

## **Phonologic Rehabilitation of Anomia in Aphasia**

**Diane L. Kendall, Ph.D.**

VA RR&D Brain Rehabilitation Center  
University of Florida, Department of Communication Science and Disorders  
University of Florida, McKnight Brain Institute  
Gainesville, Florida

**John Rosenbek, Ph.D.**

University of Florida, Department of Communicative Disorders  
VA RR&D Brain Rehabilitation  
Gainesville, Florida

**Kenneth Heilman, M.D.**

University of Florida, Department of Neurology  
VA RR&D Brain Rehabilitation Center  
Gainesville, Florida

**Tim Conway, Ph.D.**

VA RR&D Brain Rehabilitation Research Center, Malcom Randall DVA Medical Center;  
University of Florida, Department of Clinical and Health Psychology  
Gainesville, Florida

**Karen Klenberg, M.A.**

VA RR&D Brain Rehabilitation  
Gainesville, Florida

**Leslie J. Gonzalez Rothi, Ph.D.**

VA RR&D Brain Rehabilitation Research Center, Malcom Randall DVA Medical Center;  
University of Florida, Departments of Neurology and Clinical and Health Psychology  
University of Florida, McKnight Brain Institute  
Gainesville, Florida

**Stephen E. Nadeau, M.D.**

Geriatric Research, Education and Clinical Center  
VA RR&D Brain Rehabilitation Research Center, Malcom Randall DVA Medical Center;  
University of Florida, Department of Neurology  
University of Florida, McKnight Brain Institute  
Gainesville, Florida

**Correspondence to:**

Diane L. Kendall, Ph.D.  
VA Brain Rehabilitation and Research Center  
Box 151A, 1601 Archer Road  
Gainesville, Florida 32608-1197  
Phone: 352-376-1611, x5238  
Fax: 352-379-2332  
Email: [dkendall@csd.ufl.edu](mailto:dkendall@csd.ufl.edu)

Acknowledgements: This study was supported by the VA RR&D Brain Rehabilitation and Research Center and VA RR&D Career Research Development Award (C2743V). The authors would like to thank the subjects and their families for their participation and support of this research.

**ABSTRACT:** This study investigated the effects of an intensive phonologic treatment for anomia in aphasia. The theoretical motivation for this investigation was based on a parallel distributed processing model of phonology (Nadeau, 2001). We proposed that if treatment were directed at the level of the phonologic processor (e.g. phonemes and phoneme sequences), opportunities for naming via a phonological route, as opposed to a strictly whole word route, would be enhanced, thereby improving naming. Ten individuals with chronic anomia and aphasia due to left hemisphere stroke served as participants. A single-subject, repeated probe design with replication across individuals was employed. The primary outcome measure was confrontation naming. Secondary outcome measures included discourse production (word count and content information units), phonologic production and nonword repetition. Data were analyzed visually and statistically. Results showed a positive treatment effect (improved phonologic production) and generalization to confrontation naming and discourse production (word count and content information units). Effects of treatment were maintained 3 months post treatment termination for confrontation naming and discourse production (content information units). The results of this study provide sufficient evidence of efficacy to encourage further research.

## INTRODUCTION

The single most common feature of aphasia, and one of the most debilitating aspects of aphasia in most patients, is impairment in ability to name, whether it involves naming seen objects, or producing nouns, verbs and other words conveying meaning in spontaneous language (Goodglass, 1993). The traditional treatment approach to this problem is to explicitly train aphasic patients in naming. Controlled studies have shown that this approach may be effective. However, typically, generalization is very limited, that is, the knowledge gained by the patient tends to be limited to the words actually trained, and there is at best very modest improvement in performance with untrained words. This generalization may be limited mainly to words that are semantically related to those in the training corpus (Coelho *et al.*, 2000; McNeil, 1997). However, more generally, the mechanisms underlying generalization are not well understood. Because generalization is so limited with “naming therapies”, there currently exists no viable means of training patients on the full corpus of words (perhaps several thousand) they are likely to need in daily life, except in the most determined and capable of subjects (Basso, 2003). Two approaches might be taken to solving this problem: (1) develop cost effective means for providing training on several thousand words; and (2) develop alternative training methods, e.g., phonological therapy, that could potentially generalize widely. In this paper, we describe a connectionist model that provides a rationale for phonological therapy and we report the results of the first clinical trial motivated by this model.

### *Connectionist model of phonological function*

The Wernicke-Lichtheim (W-L) information processing model of language function has played a dominant role in understanding aphasic syndromes (Lichtheim, 1885) and has stood the test of time in defining the topographical relationship between the modular domains (acoustic representations, articulatory-motor representations, and concept representations) underlying spoken language function. Unfortunately, the W-L information processing model does not specify the characteristics of the representations within these domains and how they might be stored in the brain. It also does not address the means by which these domains might interact. We have

proposed a parallel distributed processing (PDP) model that uses the same general topography as the W-L model (Nadeau, 2001), but also specifies how representations are generated in the modular domains and how knowledge is represented in the links between these domains (Figure 1). Though not tested through simulations, this model is neurally plausible and provides a cogent explanation for a broad range of psycholinguistic phenomena. More generally, connectionist concepts are now deeply embedded in and receive enormous support from mainstream neuroscientific research (e.g., (Rolls, 2002; Rolls, 1998)).

INSERT FIGURE 1 ABOUT HERE

The PDP modification of the W-L model posits that the acoustic domain (akin to Wernicke's area) contains large numbers of units located in auditory association cortices that represent acoustic features of phonemes. The articulatory domain (analogous to Broca's area) contains units located predominantly in dominant frontal operculum that represent discrete articulatory features of speech (as opposed to continuously variable motor programs). The semantic or conceptual domain contains an array of units distributed throughout unimodal, polymodal and supramodal association cortices that represent semantic features of concepts. Within any domain, a representation corresponds to a specific pattern of activity of all the units, hence the term distributed representation. Each unit within each of these domains is connected via interposed hidden units to many, if not most, of the units in the other domains. During learning of a language, the strengths of the connections between the units are gradually adjusted so that a pattern of activity involving the units in one domain elicits the correct pattern of activity in the units of another domain. The entire set of connections between any two domains forms a pattern associator network. The hidden unit regions, in conjunction with nonlinear unit properties, enable the association of representations in two connected domains that are arbitrarily related to one another (e.g., word sound and word meaning).

