$) demonstrated an observable change in response time during the course of the training phase. By the end of the training phase (i.e., the last two training probes), 1-trial/session items (mean=1029 ms) and 4-trials/session (mean=1055 ms) were responded to in a similar fashion; collapsing 1- and 4-trials/session items, a 22 ms decrease in response time was observed by the end of the training session relative to baseline. During the maintenance phase, both 1-trial/session items (mean=1021 ms; range=983-1058 ms; SD=38 ms) and 4-trials/session items (mean=1065 ms; range=1001-1125 ms; SD=62 ms) were responded to in nearly identical manner a similar fashion; both 1- and 4-trials/session items returned to baseline mean performance during the maintenance phase. Refer to figure 5.20 for A104's response times for 1- vs. 4-trials/session across experimental phases.

A106. Across the four baseline probes, A106 averaged 722 ms response time for trained items (range=656-774 ms; SD=50 ms). Items selected to be 1-trial/session (mean=698; range=656-744 ms; SD=39 ms) were responded to slightly faster and more consistently than items selected to be 4-trials/session (mean=746 ms; range=664-774; SD=53 ms) during the baseline phase. A downward trend (i.e., decreased response time) was observed for the last baseline probe for both 1- and 4-trials/session items. During the first half of the training phase, both 1-trial and 4-trials/session items were responded to more slowly than they were during the

baseline phase, with 4-trials/session items demonstrating this effect to a greater degree. During the second half of the training phase, both 1- and 4-trials/session items returned to baseline mean response times, with 4-trials/session items (mean=784 ms) being responded to slightly faster than 1-trial/session items (mean=812 ms). Overall, both 1-trial/session items ($y=-25.455x+872.04$; $R^2=0.326$) and 4-trials/session items ($y=-21.942x+893.83$; $R^2=0.2063$) demonstrated a minimal decrease in response time over the course of the training phase. By the end of the training phase (i.e., the last two training probes), 1-trial/session items (mean=758 ms) and 4-trials/session (mean=749 ms) were responded to in a similar fashion; collapsing 1- and 4-trials/session items for these last two probe sessions, a 32 ms increase in response time was observed by the end of the training session relative to baseline. A106's response time increased well above baseline performance for both 1-trial/session items (mean=926 ms; SD=340 ms) and 4-trials/session items (mean=815 ms; SD=289 ms) during the maintenance phase. Refer to figure 5.21 for A106's response times for 1- vs. 4-trials/session across experimental phases.

C102. Across the four baseline probes, the control participant averaged 673 ms for trained items (range=615-766 ms; SD=49 ms). Baseline performance was relatively stable, with an observable difference in reaction time between 1-trial/session items (mean=698 ms; range=631-766 ms; SD=161 ms) and 4-trials/session items (mean=648 ms; range=615-658 ms; SD=122 ms); 4-trials/session items were responded to more quickly and more consistently than 1-trial/session items during the baseline phase. During the training phase, C102 demonstrated a small decrease in reaction time for both 1-trial/session items ($y=-19.356x+699.31$; $R^2=0.4572$) and 4-trials/session items ($y=-8.1x+621.18$; $R^2=0.3605$) relative to the baseline phase. By the end of the training phase, C102 responded to 4-trials/session items 37 milliseconds faster than 1-trial/session items trained items. Participant C102 named 1-trial/session pictures 66 ms faster during training than during the baseline phase; she named 4-trials/session pictures 53 ms faster during training than during the baseline phase. These same decreases in reaction time persisted during the maintenance phase; C102 named 1-trial/session items 58 ms faster than during baseline and named 4-trials/session items 50 ms

faster during maintenance than during baseline. Refer to figure 5.22 for C102's reaction time for 1- vs. 4-trials/session across experimental phases.

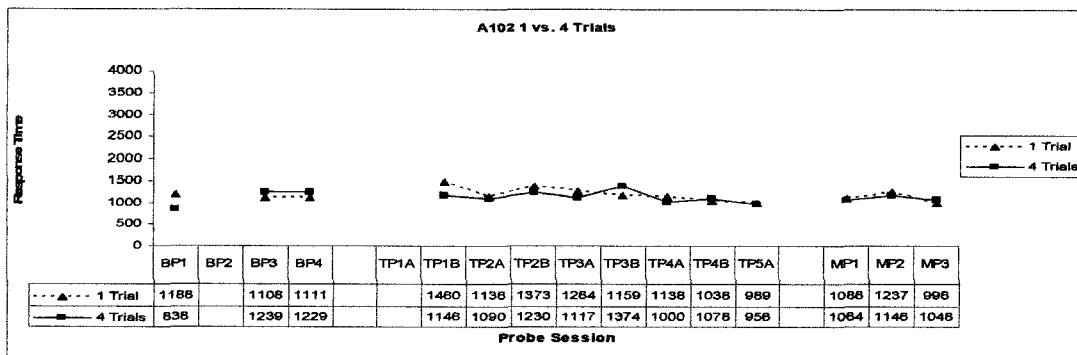


Figure 5.18 A102 Response Time for 1- vs. 4-Trials/Session Items

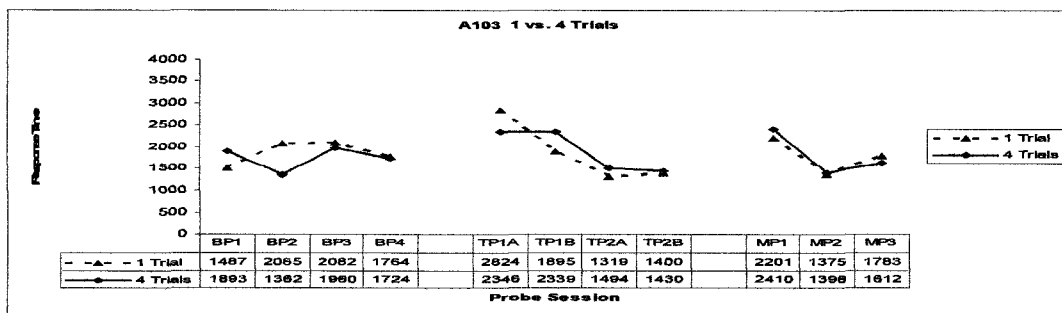


Figure 5.19 A103 Response Time for 1- vs. 4-Trials/Session Items

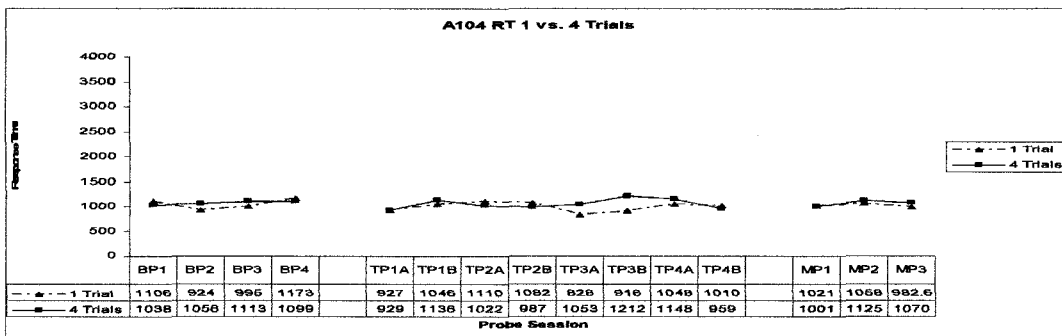


Figure 5.20 A104 Response Time for 1- vs. 4-Trials/Session

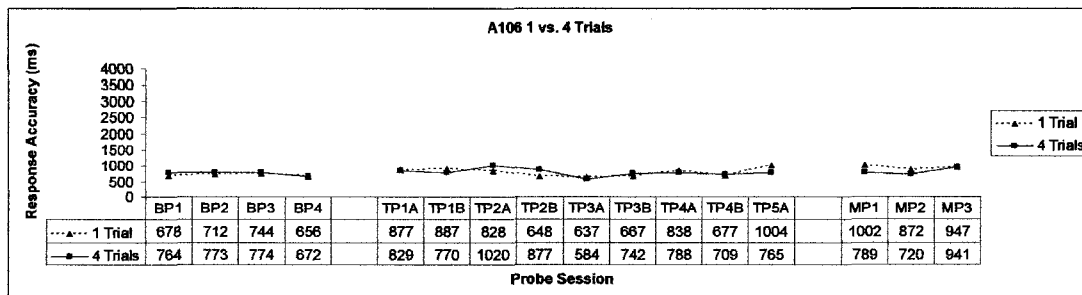


Figure 5.21 A106 Response Time for 1- vs. 4-Trials/Session Items

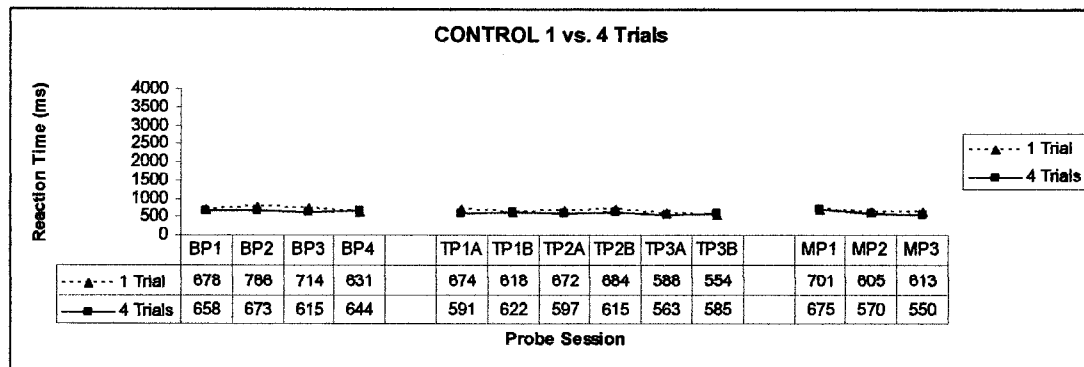


Figure 5.22 C102 Reaction Time for 1- vs. 4-Trials/Session Items

Effect Sizes for Response/Reaction Time

To determine the amount of change in response latency observed as a result of the repetition priming protocol, effect sizes were calculated for trained and untrained items, and 1-trial/session and 4-trials/session for individuals with aphasia and the age- and gender-matched control participant. Busk and Serlin's d^{46} was used to calculate the effect sizes. Refer to tables 5.13-5.16 for effect sizes calculated for each participant relative to response/reaction time for trained items, untrained items, 1-trial/session items, and 4-trials/session items.