In PDP models, knowledge is stored as patterns of connectivity not only within domains but also between domains. For example, understanding the meaning of a word that is heard is

achieved through the connections between the domain that contains the sound features of language and the domain that contains concept features (the acoustic-concepts representations pattern associator, Figure 1, pathway 6-5). This pattern associator network corresponds to the cognitive neuropsychological concept of a phonological input lexicon (Ellis & Young, 1988).

The knowledge that allows a person to translate heard sound sequences into articulatory sequences, and thereby mediates repetition of both real words and non-words, is contained in the network that connects the acoustic domain to the articulatory motor domain (the acoustic-articulatory motor pattern associator, Figure 1, pathway 7-3). Because this network has acquired, through experience, knowledge of the systematic relationships between acoustic sequences and articulatory sequences, it has learned the sound sequence regularities of the language: the phonemic sequences of joint phonemes, rhymes, syllables, affixes, morphemes and words characteristic of the language (Nadeau, 2001) (see also (Plaut *et al.*, 1996)).

The knowledge that enables a person to translate a concept into a spoken word (the phonological output lexicon (Ellis & Young, 1988)) is contained in two different pattern associator networks that connect the concept representations domain to the articulatory motor domain (Figure 1, pathways 1-2 and 4-3). These two pattern associator networks support different forms of knowledge. The indirect concept representations-articulatory motor pathway (pathway 4-3) provides a robust basis for knowledge of sequences and sublexical entities because of the sequence knowledge stored in the acoustic-articulatory motor pattern associator. However, the direct concept representations-articulatory motor pattern associator (pathway 1-2) does not contain much knowledge of sequences and sublexical entities because it translates spatially distributed patterns of activity corresponding to concepts into temporally distributed sequences of activity corresponding to articulated words. This spatial-temporal translation precludes significant acquisition of sequence knowledge and makes this fundamentally a whole word pathway. The existence of this direct, whole word naming route finds support in studies of subjects with repetition conduction aphasia; some appear to have lost most phonological sequence knowledge (pathway 3/4-7)(resulting in a severe deficit in auditory verbal short term memory), but can speak quite well, producing few if any phonological paraphasic errors, can repeat real words (with

evidence of influence by semantic attributes but little influence of word length), and are severely impaired in repeating nonwords and functors (Warrington, 1969; Friedrich, 1984; Saffran, 1975; Caramazza, 1981). It also finds some support in reports of subjects with conduction aphasia who are able to repeat words better than nonwords (McCarthy, 1984; Caramazza, 1986; Friedrich, 1984; Saffran, 1975), and who are able to repeat words better when they are given in a sentence context than when given as a single word (thereby increasing the likelihood of engaging concept representations) (McCarthy, 1984). However, a model in which the only link from the concept representations domain to the articulatory motor domain is the direct one (pathway 1-2) cannot account for observations that normal subjects exhibit phonological slips-of-the-tongue, and aphasic subjects produce phonemic paraphasias in naming and internally generated spoken language quite comparable to those produced during repetition. To explain these observations, one must posit access from concept representations to phonological sequence knowledge, as indicated in pathway 4-3 of the model. Thus, this PDP model predicts that there should be two pathways enabling naming of concepts. We have recently provided further evidence supporting this model in a report of a patient who, depending upon type of verbal cue provided, could be induced to use either the whole word naming route or the phonological naming route (Roth et al., in press) This model can also readily be mapped to the brain in a way that is consistent with established functional-anatomic correlations (Roth, In press).

*Implications of the model for treatment of anomia*

The relationship between word meaning and word form is largely arbitrary. This is likely the reason that learning to name one word provides no basis for generalization to other words (except for derivational forms and words that have similarities in meaning) (Plaut, 1996), and that to meaningfully alter daily communicative ability, one would have to train hundreds, if not thousands of words (as noted in the foregoing). If the direct pathway of the model, a substantially whole word pathway, were the only pathway available to us to name concepts, then we would be bound by this constraint. However, the existence of the indirect pathway opens up another possibility. So long as there are some remnants of this pathway left after a stroke (either in the

damaged hemisphere or in the normal hemisphere), that is, so long as there is some existing phonological sequence knowledge and some connections between neural networks supporting concept representations and the acoustic-articulatory motor pattern associator supporting phonological sequence knowledge, then it may be possible to improve word retrieval by enhancing phonological sequence knowledge. This is the fundamental hypothesis that motivates this investigation.

Studies of language acquisition in young children suggest that first they learn many of the various phonological sequence regularities of their language (Gathercole, 1995; Gathercole & Martin, 1996). Subsequently they learn to assemble these various sequences into combinations and associate these combinations with concepts (meaning) to enable word comprehension and word production. If this principle of language development also applies to language redevelopment after brain injury, it suggests two possibilities: (1) that effective retraining in phonological sequence knowledge may generalize to all words containing the trained sequences; and (2) that once given an adequate repertoire of phonological sequence knowledge during treatment, aphasic patients should be able to continue after therapy to enhance existing but inadequate connections between the substrate for concept representations and the substrate for phonological sequence knowledge and steadily rebuild their working vocabularies. It is also possible that training some phonological sequences will generalize to other phonological sequences (e.g., through shared distinctive feature and motor programming sequences).

#### *Précis of the study*

The primary purpose of this Phase II clinical rehabilitation study was to examine the effect of a phonologic based treatment on confrontation naming by individuals with anomic aphasia. We used a single-subject ABA design replicated across ten participants. The primary research question asked if phonologic treatment would improve confrontation naming. Secondary research questions addressed the impact of treatment on 1) generalization to untrained behaviors such as discourse production; 2) retention effects at 3-months; 3) phonologic production and 4) nonword repetition (potential evidence of phoneme sequence knowledge acquisition).