Table 5.13 Effect Sizes for Response/Reaction of Trained Items

	A102	A103	A104	A106	Control
Trained	-0.596	0.02	-0.29	4.02	-1.55
Direction of Effect Size	Positive	Negative	Positive	Negative	Positive
Size Relative to Benchmark	No change	No change	No change	Small	Small

* Note: negative values indicate decreased response time; positive values indicate increased response time

Table 5.14 Effect Sizes for Response/Reaction Time of Untrained Items

	A102	A103	A104	A106	Control
Untrained	3.537	-0.12	-0.48	5.50	0.36
Direction of Effect Size	Negative	Positive	Positive	Negative	Negative
Size Relative to Benchmark	Small	No change	No change	Small	No change

⁴⁶ Busk & Serlin's $d = \text{mean}(\text{post-treatment}) - \text{mean}(\text{pre-treatment}) / \text{standard deviation}(\text{pre-treatment})$

Table 5.15 Effect Sizes for Response/Reaction Time of 1-Trial/Session Items

	A102	A103	A104	A106	Control
1 Trial/Session	-0.65	-0.22	-0.26	6.28	-0.99
Direction of Effect Size	Positive	Positive	Positive	Negative	Positive
Size Relative to Benchmark	No change	No change	No change	Small	No change

Table 5.16 Effect Sizes for Response/Reaction Time of 4-Trials/Session Items

	A102	A103	A104	A106	Control
4 Trials/Session	-0.070	0.25	-0.34	1.44	-1.95
Direction of Effect Size	Positive	Negative	Positive	Negative	Positive
Size Relative to Benchmark	No change	No change	No change	Small	Small

Stimulus Generalization

Descriptive Statistics

Stimulus generalization probes were administered immediately following each training and maintenance probe to observe the effects of repetition priming on response/reaction time of alternate exemplars of the trained items. Descriptive statistics including means and standard deviations for each stimulus generalization probe were calculated for each participant across the training and maintenance phases of the experimental protocol for 1-trial/session and 4-trials/session items relative to response/reaction time. Alternate exemplars were not probed during the baseline phase of the protocol. Please refer to table 5.17 for a summary of these means.

Table 5.17 Mean Response/Reaction Time (ms) for Stimulus Generalization

		A102	A103	A104	A106	CONTROL
Training Phase	1 Trial	1784	1763	1130	1140	676
	4 Trials	1680	1945	1212	1162	655
Maintenance Phase	1 Trial	1613	2353	1202	1085	615
	4 Trials	1254	2005	1235	980	602

Visual Analysis

Line graphs were produced for each participant to depict response/reaction time for alternate exemplars across training and maintenance phases of the experimental protocol for 1-trial and 4-trials/session items (see figures 5.23-5.27).

Consistent effects of repetition priming on generalization to alternate exemplars were not observed for individuals with aphasia in terms of response time. The control participant demonstrated a nearly 200 ms decrease in reaction time from the first alternate exemplar probe to the second probe. A plateau was then observed from the second to the fourth probe. An additional 56 ms decrease in reaction time was observed for 4-trials/session times at the last maintenance probe for the control participant. This decrease was not observed for the 1-trial/session items.

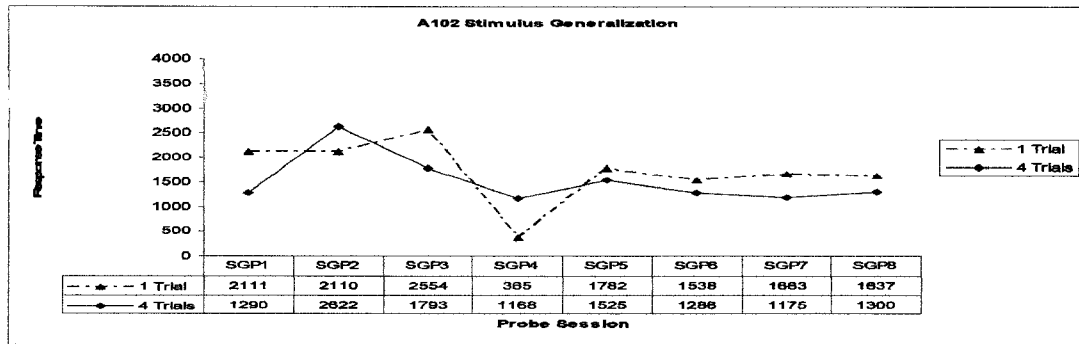


Figure 5.23 A102 Response Time for Stimulus Generalization

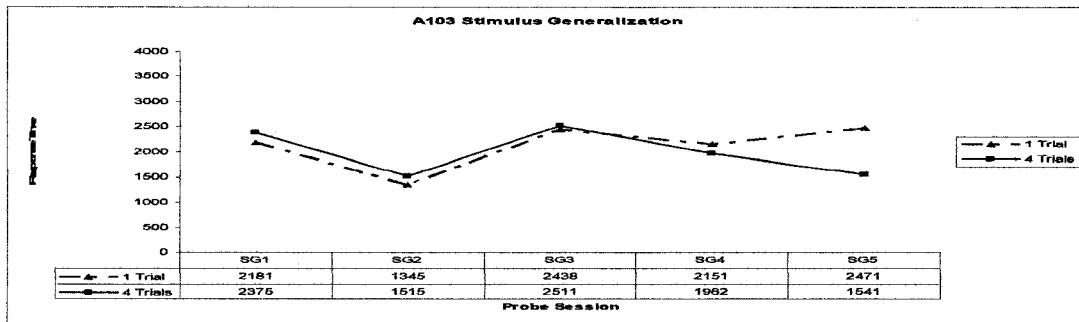


Figure 5.24 A103 Response Time for Stimulus Generalization

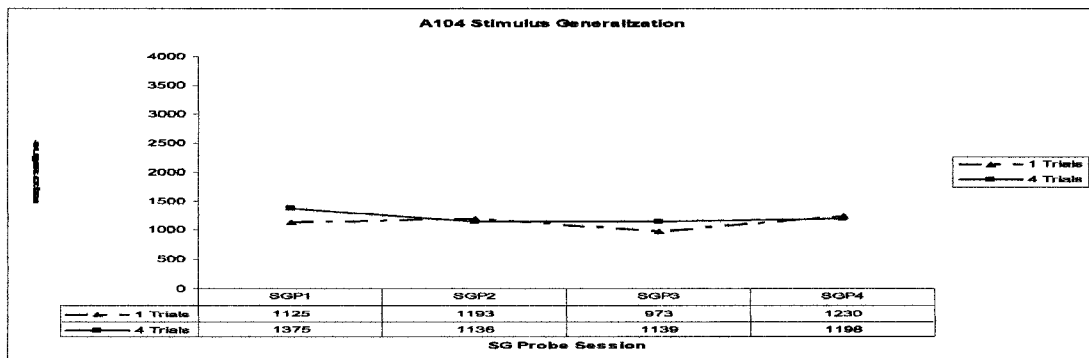


Figure 5.25 A104 Response Time for Stimulus Generalization

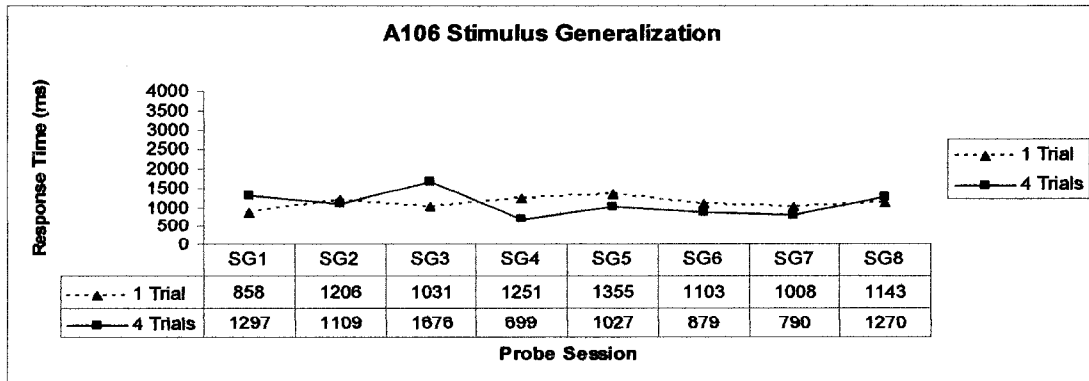


Figure 5.26 A106 Response Time for Stimulus Generalization

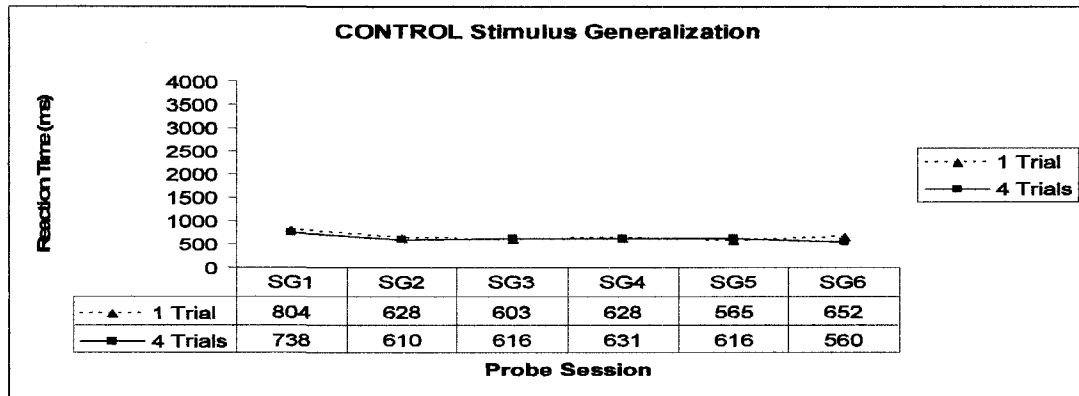


Figure 5.27 C102 Reaction Time for Stimulus Generalization

LEXICAL VARIABLES

As anticipated, high frequency words were generally responded to more accurately and faster than low frequency words and 1-syllable words were generally responded to more accurately and faster than 2-syllable words across participants. No systematic interactions were observed between repetition priming/stimulus dosage and word frequency or syllable length. Meaningful information about word frequency and syllable length is likely to emerge as data are collapsed across participants and analyzed as group data.

CHAPTER VI: DISCUSSION & FUTURE STUDIES

The primary goal of this study was to document the nature of repetition priming in individuals with aphasia with respect to response accuracy and response time of picture-naming. Results of this single-subject study indicate that repetition priming positively influences response accuracy and, less consistently, response time during picture-naming in individuals with aphasia. Consistent with the literature base, repetition priming was shown to positively influence reaction time for the gender-matched non-brain-injured control participant.

Specifically, this study addressed the following questions about the nature of repetition priming in individuals with aphasia:

- (1) Is repetition priming persistent across time?
- (2) Is repetition priming sensitive to the number of trials/session?
- (3) Is repetition priming item specific?

Prior to answering these questions directly, a brief summary of the results will be provided.

SUMMARY OF RESULTS – RESPONSE ACCURACY

Training Phase Relative to Baseline Phase

Based upon results presented in the previous section, three of the four individuals with aphasia (A102, A103, and A106) demonstrated an increased ability to accurately name pictures trained in the context of a repetition priming protocol relative to baseline naming performance. Participant A104's baseline performance was at near ceiling to ceiling levels; as such, there was little room for improvement. The control participant performed at ceiling to near-ceiling levels throughout the protocol. The three individuals with aphasia who improved relative to baseline did not share similar underlying linguistic impairments. Participant A102's anomia was characterized primarily by a phonological encoding impairment; A103's anomia was characterized primarily by a semantic encoding impairment; and A106's anomia was characterized primarily by a severe phonological encoding impairment likely to stem from conduction aphasia. Dell and colleagues have proposed that repetition priming influences phonological encoding, not semantic encoding (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997); yet, participant A103 did not demonstrate phonological

errors and was known to have aphasia stemming from a large left temporal lobe lesion, not a left frontal lesion. Given that this observation only stems from one participant, additional research is needed to replicate the influence of repetition priming on response accuracy in individuals with semantic encoding impairments. One possibility for the improvement observed with A103 is that during the training phase of the repetition priming protocol, participants are provided the orthographic name in conjunction with the picture. It is likely that A103 used the repetition priming protocol to learn the association between the orthographic name and the picture name; she likely used the learned orthographic representation as an alternate route for phonological encoding required for production purposes. Although there was little room for participant A104 to improve, her response accuracy for trained items was much more consistent during the training phase than untrained items, reflecting a positive influence of repetition priming on response variability.

Training Phase - Acquisition

Although three of the four individuals with aphasia demonstrated improved response accuracy during the training phase relative to the baseline phase for trained items, the onset and rate of improved response accuracy varied from participant to participant. Two of the three individuals with aphasia who demonstrated increased response accuracy as a result of repetition priming (A102, A103) did so after the first set of three training sessions; that is, for these two participants, the onset of increased response accuracy was observed at the first training probe (i.e., immediate acquisition). Participant A106, however, required significantly more training (9 training sessions) to elicit response accuracy above baseline performance. This lag in response to repetition priming may stem from the participant's underlying impairment; participant A106 was the only participant with conduction aphasia. Anecdotally, she demonstrated the largest number of errors during the training sessions and experienced more difficulty with perseveration than any of the other participants. Despite her inherent difficulties with repetition tasks, the repetition priming protocol eventually lead to an increased ability to name trained

items, suggesting that thresholds for the onset of repetition priming may differ as a result of underlying impairment.

The magnitude and consistency of improved response accuracy also varied across participants. Participant A102 demonstrated a steady and relatively consistent increase in response accuracy across the entire training phase. It is likely that she would have continued toward ceiling levels with additional training sessions. Participant A103, on the other hand, demonstrated an initial near-ceiling improvement of response accuracy (98% correct) after the first set of three training sessions and then her performance reached what appears to be a plateau. She only attended a total of six training sessions; as such further evidence of a plateau is not available. As mentioned in the previous paragraph, A106 did not demonstrate any noticeable gains until the third training probe session. At this time, she experienced a 10-15% increase in response accuracy. This rate of performance was stable for the following probe session and then she experienced another small gain of approximately five percent. Unlike healthy adults, repetition priming appears to continue to influence response accuracy across many sessions and many trials for individuals with aphasia.

SUMMARY OF RESULTS – RESPONSE TIME/REACTION TIME

Production tasks are inherently difficult for individuals with anomia, thus leading to frequent naming errors that must be removed from the data set before calculating descriptive statistics for response time. Furthermore, individuals with anomia frequently produce multiple false starts during naming tasks, leading to additional tokens that must be removed from the data set before analyzing response time. The result of so much data trimming is that few data points remain for calculating summary statistics. One possible methodological way around this unfortunate loss of data would be to use a software package that would allow tracking and tagging of response time continuously through naming attempts. Such a method will be investigated for future studies examining response time in individuals with aphasia.

Training Phase Relative to Baseline Phase

Based upon the results presented in the previous section, all four individuals with aphasia and the single gender-matched healthy control participant responded positively in terms of response/reaction time to the repetition priming protocol relative to baseline performance. That is, all participants demonstrated an observable decrease in response/reaction time in response to the repetition priming protocol. This decreased response time was observed even for the participant (A104) who did not demonstrate observable differences in response accuracy as a result of her near-ceiling performance during the baseline phase. As mentioned in the previous section, the fact that repetition priming has been demonstrated to occur across a variety of underlying impairments is interesting given Dell and colleagues' hypothesis that repetition priming effects stem from the stage of phonological encoding (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997).

Training Phase – Acquisition

Although all participants demonstrated a decrease in response/reaction time relative to baseline performance for trained items, the onset and rate of repetition priming varied across participants. Research involving repetition priming of control participants has documented noticeable decreases in reaction time after the first repetition (Poldrack, Wagner, Prull et al., 1999; Reber, Gitelman, Parrish, & Mesulam, 2004). This pattern was replicated in this study for the age-matched control; initial decreases in reaction time were observed during the baseline probe as a result of repeated instances of the "to-be-trained" items. Such early repetition priming effects on reaction time for the healthy control participant were expected; the methodological decision to have participants attempt to name the same items multiple times during the baseline phase stemmed from the desire to document the variability that is inherent within individuals with aphasia. Individuals with aphasia, as expected and previously demonstrated in the pilot study, did not demonstrate reliable early repetition priming effects on response time. Participant A102 did not demonstrate an onset of repetition priming in terms of response time until the fourth training probe (i.e., after 12 training sessions). Participant A103 demonstrated an onset of repetition priming in terms of response time after the second training probe

(i.e., after six training sessions). Participant A104 demonstrated an onset of repetition priming in terms of response time (albeit a very slight positive response) after the first training probe (i.e., after three training sessions). Finally, participant A106 did not demonstrate an onset of repetition priming until the third training probe (i.e., after nine training sessions).

The magnitude and consistency of repetition priming effects on response/reaction time also varied across participants. The healthy control participant demonstrated an early decrease of approximately 30 ms for trained items and then reached a plateau until the third training probe (i.e., after nine training sessions). At this time, participant C102 responded an additional 50 ms faster for trained items relative to baseline performance. Participant A102 demonstrated observable repetition priming effects (i.e., a 100 ms decrease in response time) by the fourth training probe and then she reached a plateau. Participant A103 demonstrated observable repetition priming effects (i.e., a 300 ms decrease in response time) by the second training probe. Further evaluation of her performance is impossible as she terminated the training protocol at this time. Participant A104's response to repetition priming in terms of response time was small and occurred after the initial set of training sessions. She demonstrated further decreases in response time at the third training probe, and then her response times returned to baseline performance. Finally, participant A106 demonstrated only one instance of decreased response time after the ninth training session. Her response time then returned to baseline performance. Response times for individuals with aphasia are highly variable; this inconsistent data is likely to stem from this variability, although it is possible that some of the larger fluctuations in response time may stem from an accuracy-for-response time trade-off.

PERSISTENCE OF REPTITION PRIMING

All individuals with aphasia demonstrated persistence of repetition priming in terms of response accuracy for trained items after training had been withdrawn for six weeks. Although this persistence is observable by visual inspection on line graphs for each individual with aphasia, the most compelling data are the effect sizes that directly compare maintenance performance to baseline performance while

adjusting for the variance inherent to each participant. Effect sizes for response accuracy ranged from 0.12 (no change) to 7.30 (medium change). Participant A102 demonstrated the largest effect size for trained items ($d=7.30$) which, according to Beeson and Robey's benchmarks reflects a small to medium effect of repetition priming. Participant A103 demonstrated a small effect size ($d=4.19$). Participant A104, who was at near ceiling levels in regards to response accuracy during baseline, did not demonstrate a significant effect size ($d=0.12$). Participant A106 demonstrated a small effect size ($d=2.31$). Untrained items did not follow this pattern. Three participants (A102, A103, A104) demonstrated decreased response accuracy for untrained items during the maintenance phase relative to the baseline phase. A106 demonstrated a very slight ($d=0.38$) increase in response accuracy for trained items during the maintenance phase relative to baseline. These positive effect sizes for trained items are relatively surprising given that the benchmark for effect sizes is based upon treatment studies addressing lexical retrieval in individuals with aphasia (Beeson & Robey, 2008); repetition priming alone (i.e., in the absence of a treatment protocol that is designed to target the underlying impairment) was not expected to lead to effect sizes as large as those observed in treatment studies.