## **METHODS**

### **Participants**

Participants were recruited through the VA RR&D Brain Rehabilitation and Research Center, Gainesville, Florida under IRB #545-99. Six males and 4 females, with an average age of 52 years and an average of 59.7 months post stroke onset served as participants. To be eligible for the study intervention, subjects had to have had a single left hemisphere stroke (documented by imaging with either CT or MRI), be 6-months or more post-stroke, be right handed and monolingual English speaking. Exclusion criteria included significant apraxia of speech, depression or other psychiatric illness (unless successfully treated), neurological illnesses (e.g. Alzheimer's disease, Parkinson's disease), chronic medical illness (e.g. cancer, renal failure), and severe impairment in vision or hearing. Table 1 lists relevant participant demographic information.

INSERT TABLE ONE ABOUT HERE

The presence of apraxia of speech was determined perceptually using data gathered during the evaluation. Two speech language pathologists evaluated speech/language behaviors and arrived at independent judgments. Video-taped data from, but not limited to, Western Aphasia Battery (WAB)(Kertesz, 1982) picture description, spontaneous conversation, automatic speech, repetition of words of increasing length and multiple repetition of 3-syllable words were evaluated for the following behaviors: slow rate, prolonged segment durations and intersegment durations (including intrusive schwa), distortions, prosodic abnormalities and effortful groping and struggling during articulation. Significant apraxia of speech was defined as a limited repertoire of speech sounds, speech limited to a few meaningful utterances, automatic speech not better than volitional speech, and an inability to repeat isolated phonemes.

For diagnostic and descriptive purposes, individuals were given a series of standardized test batteries pre- and post-treatment to assess linguistic and phonologic function. To be



included in the research protocol, individuals had to have: (1) demonstrated anomia on the Western Aphasia Battery (Kertesz, 1982) (subtest scores between 5-10 on fluency, 7-10 on comprehension, 7-10 on repetition and 0-9 on naming); (2) auditory comprehension sufficient to complete the training protocol (score of >30 on the WAB yes/no subtest); (3) score < 45 on the Boston Naming Test (Kaplan *et al.*, 1983)(BNT); and (4) evidence of phonological function that was present<sup>1</sup> but impaired (impaired scores on Comprehensive Test of Phonologic Processing (Wagner, R., Torgesen, J., & Rashotte, C. 1998)(CTOPP) and Lindamood Auditory Conceptualization) (Lindamood, C. H., & Lindamood, P. C. 1979) (LAC).

### **Treatment procedures**

This study investigated a phonologic based treatment using individual phonemes and nonword phoneme sequences. Treatment was administered 2 hours/day, 4 days/week for 12 weeks for a total of 96 hours by three experienced speech language pathologists. The average treatment session length was 1 hour. The Principal Investigator and a consultant provided 20 hours of training in administering the phonologic based treatment to the therapists prior to data collection. The PI and consultant observed 25% of the treatment sessions to maintain treatment integrity and evaluate clinician performance. The PI and consultant met with the therapists 1-2 hours/week in lab meetings to review subject performance. The treatment program is outlined in detail in Appendix A.

### **Treatment stimuli**

Trained consonants included /p,b,f,v,t,d,k,g,th,th,s,z,sh,zh,ch,j,l,r,w,h,wh,m,n,ng/ and trained vowels included /ee,i,e,a,ae,u,o,oe,oo/. Stimulus selection was based on prior research (Conway et al, 1998; Kendall et al 2003; Kendall et al, In Press). Phonemes were initially presented in isolation and upon mastery, sounds were combined into two phoneme combinations (CV and VC), three phoneme combinations (CVC, VCC, CCV), and eventually, into two- and three-syllable combinations (Appendix A outlines details).

### **Experimental Design**

---

<sup>1</sup> Phonological paraphasic errors in naming to confrontation or internally generated language; some benefit from phonological cueing in naming to confrontation; some ability to repeat nonwords.

A single-subject ABA repeated-probe design with replication across 10 participants with pre- and post-treatment testing was employed. Pre- and post-test measures included the Western Aphasia Battery (WAB) (Kertesz, 1982), Boston Naming Test (BNT) (Kaplan *et al.*, 1983), Controlled Word Association Test (COWA) (Benton & Hamsher, 1989), Comprehensive Test of Phonological Processes (CTOPP) (Wagner *et al.*, 1998), and the Lindamood Auditory Conceptualization Test (LAC) (Lindamood & Lindamood, 1979).

During the initial “A”, or no-treatment, phase, eight baseline data points were established for treatment (production of trained phonemes), generalization (confrontation naming and discourse production) and control (Test of NonVerbal Intelligence-TONI) (Brown *et al.*, 1990) probes. During the “B”, or treatment phase, the same repeated probes were administered after every 8 hours of therapy (or 1x/week). The treatment phase was immediately followed by 1-2 sessions of post-testing in which the repeated probes and standardized tests (described above) were administered. Follow-up testing occurred at 3-months.

### **Outcome Measures**

#### *Outcome Measure Description*

Primary and secondary research questions were answered by the administration, rating, and analysis of several outcome measures. The primary outcome question, generalization of treatment to confrontation naming, was addressed by analyzing responses on the 81-item Object Action Naming Test (Durks, J., & Masterson, J., 2000). The secondary outcome question, generalization to discourse production, was addressed by analyzing word count and content information units (CIU) elicited by Brookshire Discourse Production Test (Nicholas, L. E., & Brookshire, R. H., 1993). To assess the impact of treatment on the treated behavior, we employed a non-standardized 20-item phoneme production task using consonants trained in therapy. The impact of treatment on acquisition of phoneme sequence knowledge was tested using a non-standardized 10-item repetition test employing 2-syllable nonwords comprised of trained sounds and sound sequences.

#### *Outcome Measure Analysis*

All outcome measures were audio-taped using an analogue tape recorder. The therapist conducted the scoring online during the session, and this scoring was also later judged by a trained rater blind to the time of testing. The Object/Action Naming Test data were scored incorrect if productions included semantic or phonologic substitutions. Distortion errors were scored as correct. The discourse sample was transcribed and scored according to standard procedure for word count and content information units (CIU). Phonemes on the phoneme production test and nonword repetition test were scored as “plus” if correctly produced or “minus” if distortion, phonologic substitution or omission occurred. Inter-rater reliability was assessed using intra-class correlations (ICC) computed for 25% of the repeated probe data.

The percentage of the participants’ correct responses was graphed for analysis. The data were then analyzed visually and statistically.

#### *Visual Analysis*

Visual analysis of outcome measure data was completed by three judges, all speech-language pathologists with at least 3 years experience judging data via visual inspection. Each independently judged the stability of both baseline phases for each participant and then considered the relative slope and height of the data displays during the treatment phase. Figure 1 displays a sample of the graphs the judges used for visual analysis.

#### *Statistical Analysis*

Repeated probe data were analyzed in terms of effect sizes (ES) (Kromrey & Foster-Johnson, 1996). The formula used to calculate ES was:

$$ES = (\text{Mean}_{\text{therapy}} - \text{Mean}_{\text{baseline}}) / SD_{\text{baseline}}$$

Effect sizes > .8 were considered large, >.5 medium and >.2 small (Cohen, 1988). Paired Student’s t-tests were used to analyze standardized test differences before and after treatment.

## **RESULTS**

INSERT TABLE 2 ABOUT HERE

#### *Primary Outcome Results:*

For the Object/Action Naming Test (confrontation naming), the average ES was 1.63 (Table 3). Graphs were judged to show evidence of generalization for 9/10 individuals (Figures 2 and 3).

INSERT TABLE 3 ABOUT HERE

INSERT FIGURES 2 AND 3 ABOUT HERE

#### *Secondary Outcome Results*

For discourse production, of the average ESs were 1.49 (word count) and 1.71 (CIU). Graphs were judged to show evidence of generalization for 5/8 individuals (word count) and 4/8 individuals (CIU). For phoneme production, the average ES was 6.88 and graphs were judged to show evidence of a treatment effect in 10/10 individuals. For nonword repetition, the average ES was .95 and graphs were judged to show evidence of a treatment effect in 5/6 individuals.

In analysis of retention of gains 3-months post-treatment termination, the average ES for confrontation naming was 1.53 and graphs were judged to show maintenance in 7/8 individuals. The average ES's for discourse were 1.40 for word count and 1.41 for CIU and graphs were judged to show maintenance effects in 1/6 for word count and in 4/6 for CIU. The Average ESs for phonologic production and nonword repetition were 5.73 and 1.12, respectively, and graphs were judged to demonstrate maintenance effects in 5/8 (phonologic production) and 5/6 (nonword repetition).

#### *Standardized Pre- and Post-test Results:*

Using Student's paired t-tests, significant changes in WAB ( $p=0.001$ ), CTOPP-PA ( $p=0.039$ ), CTOPP-APA ( $p=0.008$ ) and LAC ( $p=0.021$ ) scores reflecting acquisition effects (immediately post treatment – pre-treatment) were found. Significant changes in BNT ( $p=0.017$ ), COWA ( $p=0.011$ ) and LAC ( $p=0.006$ ) reflecting retention effects (3 months post treatment – pre-treatment) were found (Table 2). Data for two subjects (P1 and P2) were not collected on the

CTOPP due to clinician error. Two subjects (P2 and P6) failed to return for follow-up testing because of intercurrent medical problems and travel distance.

*Reliability:*

Inter-class correlations assessing inter-rater reliability were 0.97 for confrontation naming, 0.99 for discourse word count, and 0.70 for discourse CIU.

## **DISCUSSION**

The current study is a Phase II investigation designed to test the effect of a phonologic treatment for anomia in aphasia. The data from the ten participants provide evidence to support our hypothesis that by focusing treatment at the level of the phonologic processor (e.g. phonemes and phoneme sequences) it is possible to improve naming, presumably by increasing the opportunity for naming via the phonological route. The effect sizes for the outcome measures were large by traditional standards (Cohen, 1988) and within the range of effect sizes in the aphasiology literature (Robey, 1999). It is also noteworthy that subjects demonstrated a mean gain by three months after completion of therapy of 9.5 points on the BNT and 5.12 points on the COWA; both changes were statistically significant. Most of these gains were achieved after completion of therapy. This finding provides tentative support for our corollary hypothesis that once given an adequate repertoire of phonological sequence knowledge during treatment, aphasic subjects with anomia may, on their own, be able to continue after therapy to enhance connections between the substrates for concept representations and phonological sequence knowledge and thereby continue to build their working vocabulary.

The therapy reported here is the first version of phonologic treatment developed in our laboratory. We regard these initial results as providing only tentative evidence of efficacy. Our experience with these first 10 subjects has provided a number of ideas for modifications of the therapy that might enhance or better measure efficacy, discussed below.

In this study, all subjects with anomia, largely spared comprehension, and evidence of both some residual phonological sequence knowledge and impairment in that knowledge were recruited. While subjects with severely impaired phonological perception were able to learn individual sounds, as evidenced in repeated probe phonologic production, they showed less

generalization to nonword repetition and confrontation naming. This suggests that subjects with severely impaired phonological perception are not likely to be good candidates for this treatment.

This study employed 96 hours of treatment. It is possible that eventually, with a sufficiently effective treatment and sufficiently good empirical demonstration of effectiveness, medical economic calculations would justify 96 hours of treatment. However, there is no question that our treatment, as currently designed and implemented, has unacceptably low efficiency. Visual inspection of our probe measure suggests that, even for variables marked by more gradual gains (e.g., confrontation naming, nonword repetition), to the extent that there were gains, they had been substantially achieved by 60 hours. It may also be possible to alter the therapy such that gains/hour of treatment are increased. One way might be to limit treatment to common phonemes and phoneme sequences (phonemes and sequences of high phonotactic probability (Vitevitch & Luce, 1999). Work in children has recently shown that common phonemes and phonological sequences and the ability to link these sequences to semantic representations are acquired more rapidly than are uncommon phonemes and sequences (Storkel, 2001).

Another manipulation of the therapy that could potentially improve its efficiency would be to incorporate real words into the training process. The hypothesis to be tested is that the incorporation of real words will improve outcome by enhancing Hebbian learning in the process of phonological sequence knowledge acquisition. The training in the current study utilized auditory, visual and tactile-kinesthetic input to develop phonological sequence knowledge. Because we did not use real words, we did not take advantage of potential top down effects of residual lexical semantic knowledge (in either hemisphere) that might have enhanced co-activation of phonemes in specific phonological sequences, thereby increasing the opportunity for Hebbian learning to occur that would increase binding of the phonemes into sequences.