Individuals with aphasia did not demonstrate persistent effects of repetition priming relative to the dependent measure of response time. The control participant demonstrated a persistent but small effect size for trained items during maintenance relative to baseline ($d=-1.55$); this decrease in response time was not observed for untrained items ($d=0.36$).

In order to meet current concerns in the field of aphasiology about the long-term persistence of treatment effects, future repetition priming protocols should follow-up with individuals with aphasia at intervals greater than six weeks (e.g., six months, one year, two years) to determine how long priming effects persist.

In addition to long-lasting persistence, short term persistence of priming effects was assessed by comparing immediate training probes (i.e., immediately following a training session) to delayed training probes (i.e., immediately preceding the subsequent training session). Consistent patterns of immediacy were not observed across participants. However, all participants did, at some point during the

training phase, demonstrate sensitivity to the immediacy of the training probe for both response accuracy (ranging from 5-20%) and response time (ranging from 100-200 ms for individuals with aphasia). The control participant demonstrated sensitivity to immediacy on the order of 10-15 ms.

SENSITIVITY TO THE NUMBER OF TRIALS PER SESSION

Upon visual inspection, the number of trials per session (i.e., stimulus dosage) did not consistently influence naming response accuracy during the training phase of the repetition priming protocol. Four-trials/session items revealed small to large positive changes in response time for two of the individuals with aphasia (A102 and A106). One-trial/session items produced a small positive change in response time for one participant, A106. However, a more consistent pattern emerged when the maintenance phase of the protocol was taken into consideration. All four of the individuals with aphasia demonstrated positive changes in terms of response accuracy for 4-trials/session items during the maintenance phase relative to the baseline phase. Three individuals with aphasia demonstrated positive changes in terms of response accuracy for 1-trial/session items. These observations were mirrored in the effect sizes: 4-trials/session items produced an extremely large change in response accuracy for A102 ($d=19.1$), a small change for A103 ($d=4.53$); nearly no change for A104 ($d=0.56$), and small change for A106 (2.34). One-trial/session items produced much smaller effect sizes in terms of response accuracy (ranging from -1.5 to 3.54).

Individuals with aphasia did not demonstrate consistent effects of stimulus dosage relative to the dependent measure of response time with the exception of participant A103. Based on visual analysis of the trend line, participant A103 demonstrated large decreases in response time during the training phase. Effect sizes revealed no change for response time for any of the participants for the maintenance phase relative to the baseline phase. The control participant demonstrated a small effect size ($d=-1.95$) for 4-trials/session items and no change for 1-trial/session items ($d=-0.99$).

Overall, the number of repetitions within a session appears to elicit inconsistent responses in individuals with aphasia for both response accuracy and

response time. Patterson et al. (1983) did not find any differences between the one and five repetitions within a single session. Two explanations were suggested in response to this early finding: (1) repetition priming protocols are not salient/meaningful enough to the individual with aphasia; (2) response accuracy is not a sensitive enough measure of repetition priming. Both of these explanations have been ruled out in the present study. Response/reaction time was actually a less sensitive to repetition priming for individuals with aphasia than response accuracy. As will be discussed below, all four participants reported that they enjoyed the protocol and found that it was a significant contributor to improved spontaneous spoken language production outside of the research environment. One possible explanation for the lack of observable difference between 1-trial/session and 4-trials/session items in the current study may be the large number of repetitions attempted by the participants by the time the first probe session occurred. Three training sessions occurred prior to the first probe session, in addition to the four baseline probes during which trained items were probed. Collectively, by the time the first training probe was administered participants had attempted to name 1-trial/session items a total of 10 times and each of the 4-trials/session items at total of 28 times. Future investigations will need to examine single repeated attempts at naming in contrast to 2, 3, 4, 5, etc. attempts at naming to better understand the influence of stimulus dosage on repetition priming.

GENERALIZATION OF REPTITION PRIMING

For all individuals with aphasia during acquisition and maintenance phases, trained items were responded to more accurately than untrained items, reflecting a lack of generalization to the skill of picture naming. This finding was expected given the nature of repetition priming documented in the literature; repetition priming is, by definition, item specific and is not theorized to elicit skill learning. In healthy participants; however, repetition priming has been shown to generalize to alternate exemplars of items previously named. That is, repetition of one example of "coffee" is expected to elicit faster and more accurate future productions of other pictures of "coffee". For individuals with aphasia, however, this was not the case. No consistent patterns were observed relative to alternate exemplars. Interestingly,

however, is the observation that all four individuals with aphasia were able to name some of items depicted by black and white line drawings in subtest 54 of the PALPA that happened to be trained in the repetition priming protocol using color photographs. That is, all four individuals with aphasia demonstrated post-training PALPA scores that were higher than prior to the repetition priming protocol and some of the items that contributed to their improved scores happened to be trained items in the repetition priming protocol. Frequent users of the PALPA may also suggest that test-retest reliability has not been properly investigated.

Two additional linguistically-based outcome measures were also administered following the repetition priming protocol to further examine generalization to picture naming (BNT) and generalization to language production across modalities (WAB). Three of the four individuals with aphasia demonstrated improvement of naming abilities as assessed by the BNT (A102, A103, A106); improvement ranged from 2-5 items. The Western Aphasia Battery AQ scores did not reflect any generalization to improved language production across modalities.

SUMMARY & CONCLUSIONS

The results of this investigation demonstrate that individuals with aphasia respond positively to repetition priming for both response accuracy and response time. This is an important finding when applying the principles of neuroplasticity and learning to rehabilitative medicine. Four tenets of learning have been widely discussed: (1) practice makes perfect, (2) use it or lose it, (3) fire together, wire together, and (4) you have to dream it to achieve it (Elbert & Rockstroh, 2004). Kleim and Jones (2008) have further specified the “practice makes perfect” tenet to include such principles as: (a) specificity of the task influences the nature of plasticity, (b) sufficient repetition is necessary, (c) sufficient training intensity is necessary, (d) time of delivery of treatment influences performance, and (e) the type of training must be salient to the learner. Results of this investigation contribute to this notion of “practice makes perfect”:

1. Specificity: repetition priming, by definition, reflects item-specific learning.

This type of learning was observed for both the individuals with aphasia and the control: trained items were responded to positively while untrained items

demonstrated no change or negative influences on response accuracy and/or response time. Generalization to the skill of naming was not observed.

2. Repetition: this investigation provided a starting point for future investigations of stimulus dosage. The amount of repetition in this study was sufficient to produce improvements across response accuracy and response time for individuals with a variety of underlying linguistic impairments. More detailed investigation of stimulus dosage, however, is warranted.
3. Treatment Intensity: This study cannot contribute reliable data about the total number of training sessions (i.e., treatment intensity). Although the largest effect size observed in this study stemmed from one of the participants who participated for all 15 training sessions (A102), the other participant who participated for 15 training sessions (A106) did not demonstrate such large gains. Subject variables including underlying linguistic impairment, time to fatigue, time post onset, and motivation are likely to contribute to the influence of treatment intensity on performance.
4. Time of Delivery: The protocol was not designed to directly examine time post onset relative to performance. However, the largest gains were observed for A102 whose stroke occurred the most recently relative to the onset of the repetition priming protocol (six months). Interestingly, however, the participant whose stroke was 3.5 years prior to enrollment in the study (A103) demonstrated the second highest effect sizes for changes in response accuracy. Based on this limited sample of individuals with aphasia, repetition priming does not appear to be particularly sensitive to the time of delivery relative to the time post onset of aphasia.
5. Saliency: All individuals with aphasia who enrolled in this study listed anomia as their most troublesome symptom of aphasia. As such a protocol that was designed to address picture naming was particularly salient to all four of the participants in this study. Anecdotally, all participants with aphasia reported some at-home practice of the items presented during the training phase. At the completion of the experimental protocol, all of the participants asked for complete lists of the items practiced so that they could continue work at

home. Reports of word-finding difficulties at time of enrollment coupled with anecdotal reports of at-home practice indicate that this repetition priming protocol was salient to these participants with aphasia. From a clinical perspective, it was very interesting that the participants found this protocol to be interesting – they received no immediate feedback about their performance and had minimal interactions with the experimenter during each session.

The current investigation has demonstrated that these principles of neuroplasticity are important to consider when designing future anomia treatment protocols. The underlying linguistic impairment may contribute a fair amount to the response to various treatment protocols; however, it is likely that if these principles of learning/neuroplasticity are implemented in a systematic fashion, aphasiologists will observe much greater change in a much shorter time. Not only did this intensive repetition priming protocol demonstrate improved response accuracy, these improvements persisted after six weeks of no practice. Mechanisms of learning known to be intact in healthy non-brain injured participants appear to be intact in individuals with aphasia as well. Therefore, it is our obligation as aphasiologists to apply these principles known to influence behavioral and neural plasticity in healthy individuals to individuals with aphasia.

FUTURE DIRECTIONS

Several additional analyses should be conducted on the current data:

1. Token analysis for probe data
2. Analyze training data to look at the pattern of errors within and across sessions.
3. Assess the influence of perseveration on probe data.
4. Collect a conversational sample of spontaneous speech pre and post repetition priming protocol.