Finally, it is likely that our outcome measures were not adequate. All subjects demonstrated evidence of learning the phonemes (repeated probe phoneme production). Most subjects showed significant gains in repetition of nonwords incorporating trained sequences. Some showed modest gains on standard measures of naming (e.g., the Object/Action Naming Test) (Durks & Masterson, 2000) and discourse production in response to pictures (the

Brookshire Discourse Production pictures) (Nicholas & Brookshire, 1993). However, subjects and families commonly reported major improvements in ability to communicate in daily life. It is uncertain whether these reports reflected a genuine but not measured improvement in communicative ability, or merely a perception of improvement that was an artifact of the training experience. It did appear that our discourse stimuli (the Brookshire Discourse Production pictures) tended to elicit confrontation naming rather than internally generated language. Therefore, future research should incorporate a conversational discourse production assessment.

## REFERENCES

- Basso, A. (2003). *Aphasia and its therapy*. New York: Oxford University Press.
- Benton, A. L., & Hamsher, K. d. (1989). *Multilingual aphasia examination*. Iowa City: AJA Associates.
- Brown, R., Sherbenou, J., & Johnson, S. (1990). *Test of nonverbal intelligence*. Austin, TX: PRO-ED Publishing.
- Caramazza, A., Basili, A. G., Koller, J. J., & Berndt, R. S. (1981). An investigation of repetition and language processing in a case of conduction aphasia. *Brain and Lang*, 14, 235-271.
- Caramazza, A., Miceli, G., & Villa, G. (1986). The role of the (output) phonological buffer in reading, writing, and repetition. *Cogn Neuropsychol*, 3, 37-76.
- Coelho, C., McHugh, R., & Boyle, M. (2000). Semantic feature analysis as a treatment for aphasic dysnomia: A replication. *Aphasiology*, 14, 133-142.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, N.J.: Lawrence Erlbaum Associates.
- Conway, T., Heilman, P, Rothi, L., Alexander, A., Adair, J., Corsson, B., and Heilman, K. (1998). Treatment of a case of phonological alexia with agraphia using the Auditory Discrimination in Depth Program. Journal of the International Neuropsychological Society, 4, 608-620.
- Durks, J., & Masterson, J. (2000). *An object and action naming battery*. East Sussex, UK: Psychology Press.
- Ellis, A. W., & Young, A. W. (1988). *Human cognitive neuropsychology*. Hillsdale, New Jersey: Lawrence Erlbaum Associates.
- Friedrich, F. J., Glenn, C. G., & Marin, O. S. M. (1984). Interruption of phonological coding in conduction aphasia. *Brain Lang*, 22, 266-291.
- Gathercole, S. E. (1995). Is nonword repetition a test of phonological memory or long-term knowledge? It all depends on the nonwords. *Memory and Cognition*, 23, 83-94.



- Gathercole, S. E., & Martin, A. J. (1996). Interactive processes in phonological memory. In S. E. Gathercole (Ed.), *Models of short term memory* (pp. 71-100). Hove, East Sussex, UK: Psychology Press.
- Goodglass, H. (1993). *Understanding aphasia*. San Diego: Academic Press.
- Kaplan, E., Goodglass, H., Weintraub, S., & Segal, O. (1983). *Boston naming test*. Philadelphia: Lea and Febiger.
- Kendall, D., Conway, T., Rosenbek, J., & Gonzalez-Rothi, L. (2003). Phonological rehabilitation of acquired phonologic alexia. *Aphasiology*, 17 (11), 1073-1095.
- Kendall, D., Nadeau, S., Conway, T., Fuller, R., Riestra, A., Gonzalez Rothi, L.J. (In Press). Treatability of Different Components of Aphasia — Insights from a Case Study.
- Kertesz, A. (1982). *Western aphasia battery*. New York: Grune and Stratton.
- Lichtheim, L. (1885). On aphasia. *Brain*, 7, 433-484.
- Lindamood, C. H., & Lindamood, P. C. (1979). *Lindamood auditory conceptualization test*. Austin, Tx: PRO-ED Publishing.
- McCarthy, R., & Warrington, E. K. (1984). A two-route model of speech production. Evidence from aphasia. *Brain*, 107, 463-485.
- McNeil, M. R. (1997). *Clinical management of sensorimotor speech disorders*. New York: Thieme.
- Nadeau, S. E. (2001). Phonology: A review and proposals from a connectionist perspective. *Brain Lang*, 79, 511-579.
- Nicholas, L. E., & Brookshire, R. H. (1993). A system for quantifying the informativeness and efficiency of the connected speech of adults with aphasia. *J Speech Hearing Res*, 36, 338-350.
- Plaut, D. C. (1996). Relearning after damage in connectionist networks: Toward a theory of rehabilitation. *Brain Lang*, 52, 25-82.
- Plaut, D. C., McClelland, J. L., Seidenberg, M. S., & Patterson, K. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychol Rev*, 103, 56-115.

- Robey, R. R., Schultz, M. C., Crawford, A.,B. & Sinner, C.A. (1999). Single-subject clinical-outcome research: designs, data, effect sizes and analyses. *Aphasiology*, 13, 445-473.
- Rolls, E. T., & Deco, G. (2002). *Computational neuroscience of vision*. Oxford: Oxford University Press.
- Rolls, E. T., & Treves, A. (1998). *Neural networks and brain function*. New York: Oxford University Press.
- Roth, H. L., Nadeau, S. E., & Heilman, K. M. (In press). Naming concepts: Evidence of two routes. *Neurocase*.
- Saffran, E. M., & Marin, O. S. M. (1975). Immediate memory for word lists in a patient with deficient auditory short-term memory. *Brain Lang*, 2, 420-433.
- Storkel, H. L. (2001). Learning new words: Phonotactic probability in language development. *J Speech Lang Hearing Res*, 44, 1321-1337.
- Vitevitch MS, Luce PA (1999). Probabilistic phonotactics and neighborhood activation in spoken word recognition. *J Memory Lang*, 40, 374-408.
- Wagner, R., Torgesen, J., & Rashotte, C. (1998). *The comprehensive test of phonological processes*. Austin, TX: PRO-ED Publishing.
- Warrington, E. K., & Shallice, T. (1969). The selective impairment of auditory verbal short-term memory. *Brain*, 92, 885-896.