Future protocols should address the following:

1. Assess the influence of stimulus dosage systematically (i.e., single repetitions vs. two, three, four repetitions).

2. Assess the effects of massed versus spaced (i.e., distributed) delivery schedules.
3. Systematically add on components typically used during anomia treatment protocols (e.g., item-by-item feedback for response accuracy, cuing hierarchies, etc).
4. Assess time of delivery (i.e., acute vs. chronic)

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APPENDIX A: CONSENT FORM – CONTROL PARTICIPANTS

UNIVERSITY OF WASHINGTON

INFORMATION AND CONSENT FORM

TITLE: Investigations of Spoken Language Production in
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206-295-3245***Researcher's Statement***

We want you to be in a research study.

This form will help you decide if you want to do this.

Please read this form carefully.

You may ask questions.

You can decide **yes** or **no** to be in the study.

We will give you a copy of this form to take home.

PURPOSE OF THE STUDY

We want to know more about the processes that occur in our brains. We are hopeful that this knowledge will provide insight into the speech problems that occur in people following a brain injury.

PROCEDURES

We want to videotape and audiotape you during the sessions so that we can have an accurate record of your responses. Only the researchers will have access to the tapes, which will be kept in a locked file cabinet. Your name will not appear on the tape. We will keep the tapes for 2 years, and then we will destroy them. You will have an opportunity to review and edit the recordings prior to our storing them. We may want to use the recordings for future studies. If you agree to be contacted about using your recordings for future studies, we will ask you for a separate written consent to use your recordings for this purpose. If you agree to be contacted for future studies, we will keep the video and audiotapes for 5 years prior to destroying them.

If you agree to be in this study, you will:

- (1) Fill out a questionnaire about personal and medical history;
- (2) Have your hearing and vision checked;
- (3) Have your speech, language, and thinking skills tested;
 - a. 1-2 hours total for testing
 - b. 1 session
 - c. **No** payment, but we can give you test results
- (4) Complete the experiment which will include a maximum of 26 sessions within a 11 week period:
 - a. **Sessions 1-5 (BASELINE TESTING):**
 - i. About 60 minutes (1 hour) each session
 - ii. May take place on the same day or separate days within a 2 week period, at your preference
 - iii. Your job:
 1. See pictures on a computer, and try to name them.
 2. We will measure how long it takes you, using a microphone on your neck.
 - b. **Sessions 6-12 (TRAINING SESSIONS):**
 - i. About 60-90 minutes per session
 - ii. Ideally, you will complete these 7 sessions within 4 days. You may participate in multiple sessions in one day. You will be asked to complete all 7 of the training sessions within a 2-week period.
 - iii. Your job:
 1. Try to name pictures on a computer.
 2. We will measure how long it takes you, using a microphone on your neck.
 - c. **Sessions 13-15 (FOLLOW-UP SESSIONS):**
 - i. Three sessions will be scheduled between 3 and 6 weeks AFTER you have finished the training
 - ii. About 60-90 minutes per session
 - iii. Your job:

1. Try to name pictures on a computer as you have done before.
- d. You will be asked to participate in the 7 training sessions and then come back for the 3 follow-up sessions several weeks later.
- e. You will be compensated for transportation or parking costs.

RISKS, STRESS OR DISCOMFORT

Some people do not like to be audio- or video-taped.
Some people feel uncomfortable when they are being tested.

All information we have about you will be confidential.

We will not share it with anyone.

But if you tell us about plans to hurt yourself, we will protect you by telling the appropriate people (like your doctor or family). If so, you will not be able to be in the study.

BENEFITS OF THE STUDY

We hope the results of this study will lead to important changes in how we diagnose and provide treatment for persons with neurological communication disorders.

Although we hope the findings from this study will benefit society, you may not directly benefit from taking part in the study.

OTHER INFORMATION

- 1) Taking part in this study is your choice.
 - a. You can stop at any time.
- 2) All of our information will have code numbers, not names.
 - a. The link between the code and your name will be kept in a locked location, separate from the study information.
- 3) We want to videotape your speech and language testing, and audiotape your responses in the experiment.
 - a. This will give us a record of what happens, so we can go back to it later.
 - b. The tapes will also be kept in a locked file cabinet.
 - c. Your name will not be on the tape.
 - d. We will keep your tapes and the link between your code and your name for 2 years, and then we will destroy them.
 - e. If you tell us that we can contact you for more research later, we will keep this information for 5 years, or until you tell us that you do not want to be contacted.
- 4) If we publish the results of this study, we will not use your name.
- 5) UW oversight review offices or federal regulators might need to see our study records about you.
 - a. This is to be sure we are being ethical and doing the research that we said we would.

Subject's statement

This study has been explained to me.

I have had a chance to ask questions.

I volunteer to take part in this research.

If I have questions later, I can ask one of the researchers listed above.

If I have questions about my rights as a research subject, I can call the Human Subjects Division at (206) 543-0098.

I give the researcher permission to audio- and/or video-tape record my sessions.

I will receive a copy of this consent form.

Printed name of participant

Signature of participant

Date

Participation in Future Research Protocols:

We may want to re-contact you about future related research. We will not share your name or contact information with any other research teams. You may contact us and have your name removed from our list of potential study participants at any time. Giving your permission for me to re-contact you does not obligate you in any way.

Can we contact you for future studies?

_____ **YES.** You may contact me in the future for research studies.

Signature of Subject

_____ **NO.** Thank you, but I am not interested.

APPENDIX B: CONSENT FORM – INDIVIDUALS WITH APHASIA

UNIVERSITY OF WASHINGTON

INFORMATION AND CONSENT FORM

TITLE: Investigations of Spoken Language Production in
Individuals with Aphasia

Principle Investigator

Catherine A. Off, Ph.C.
Doctoral Candidate - Researcher

Department of Speech and Hearing
Sciences
University of Washington
1417 NE 42nd Street
Seattle, WA 98105
206-685-2576

Faculty Sponsor:

Kristie Spencer, Ph.D.
Assistant Professor

Department of Speech and Hearing Sciences
University of Washington
1417 NE 42nd Street
Seattle, WA 98105
206-543-7980

Co-Investigator

JoAnn Silkes, Ph.C.
Doctoral Candidate - Researcher

Department of Speech and
Hearing Sciences

University of Washington
1417 NE 42nd Street
Seattle, WA 98105
206-295-3245

Researcher's Statement

We want you to be in a research study.

This form will help you decide if you want to do this.

Please read this form carefully.

You may ask questions.

You can decide **yes** or **no** to be in the study.

We will give you a copy of this form to take home.

PURPOSE OF THE STUDY

We want to learn about what happens when people with aphasia talk. This will help us understand aphasia better and possibly understand how the brain works when speaking.

PROCEDURES

If you agree to be in this study, you will:

- (1) Fill out a questionnaire about personal and medical history;
- (2) Have your hearing and vision checked;
- (3) Have your speech, language, and thinking skills tested;
 - f. 4-6 hours total for testing
 - g. 2-3 sessions
 - h. **No** payment, but we can give you test results
- (4) You will be video-taped during the testing sessions so that we have an accurate record of your responses so we can ensure accurate scoring of your responses; you will be audio-taped during the experiment itself so that we have an accurate record of your responses.
- (5) Complete the experiment which will include a maximum of 26 sessions within a 11 week period:
 - i. **Experimental Sessions 1-5 (BASELINE TESTING):**
 - i. About 60 minutes (1 hour) each session
 - ii. Must take place on separate days within a 2 week period
 - iii. Your job:
 1. See pictures on a computer, and try to name them.
 2. We will measure how long it takes you, using a microphone on your neck.
 3. You will also be asked to describe a complex picture scene – this is not timed.
 - j. **Experimental Sessions 6-20 (TRAINING SESSIONS):**
 - i. About 60-90 minutes per session
 - ii. Three times per week until you can name 80% of our pictures **OR** for a maximum of 15 training sessions
 - iii. Your job:
 1. Try to name pictures on a computer.
 2. We will measure how long it takes you, using a microphone on your neck.
 - iv. On the day of your last training session, we will re-test your language, speech and thinking skills. Some of these tests will be the same as those done in the first two sessions.
 - k. **Experimental Sessions 21-23 (FOLLOW-UP SESSIONS):**
 - i. Three sessions will be scheduled between 3 and 6 weeks **AFTER** you have finished the training
 - ii. Your job:
 1. Try to name pictures on a computer as you have done before.
 2. Try to describe a complex picture scene that contains many of the pictures you have been learning.
- l. You **MUST** be able to participate for 5 weeks in a row and then come back for the 3 follow-up sessions several weeks later.

m. You will be compensated for transportation or parking costs.

If you participate, we would like to get your medical records. This will help us know:

- When your stroke was.
- What part of your brain was affected. We will get this from any reports of MRI or CT scans.
- What your symptoms have been since your stroke.
- Whether you have any history of substance abuse or psychiatric disorders. If you do, we might not be able to use you in the study.
- Your current medications.
- Current speech, language or audiology reports (if any).

RISKS, STRESS OR DISCOMFORT

Some people do not like to be audio- or video-taped.
Some people feel uncomfortable when they are being tested.

All information we have about you will be confidential.

We will not share it with anyone.

But if you tell us about plans to hurt yourself, we will protect you by telling the appropriate people (like your doctor or family). If so, you will not be able to be in the study.

BENEFITS OF THE STUDY

It might help us learn how to do better therapy for aphasia.

This study **will not help you directly**, but the results might help other people in the future.

OTHER INFORMATION

- 2) Taking part in this study is your choice.
 - a. You can stop at any time.
- 3) All of our information will have code numbers, not names.
 - a. The link between the code and your name will be kept in a locked location, separate from the study information.
- 6) We want to videotape your speech and language testing, and audiotape your responses in the experiment.
 - a. This will give us a record of what happens, so we can go back to it later.
 - b. The tapes will also be kept in a locked file cabinet.
 - c. Your name will not be on the tape.
 - d. We will keep your tapes and the link between your code and your name for 2 years, and then we will destroy them.
 - e. If you tell us that we can contact you for more research later, we will keep this information for 5 years, or until you tell us that you do not want to be contacted.
- 7) If we publish the results of this study, we will not use your name.

- 8) UW oversight review offices or federal regulators might need to see our study records about you.
 - a. This is to be sure we are being ethical and doing the research that we said we would.

Subject's statement

This study has been explained to me.

I have had a chance to ask questions.

I volunteer to take part in this research.

If I have questions later, I can ask one of the researchers listed above.

If I have questions about my rights as a research subject, I can call the Human Subjects Division at (206) 543-0098.

I give the researcher permission to audio- and/or video-tape record my sessions.

I will receive a copy of this consent form.

Printed name of participant

Signature of participant

Date

Participation in Future Research Protocols:

We are planning more studies to understand aphasia.
Can we contact you for future studies?

There would be no obligation.
You can tell us any time to take your name off of our list.
We will not give your name or contact information to any other research team.

_____ **YES.** You may contact me in the future for research studies.

Signature of Subject

_____ **NO.** Thank you, but I am not interested.

APPENDIX C: AUDIO RECORDING PUBLICATION CONSENT FORM

UNIVERSITY OF WASHINGTON

AUDIO RECORDING PUBLICATION CONSENT FORM

TITLE: Investigations of Spoken Language Production in Individuals with Aphasia

<u>Principal Investigator:</u> Catherine A. Off, Ph.C., CCC-SLP	<u>Co-Investigators:</u> JoAnn P. Silkes, Ph.C, CCC-SLP
Doctoral Candidate - Researcher Department of Speech & Hearing Sciences University of Washington 1417 NE 42 nd Street Seattle, WA 98105 206.685.2576	Doctoral Student Researcher Department of Speech & Hearing Sciences University of Washington 1417 NE 42 nd Street Seattle, WA 98105 206-295-3245
<u>Faculty Sponsor</u> Kristie Spencer, Ph.D. Assistant Professor Department of Speech & Hearing Sciences University of Washington 1417 NE 42 nd Street Seattle, WA 98105 206-543-7980	

Researchers' Statement

USES OF THE AUDIO RECORDINGS

We want to know more about the language processes that occur in our brains. We are hopeful that this knowledge will provide insight into the speech problems that occur in people following a brain injury. We videotaped your participation during the assessment batteries and audio-taped your responses during naming tasks so that we can review the tapes for scoring accuracy. We would like to keep the audio and videotapes to use for our research for 5 years.

It is possible for someone who knows you to recognize your voice from the audiotape.

You have been given an opportunity to review the above audiotape(s) and we request your permission to keep the audio and videotapes until _____.

Only the researcher(s) listed on this consent form will have access to the audiotapes. The audiotapes will only be used for research purposes.

Printed name of researcher_____
Signature of researcher_____
Date**Subject's statement**

I have had an opportunity to review the recordings referenced above. I give my permission to the researchers to use the items as I have indicated above in this consent form. I understand that my name will not be published in connection with any publication. I will not receive any compensation for the use of the audio recordings. I will receive a copy of this consent form.

Printed name of subject_____
Signature of subject_____
Date

**APPENDIX D: MEDICAL RELEASE OF INFORMATION FORM
UNIVERSITY OF WASHINGTON
MEDICAL RELEASE OF INFORMATION FORM**

Name of Study: Investigations of spoken language production in individuals with aphasia.

Catherine A. Off, Ph.C. would like to obtain the following information from your medical records:

1. **Reports of any CT or MRI scans of your brain**
Purpose: To document the specific areas of your brain that have been affected by your stroke.
2. **Reports from your neurologist that provide information about the neurologically-based symptoms that have resulted from your neurologic condition**
Purpose: To be able to best describe the neurological deficits resulting from your stroke. Your neurologically-based symptoms may influence your performance in this study, so it is important for us to be aware of those symptoms.
3. **Medical information pertaining to past neurological events, disorders or diseases, psychiatric conditions, and/or substance abuse.**
Purpose: To ensure that you have not had any past cerebrovascular accidents (strokes) or other neurological disorders or diseases and to ensure that you do not have any psychiatric or substance abuse conditions that may affect your ability to participate in our study.
4. **Speech-language pathology or audiology reports (if applicable)**
Purpose: To add to the information we will obtain during our testing of your speech, language, and hearing.
5. **A list of your current medications**
Purpose: To identify any medications that may influence your performance during this study. For example, some medications may slow your reaction time. It would be important for us to know if your reaction times are different because of your medications.

This information may be obtained from the following medical center:

This information will be used for research purposes only, and will be received and reviewed only by the following investigators: Catherine A. Off (primary investigator), Kristie Spencer (faculty sponsor), and JoAnn Silkes (doctoral student). Your medical information will remain confidential. Your name will not be on the form to collect your medical information. Your study code number will be the only identifier associated with your medical information, and this information will be kept until _____ . Any medical records we receive that contain identifying information will be destroyed within 5 days after the relevant information is recorded on your Medical Records Data Collection Form.

You have the right to change your decision about our access to your medical records at any time. If you wish to withdraw your approval, please contact Catherine A. Off, Ph.C., at 206-685-2576. You have the right to refuse to sign this form.

Subject's Statement

I give permission to the researchers to use my medical records as described in this form.

Printed name of subject

Signature of subject

Date

APPENDIX E: HIPPA PRIVACY FORM**UNIVERSITY OF WASHINGTON****AUTHORIZATION TO USE, CREATE AND SHARE HEALTH INFORMATION FOR
RESEARCH FOR PROJECT ENTITLED*****INVESTIGATIONS OF SPOKEN LANGUAGE PRODUCTION IN INDIVIDUALS
WITH APHASIA***

Principal Investigator: Catherine A. Off, Ph.C., CCC-SLP

Faculty advisor: Kristie A. Spencer, Ph.D., CCC-SLP

University of Washington

Department of Speech and Hearing Sciences

Neurogenic Communication Disorders Laboratory

1417 NE 42nd St.

Seattle, WA 98105

206-685-2140

By law, researchers must protect the privacy of health information about you. In this form the word "you" means both the person who takes part in the research and the person who gives permission for another person to be in the research. Researchers may use, create, or share your health information for research **only if you let them**. This form describes what researchers will do with information about you. Please read it carefully. If you agree with it, please sign your name at the bottom. You will get a copy of this form after you have signed it.

If you sign this form, health information about you will be shared with the people who conduct the research. In this form, all these people together are called "researchers." Their names will appear on the research consent form that you sign.

The researchers will use the health information only as described in the research consent form that you sign.

1. What "health information" includes

- Information about you that is created during the research study. This might include the results of tests or exams that become part of the study records, diaries and questionnaires that you might fill out as part of the study, and other records from the study.
- Information in your medical record that is needed for this research study. This might include the results of physical exams, blood tests, x-rays, diagnostic and medical procedures and your medical history.

2. What the researchers may do with health information

The researchers may use and create health information about you for the study. They may also share your health information with certain people and groups. These may include:

- Government agencies, regulators, review boards, and others who watch over the safety, effectiveness, and conduct of the research. These may include such groups in the US and in other countries.
- Other researchers when a review board approves the sharing of the health information.
- Your health insurer if they are paying for care provided as part of the research study.
- Others, if the law requires.

3. Removing your name from health information

The researchers may remove your name (and other information that could identify you) from your health information. No one would know the information was about you.

If the identifiers are removed, the information may be used, created, and shared by the researchers and sponsor as the law allows. (This includes other research purposes.) This form would no longer limit the way the researchers use, create, and share the information.

4. How the researchers protect health information

The researchers will follow the limits in this form. If they publish the research, they will not identify you unless you allow it in writing. These limitations continue even if you take back this permission.

5. After the researchers learn health information

The limits in this form come from a federal law called the Health Insurance Portability and Accountability Act. This law applies to your doctors and other health care providers.

Once the researchers and others who are not your doctors and health care providers get your health information, this law may no longer apply. But other privacy protections will still apply.

6. Storing your health information

Your health information may be added to a database or data repository. This permission will end when the database or data repository is destroyed.

7. Please note

You do not have to sign this permission ("authorization") form. If you do not, you may not be allowed to join the study. You may change your mind and take back your permission at any time. To take back your permission, write to: Catherine Off, Department of Speech and Hearing Sciences, 1417 NE 42nd St., Seattle, WA, 98105. If you do this, you may no longer be allowed to be in the study. The researchers will keep any information about you they have already collected.

8. Expiration

This permission will expire when the purposes of the study have been met. This will happen no later than March 1, 2008.

9. Your signature

I agree to the use, creation, and sharing of my health information for purposes of this research study

Signature of research subject or subject's legal representative

Date

Printed name of research subject or subject's legal representative

Representative's relationship to subject

APPENDIX F: TELEPHONE SCREENING FORM – INDIVIDUAL WITH APHASIA

UNIVERSITY OF WASHINGTON Telephone Contact Protocol

Thank you for calling! How did you find our about our study? Were you given a flyer?

IF YES: Then you may already know that we are doing research at the University of Washington to learn more about what happens in the brain when we speak.

The study involves three parts: preliminary screening; testing of speech, language, and cognition; and experimental sessions. Would you like me to review with you what these three parts involve? ***IF YES...***The experimental screening should last approximately 1 hour. The testing should last 2-6 hours – you can do this all at once or split across 2 or 3 sessions. The experiment itself involves quickly naming pictures from a computer screen. Most importantly, our study tracks your progress over several weeks. The study involves coming in for 5 baseline sessions and then 3 experimental sessions per week until you are able to name pictures at 80% accuracy or for a maximum of 5 weeks/15 sessions. Each session will last between 1 and 2 hours. You will also be asked to participate in three follow up sessions between 3 and 6 weeks after your last experimental session.