**Table 1.** Subject demographic information

Subject	Age	Gender	Education Level	Duration Post Onset (mo)	Lesion Localization
1	41	F	14	26	Left putamenal hemorrhage; cavity involving entire left putamen, extending posteriorly into optic radiations, superiorly into lenticulostriate endzone, anteriorly well into anterior limb of internal capsule. Contiguous large cavity in left frontal lobe with sparing of most medial, superior, and lateral portions
	40	F	18	18	Left hemisphere anterior and middle cerebral artery (MCA) distribution infarct with dense involvement of left anterior cingulate cortex and subjacent white matter, and patchy involvement of left posterior frontal and parietal cortex including immediately subjacent white matter but sparing the deep hemispheric white matter
3	50	M	12	46	Left MCA distribution infarction involving extensive frontal and anterior temporal cortex, much of putamen, insula and lenticulostriate endzone, probably sparing posterior superior temporal gyrus
4	49	M	21	53	Left MCA distribution infarct involving operculum, extensive portions of frontal cortex and contiguous dorsal parietal cortex; temporal lobe and lenticulostriate endzone largely spared
5	61	M	14	105	6 cm anterior-posterior diameter left MCA distribution infarct involving operculum and surrounding frontal, parietal and temporal cortex, putamen, insula and posterior two thirds of lenticulostriate endzone
6	65	F	12	16	Left putamenal hemorrhage with involvement of adjacent frontal, temporal and parietal white matter.
7	48	M	12	72	Left MCA territory infarct involving striatocapsular region, insula, and extensive portions of frontal convexity cortex
8	76	M	12	120	Left MCA territory infarct involving striatocapsular region, insula, lenticulostriate endzone, and frontal convexity cortex
9	46	F	12	60	Left MCA aneurysmal rupture with associated 4 by 4 cm hemorrhage into putamen and deep frontal, temporal and parietal white matter
10	48	M	12	81	8 cm anterior posterior diameter left MCA infarct involving operculum and fronto-parietal convexity cortex extending up to anterior cerebral artery territory and deep to ventricular surface

**Table 2.** Acquisition and Retention Data for Pre- and Post-treatment testing.

Western Aphasia Battery (WAB), Boston Naming Test (BNT), Controlled Oral Word Association Test (COWA), Comprehensive Test of Phonologic Processes (CTOPP)(PA = phonologic awareness/real words)(APA = alternate phonologic awareness/nonwords) and Lindamood Auditory Conceptualization (LAC).

**Immediately post treatment termination - Pre-treatment (ACQUISITION)**

	n	mean diff	SD	p-value
WAB	10	5.70	3.80	*0.001
BNT	10	1.8	5.39	0.319
COWA	10	0.90	3.18	0.394
CTOPP-PA	8	7.88	8.77	*0.039
CTOPP-APA	8	9.38	7.25	*0.008
LAC	10	16	18.2	*0.021

**Post 3-months - Pre-treatment (RETENTION)**

	n	mean diff	SD	p-value
WAB	8	2.62	11.7	0.633
BNT	8	9.50	8.60	*0.017
COWA	8	5.12	4.22	*0.011
CTOPP-PA	7	5.43	10.8	0.233
CTOPP-APA	7	8.71	7.85	0.26
LAC	8	10.6	7.74	*0.006

\* Significant at  $p \leq .05$ ; because these are all secondary outcome measures, statistics were not corrected for multiple comparisons.

**Table 3.** Repeated probe results primary and secondary outcome measures (effect size and visual inspection)

	Primary Outcome Confrontation Naming		Secondary Outcome Discourse Production <i>Word Count</i>		Secondary Outcome Discourse Production <i>CIU</i>		Secondary Outcome Phonologic production		Secondary Outcome Nonword repetition	
	effect size	Visual inspection	effect size	Visual inspection	effect size	Visual inspection	effect size	Visual inspection	effect size	Visual inspection
Post 1-week	1.63	9/10	1.49	5/8	1.71	4/8	6.88	10/10	.95	5/6
Post 3-mos	1.53	7/8	1.40	1/6	1.41	4/6	5.73	5/8	1.12	5/6

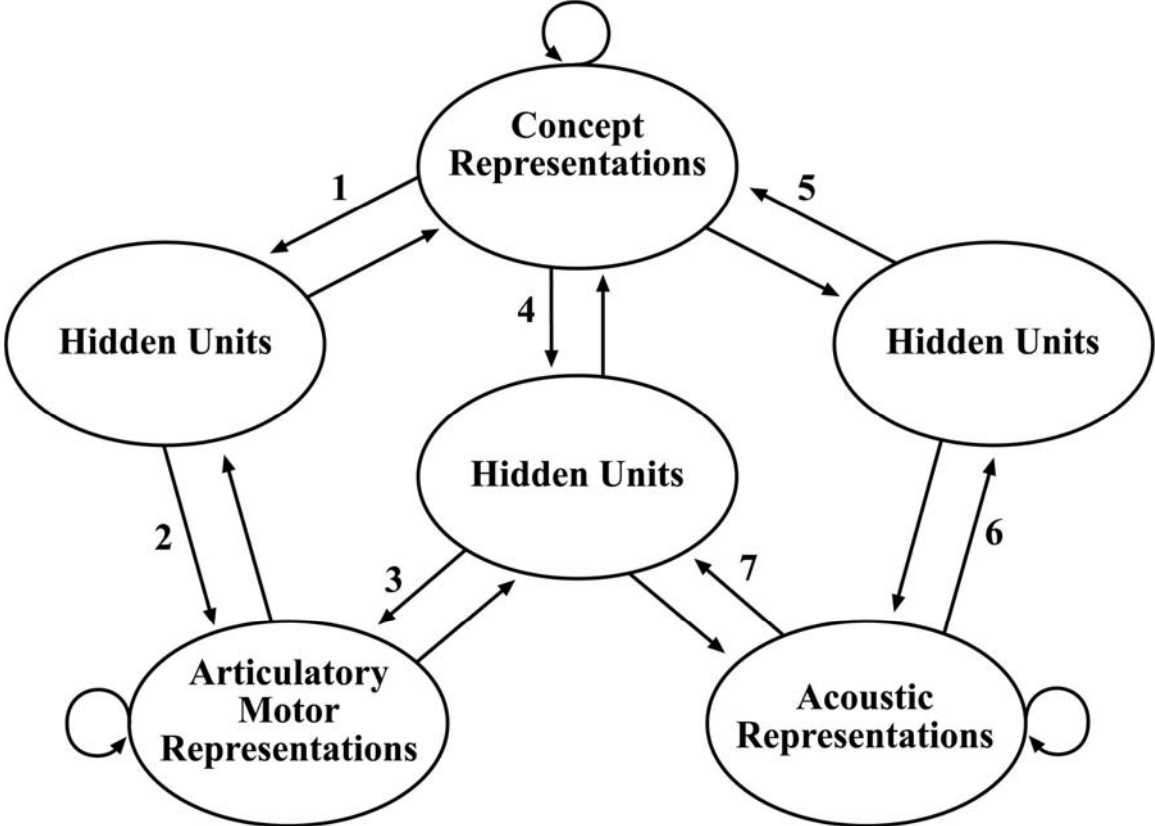
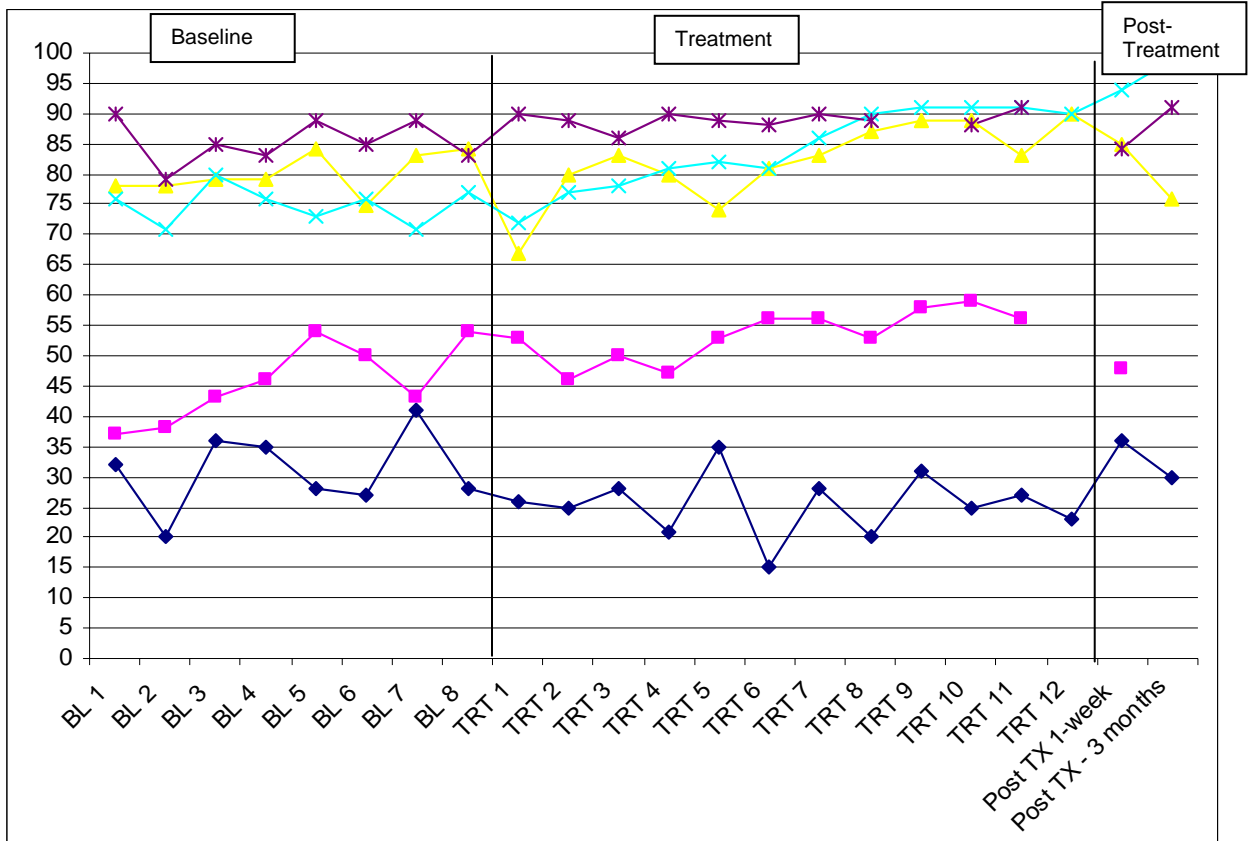
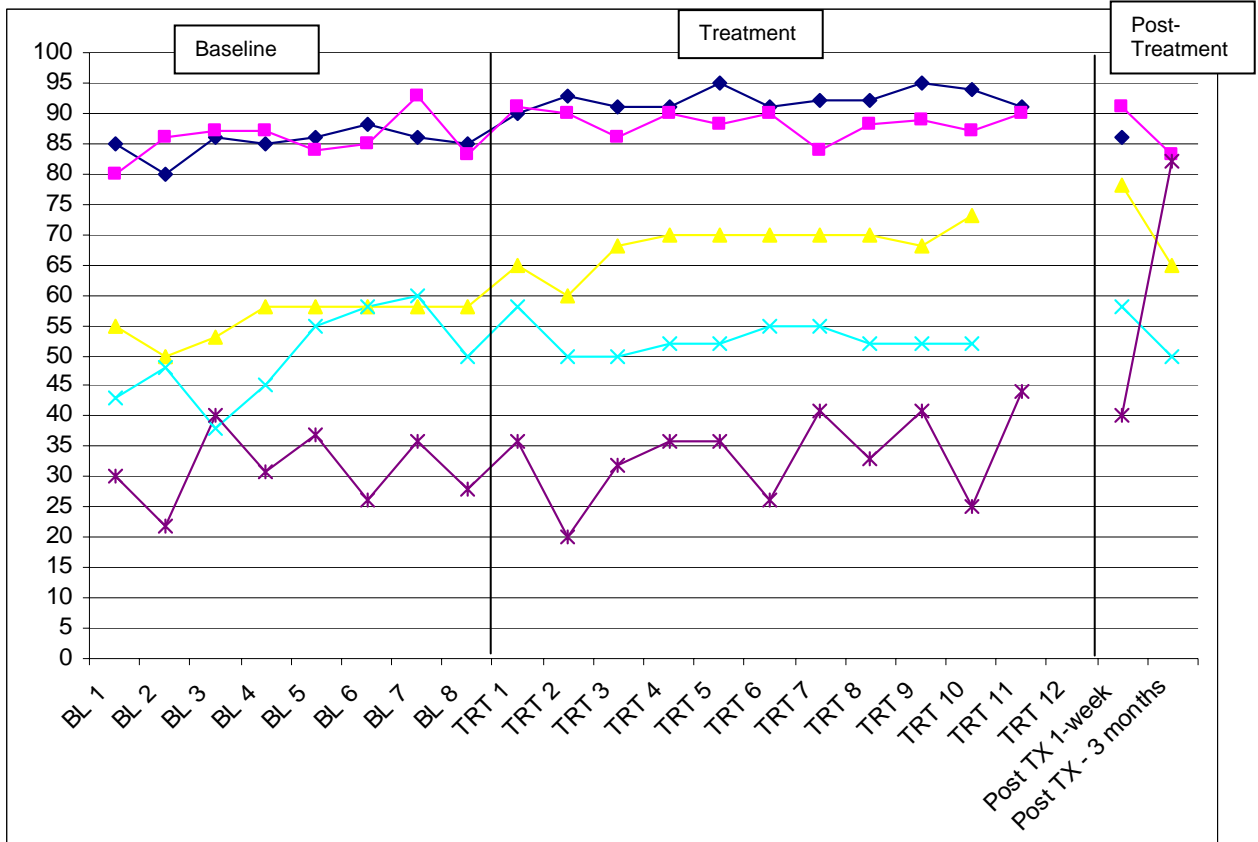