IF NO: Then let me tell you a little bit about our study. We are researchers in the Speech and Hearing Sciences department at the University of Washington. We are doing a study to learn more about what happens in the brain when we speak. The study involves three parts: preliminary screening; testing of speech, language, and cognition; and experimental sessions. The experimental screening should last approximately 1 hour. The testing should last 2-6 hours – you can do this all at once or split across 2 or 3 sessions. The experiment itself involves quickly naming pictures from a computer screen. Most importantly, our study tracks your progress over several weeks. The study involves coming in for 5 baseline sessions and then 3 experimental sessions per week until you are able to name pictures at 80% accuracy or for a maximum of 5 weeks/15 sessions. Each session will last between 1 and 2 hours. You will also be asked to participate in three follow up sessions between 3 and 6 weeks after your last experimental session.

We are particularly interested in people who have had a stroke and an onset of aphasia at least 6 months ago. Does this apply to you?

IF YES:

- Can you tell me about the stroke? When did it occur? Do you know where, in the brain, the stroke happened?
- Can you tell me about the problems you have had as a result of the stroke?

- Can you tell me about the types of speech and language problems you have had as a results of the stroke?

IF NO:

- What is the nature of your problem?
 - *EITHER:* Yes, that is the sort of problem we are doing research on now...
 - *OR:* I'm sorry to hear of your difficulties. At this point, we're not doing research on the types of problems you're having. Perhaps in the future our research will cover more areas. Are there any questions I can answer for you? Thank you so much for calling...

IF STILL A POTENTIAL SUBJECT: May I ask you a few more questions to see if you meet our criteria for this particular study?

- (*IF NOT ALREADY STATED*): Are you between the ages of 21-85?
- What language did you first speak?
- Do you speak more than one language?
 - *IF YES:* What other language or languages do you speak in addition to English?
- Do you have problems with hearing or vision?
- Are you right handed or left handed?
- Have you had previous strokes or other neurological problems?
- Do you now or have you ever had any severe psychological conditions or substance abuse problems?
- Are you available to attend 3 1-2 hours sessions every week for approximately 5 consecutive weeks?

IF NOT APPROPRIATE BASED ON SCREENING QUESTIONS:

Thank you so much for your interest and for all of the information you've just given me. At this time, our study is not a good fit with you. Do you have any questions? Thank you so much for calling; we greatly appreciate your time and interest in this study.

IF STILL A POTENTIAL SUBJECT BASED ON SCREENING QUESTIONS:

At this point, it sounds as though you are a good fit for our study. If you agree to participate, you will be asked to fill out a short questionnaire regarding your basic medical history, complete a hearing and vision screening, and complete the testing and experiment I mentioned earlier. We will also need to access your recent medical history from your medical records. Is this okay with you? All we really need to know is where your stroke occurred, medications you are taking at this time, and any history of past neurological or psychological disorders, or substance abuse problems. You can stop participating in the study at any point in time, for any reason.

Are you still interested in participating?

IF YES: Great! Then let's get you scheduled to come in...Can you tell us where you were treated for the stroke? Thank you. Do you have contact information for this facility, including the doctor that treated you? I can get that from you now or you can bring it in with you on your first appointment.

IF NO: That's fine. Thank you so much for calling to find out about our study.

Should you have any additional questions about this study, please feel free to contact:

Catherine Off, doctoral candidate, at (206) 685-2576; cattalk@u.washington.edu

Please note that we cannot guarantee the confidentiality of information sent via email.

APPENDIX G: PRELIMINARY SCREENING QUESTIONNAIRE**BASIC PERSONAL INFORMATION**

Last Name: _____ First Name: _____

Telephone #: _____ Email: _____

Street Address:

City: _____ State: _____ Zip Code: _____

Date of Birth: _____ Age: _____

Place of Birth: _____

*****new page*******SOCIOECONOMIC INFORMATION (for descriptive purposes only)**

Race/Ethnicity: _____

Native Language (First Language Spoken): _____

If native language is not English, stop interview here, as participant does not qualify for study inclusion.

Other Languages Spoken: _____

Marital/Relationship Status
(CIRCLE ONE)**Single
Partner****Married****Living with Domestic****Living with Significant Other****Divorced****Widowed****Other: _____**

Number of Children: _____ Number of Children Currently in Household: _____

Education:
(CIRCLE ONE)**Less than High School
Grad****High school/Vocational/Some College****College**

Total Years of Education: _____
 Highest Degree Obtained: _____

Employment History:
(CIRCLE ONE)

Full Time Part Time Not Working Full Time Part Time
Not Working

Occupation: _____

Current Employment: _____

Retired from: _____

Year Retired: _____

Do you Receive Aid? (e.g., welfare,
 etc): _____

MEDICAL INFORMATION

(PLEASE CIRCLE ANSWERS)

Which hand do you use to do most things (e.g., write)? **LEFT** **RIGHT**

Do you have any problems with your hearing? **YES** **NO**

If yes, do you wear a hearing aid? **YES** **NO**

Do you have any problems with your vision? **YES** **NO**

If yes, is your corrected vision adequate enough
 to see most things? **YES** **NO**

When did your stroke occur? _____

What are your current symptoms/problems related to the stroke?

What medication(s) are you taking? And when do you usually take them?

Describe any problems with your speech/language.

APPENDIX H: MEDICAL RECORDS DATA COLLECTION FORM**Medical Records Data Collection Form**

Participant Code: _____ Age: _____ Year of birth: _____

Results of head CT, MRI, PET scans:**Notes from Neurology Reports:**Cerebrovascular accident(s):

- Most recent CVA:
 - Date of stroke:
 - Type of stroke:
 - Anoxia at time of stroke? YES NO
 - Lesion site(s):
 - Language Symptoms:
 - Cognitive Symptoms:
 - Motor Symptoms:
- Past CVA(s):
 - Date(s) of stroke(s):

Other neurological events, diseases, or disorders:

- Present:
- Past:

Medical information pertaining to psychiatric conditions or substance abuse:Psychiatric Conditions:

- Present:
- Past:

Substance Abuse Conditions:

- Present:
- Past:

Results of speech-language pathology, audiology, or neuropsychological testing:**Speech-language therapy (past or ongoing):**

List of current medications and dosage:

**APPENDIX I: TELEPHONE CONTACT SCREENING FORM –
CONTROL PARTICIPANTS**

**UNIVERSITY OF WASHINGTON
Telephone Contact Protocol**

Thank you for calling! How did you find our about our study? Were you given a flyer?

If YES: Then you may already know that we are doing research at the University of Washington to learn more about what happens in the brain when we speak. The study involves three parts: preliminary screening; testing of speech, language, and cognition; and experimental sessions. Would you like me to review with you what these three parts involve? ***IF YES...***The experimental screening and speech, language, and cognitive testing should last approximately 1-2 hours. You should be able to complete all of this in one session. The experiment itself involves quickly naming pictures from a computer screen. Most importantly, our study tracks your progress over 5 baseline sessions and 6 training sessions. The study involves coming in as often as is acceptable to you for a maximum of 3 weeks. You may participate in multiple sessions each day if you prefer this option. Each session will last between 1 and 2 hours. You will also be asked to come in for 3 follow-up sessions between 3 and 6 weeks after your final experimental session. Each of these sessions will last between 1 and 2 hours.

If NO: Then let me tell you a little bit about our study. We are researchers in the Speech and Hearing Sciences department at the University of Washington. We are doing a study to learn more about what happens in the brain when we speak. The study involves three parts: preliminary screening; testing of speech, language, and cognition; and experimental sessions. The experimental screening and speech, language, and cognitive testing should last approximately 1-2 hours. You should be able to complete all of this in one session. The experiment itself involves quickly naming pictures from a computer screen. Most importantly, our study tracks your progress over 5 baseline sessions and 6 training sessions. The study involves coming in as often as is acceptable to you for a maximum of 3 weeks. You may participate in multiple sessions each day if you prefer this option. Each session will last between 1 and 2 hours. You will also be asked to come in for 3 follow-up sessions between 3 and 6 weeks after your final experimental session. Each of these sessions will last between 1 and 2 hours.

IF STILL A POTENTIAL SUBJECT: May I ask you a few more questions to see if you meet our criteria for this particular study?

- ***(If NOT ALREADY STATED):*** Are you between the ages of 21-85?
- What language did you first speak?
- Do you speak more than one language?
 - ***IF YES:*** What other language or languages do you speak in addition to English?
- Do you have problems with hearing or vision?
- Are you right handed or left handed?
- Have you had strokes or other neurological problems?

- Do you now or have you ever had any severe psychological conditions or substance abuse problems?
- Are you available to attend several 1-2 hours sessions within a 3 week period until you have completed 5 baseline sessions and 6 training sessions?

IF NOT APPROPRIATE BASED ON SCREENING QUESTIONS:

Thank you so much for your interest and for all of the information you've just given me. At this time our study is not a good fit with you. Do you have any questions?

IF STILL A POTENTIAL SUBJECT BASED ON SCREENING QUESTIONS:

At this point, it sounds as though you are appropriate for our study. If you agree to participate, you will be asked to fill out a short questionnaire regarding your basic medical history, complete a hearing and vision screening, and complete the testing and experiment I mentioned earlier. You can stop participating in the study at any point in time, for any reason.

Are you interested in participating?

IF YES: Great! Then let's get you scheduled to come in...

IF NO: That's fine. Thank you so much for calling to find out about our study.

Should you have any additional questions about this study, please feel free to contact: Catherine Off, doctoral student, at (206) 579-6877; cattalk@u.washington.edu
Please note that we cannot guarantee the confidentiality of information sent via email.

APPENDIX J: PRELIMINARY SCREENING PROTOCOL

BASIC PERSONAL INFORMATION

Last Name: _____ First Name: _____
 Telephone #: _____ Email: _____
 Street Address: _____
 City: _____ State: _____ Zip Code: _____
 Date of Birth: _____ Age: _____
 Place of Birth: _____

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SOCIOECONOMIC INFORMATION (for descriptive purposes only)

Race/Ethnicity: _____
 Native Language (First Language Spoken): _____
If English is not the native language, stop interview here as participant does not qualify for study inclusion.
 Other Languages Spoken: _____

Marital/Relationship Status

(CIRCLE ONE)

Single Married
 Living with Domestic Partner
 Living with Significant Other Divorced Widowed
 Other: _____
 Number of Children: _____ Number of Children Currently in Household: _____

Education:

(CIRCLE ONE)

Less than High School High school/Vocational/Some College
 College Grad
 Total Years of Education: _____
 Highest Degree Obtained: _____

Employment History:

(CIRCLE ONE)

Full Time Part Time Not Working Full Time Part Time Not Working
 Occupation: _____
 Current Employment: _____
 Retired from: _____ Year Retired: _____
 Do you Receive Aid? (e.g., welfare, etc): _____

MEDICAL INFORMATION**(PLEASE CIRCLE ANSWERS)**

	LEFT	RIGHT
Which hand do you use to do most things (e.g., write)?		
Do you have any problems with your hearing?	YES	NO
If yes, do you wear a hearing aid?	YES	NO
Do you have any problems with your vision?	YES	NO
If yes, is your <u>corrected</u> vision adequate enough to see most things?	YES	NO
Do you have any history of speech, language, or neurological deficits or disorders? Please Describe (if any).		
Do you have any history of psychiatric conditions or substance abuse problems? Please Describe (if any).		

APPENDIX K: TRAINED AND UNTRAINED ITEMS

Trained Items

Item #	Stimulus	# Syllable	Freq	# Presentations
1	ant	1	LF	1
2	bacon	2	HF	1
3	book	1	HF	1
4	bottle	2	HF	1
5	bracelet	2	LF	1
6	candle	2	HF	1
7	car	1	HF	1
8	chair	1	HF	1
9	coffee	2	HF	1
10	cricket	2	LF	1
11	door	1	HF	1
12	drawer	2	LF	1
13	dryer	2	LF	1
14	eyes	1	HF	1
15	farmer	2	HF	1
16	flashlight	2	LF	1
17	football	2	HF	4
18	frog	1	LF	1
19	glass	1	HF	4
20	hanger	2	LF	4
21	harp	1	LF	1
22	heart	1	HF	4
23	island	2	HF	4
24	key	1	HF	4
25	leg	1	HF	4
26	lemon	2	HF	4
27	money	2	HF	4
28	mouse	1	LF	1
29	paintbrush	2	LF	4
30	paper	2	HF	4
31	pliers	2	LF	4
32	plunger	2	LF	4
33	rose	1	HF	4
34	rug	1	LF	1
35	shrimp	1	LF	4
36	stool	1	LF	4
37	stove	1	LF	4
38	syrup	2	LF	4
39	vase	1	LF	4
40	vest	1	LF	4

Untrained Items

Item #	Stimulus	#Syllables	Freq
1	arm	1	HF
2	baby	2	HF
3	ball	1	HF
4	barrel	2	HF
5	bed	1	HF
6	bra	1	LF
7	bridge	1	HF
8	broom	1	LF
9	bubble	2	HF
10	bucket	2	LF
11	bush	1	LF
12	canoe	2	HF
13	cans	1	LF
14	castle	2	HF
15	cheese	1	LF
16	church	1	HF
17	cigar	2	HF
18	clown	1	LF
19	comb	1	LF
20	corner	2	HF
21	crackers	2	LF
22	desk	1	HF
23	dime	1	LF
24	dog	1	HF
25	donkey	2	LF
26	dresser	2	LF
27	dustpan	2	LF
28	eagle	2	HF
29	earrings	2	LF
30	elbow	2	HF
31	faucet	2	LF
32	feet	1	HF
33	fire	2	HF
34	floor	1	HF
35	girl	1	HF
36	grapes	1	LF
37	hand	1	HF
38	hose	1	LF
39	hot dog	2	LF
40	iron	2	HF
41	judge	1	HF
42	kitchen	2	HF
43	kite	1	LF
44	knife	1	HF
45	ladle	2	LF
46	leaf	1	LF

47	leopard	2	LF
48	level	2	HF
49	lipstick	2	LF
50	mail	1	HF
51	man	1	HF
52	mattress	2	HF
53	menu	2	LF
54	mop	1	LF
55	moth	1	LF
56	mouth	1	HF
57	mushroom	2	HF
58	nails	1	LF
59	neck	1	HF
60	necklace	2	LF
61	nickel	2	LF
62	pants	1	LF
63	parrot	2	LF
64	peach	1	LF
65	pear	1	LF
66	pig	1	LF
67	pigeon	2	LF
68	pillow	2	LF
69	pizza	2	LF
70	plant	1	HF
71	popcorn	2	LF
72	present	2	HF
73	pumpkin	2	HF
74	record	2	HF
75	river	2	HF
76	robe	1	LF
77	rooster	2	LF
78	salad	2	HF
79	sandwich	2	LF
80	saw	1	HF
81	shears	1	LF
82	shoulder	2	HF
83	skunk	1	LF
84	spider	2	LF
85	steps	1	HF
86	teacher	2	HF
87	toe	1	LF
88	toothbrush	2	LF
89	top	1	HF
90	train	1	HF
91	vet	1	LF
92	waffles	2	LF
93	wall	1	HF
94	wallet	2	LF

95	water	2	HF
96	well	1	HF
97	wheel	1	HF
98	window	2	HF
99	wolf	1	LF
100	woman	2	HF

APPENDIX L: TIMING PARAMETERS

<u>Trial Run</u>	<u>Aphasics</u>	<u>Controls</u>
Instruction Screen	Infinite	Infinite
Warning Dot	2000 ms	1000 ms
Picture	17000 ms (100 prerelease)	2000 ms (100 prerelease)
Red X	4000 ms	1000 ms
Goodbye Screen	5000 ms	5000 ms
<u>Baseline Probes</u>		
Instruction Screen	Infinite	Infinite
Warning Dot	2000 ms	1000 ms
Picture	17000 ms (100 prerelease)	2000 ms (100 prerelease)
Red X	4000 ms	1000 ms
Goodbye Screen	5000 ms	5000 ms
<u>Training Session</u>		
Instruction Screen	Infinite	Infinite
Warning Dot	2000 ms	2000 ms
Picture	10000 ms (100 prerelease)	2000 ms (100 prerelease)
Red X	2000 ms	1000 ms
Picture+audio+ortho	12000 ms (100 Prerelease)	2000 ms
Red X	4000 ms	1000 ms
Goodbye Screen	5000 ms	5000 ms
<u>Training Probes</u>		
Instruction Screen	Infinite	Infinite
Warning Dot	2000 ms	1000 ms
Picture	17000 ms (100 prerelease)	2000 ms (100 prerelease)
Red X	4000 ms	1000 ms
Goodbye Screen	5000 ms	5000 ms
<u>Stimulus</u>		
<u>Generalization Probes</u>		
Instruction Screen	Infinite	Infinite
Warning Dot	2000 ms	1000 ms
Picture	17000 ms (100 prerelease)	2000 ms
Red X	4000 ms	1000 ms
Goodbye Screen	5000 ms	5000 ms
<u>Maintenance Probes</u>		
Instruction Screen	Infinite	infinite
Warning Dot	2000 ms	1000 ms
Picture	17000 ms (100 prerelease)	2000 ms
Red X	4000 ms	1000 ms
Goodbye Screen	5000 ms	5000 ms

APPENDIX M: ERROR CODE TAXONOMY

Error Description	Error Code	Example
I. Accurate		
	A. Target only	
	B. Filler + target	um, uh, a, the
	C. Multiple correct productions	"mattress mattresses"
	D. Multiple productions the first correct	"bra brave"
II. Errored		
	A. No response or "I don't know" "I'm sorry"	
	B. Mixed	
	i. Phonological + semantic	/dan/ for /kæt/
	ii. Phonological + unrelated word	/flon/ for /kæt/
	C. Semantic	
	i. Unrelated	"shoe" for "cat"
	ii. Supraordinate	"mammal" for "cat"
	iii. Coordinate	"dog" for "cat"
	iv. Subordinate	"siamese" for "cat"
	v. Related adjective	"white" for "milk"
	vi. Related verb	"drink" for "milk"
	D. Perseveration	Produces any previously produced item
	E. Phonological	
	i. Omission	/æ/ for /kæt/
	ii. Substitution	/kɪ/ for /kæt/
	iii. Addition	/kræt/ for /kæt/
	iv. Nonword	
	F. Picture description	"a woman washing dishes"

VITA

Catherine A. Off was born in Santa Monica, California. She began her academic career at the University of California, Berkeley where she obtained her Bachelor of Arts in Linguistics. She then moved to Laramie, Wyoming to complete her Masters of Science in Communication Disorders. Catherine then relocated to complete her Clinical Fellowship Year (CFY) in Speech-Language Pathology at the Veteran's Affairs Medical Center (VAMC) in Philadelphia, Pennsylvania. After completing her CFY, she remained at the VAMC in Philadelphia as a full-time Speech-Language Pathologist. After two and a half years of clinical practice, Catherine returned to academia to pursue her doctorate in Speech and Hearing Sciences under the mentorship of Margaret Rogers, Ph.D. and Kristie Spencer, Ph.D. In 2008, Catherine earned her Ph.D. in Speech and Hearing Sciences at the University of Washington.