Figure 1. Connectionist model of phonological processing

**Figure 2.** Primary outcome measure – Object/Action Naming Test. Repeated probe data for patients 1 - 5. Graphs reflect percent accurate production for baseline, treatment and post-treatment phases.



**Figure 3.** Primary outcome measure – Object/Action Naming Test. Repeated probe data for patients 6 - 10. Graphs reflect percent accurate production for baseline, treatment and post-treatment phases.





## APPENDIX A: Treatment Description

### Stage I Treatment – Consonants/Vowels in Isolation:

- Background for Stage 1: Stage one includes exploration of sounds, teaching motor descriptions, perceptual, production and graphemic tasks. Stage I is performance-based. Progression to Stage II is dependent upon 80% accuracy on treatment tasks over 3 treatment sessions.
- The subject is seated at a treatment table directly across from the therapist. A mirror is placed on the table for the participant to use for visual feedback for recognition and correction of errors. Trained sounds include consonants /p,b,f,v,t,d,k,g,th,th,s,z,sh,zh,ch,j,l,r,w,h,wh,m,n,ng/ and vowels /ee,i,e,a,ae,u,o,oe,oo/. Consonant minimal pairs are represented by one picture. For example, the picture illustrated is for sounds /t/ and /d/. Voiced/voiceless contrasts are distinguished verbally by the term noisy (voiced) and quiet (voiceless). Mouth pictures are presented to the participant one at a time (e.g. minimal pair). Sounds are introduced in the following order: /p,b/, /f,v/, /t,d/, /k,g/, /th, th/, /s,z/, /s, sh/. One vowel is introduced following each minimal pair in the following order /ee, i, e, a, ae/. The sequence of tasks is described below. Following “exploration of sounds”, the perception, production and graphemic exercises are used interchangeably throughout one treatment session.
- Exploration of sounds:
  - The participant is shown one mouth picture and asked to look in the mirror and repeat after the therapist to make the sound.
  - Knowledge of results (KR) is given at 100% frequency following each production.
  - Following KR, the therapist asks the participant what they saw and felt when the sound was made. Socratic questioning is used to enable the participant to “discover” the auditory, visual, articulatory and tactile/kinesthetic attributes of the sounds.
  - Socratic questioning examples: “What do you feel when you make that sound? What’s moving? What do you see? Is it a quiet (unvoiced), or noisy (voiced) sound?”
  - Through practice and repetition the participant becomes adept at recognizing what they actually need to feel, see, hear and do to make the sound. The participant is encouraged to check themselves with their eyes (using mirror), ears and what they feel.
  - The voiced or voiceless cognate of that sound is introduced using the above steps.
- Motor description
  - A description of each sound is provided.
  - The therapist describes what articulators are moving and how they move (e.g. for /p/ the lips come together and blow apart, the voice box is turned off, the tongue is not moving).
  - The subject is asked to repeat the sound and then asked to describe how the sound was made.
  - 100% KR is provided.
  - Socratic questioning is used to probe the participant about motor description. For example, “Does your lips or tongue move to make that sound?” “Did your lips blow apart or stay together?”
- Perception Task
  - The therapist makes a sound (e.g. /p/) and asks the participant to choose that sound from an array of pictures (e.g. /f/, /g/, /p/).
  - Following each response, 100% KR is provided.
  - Socratic questioning is used for correct and incorrect responses.
- Production Tasks (used interchangeably)



- Production of sounds can be elicited auditorily (repetition), visually (mouth picture), and via motor description (e.g. make the sound where your lips come together and blow apart).
- Following each production, 100% KR is provided.
- Socratic questioning is used for correct and incorrect responses. For example, “you said /b/, is that the sound where you tongue tips at the roof of your mouth?”
- Graphemes
  - Graphemic tiles representing sounds are placed on the table with the mouth pictures. The subject is given time to review the tiles and corresponding pictures, then the therapist removes all tiles.
  - The participant is asked to select a single grapheme and place it on a picture that represents that sound.
  - When they are finished the therapist uses Socratic questioning (e.g. this letter says “/f/”, does this picture represent the sound /f/?).
  - If correct, the therapist moves onto the next letter tile, if incorrect the therapist sets aside the letter tile and moves onto the next tile. After all sounds are reviewed, only the correct letters remain on the pictures. The letter tiles that were incorrectly placed now are systematically placed on the correct picture. In order to do so the therapist says “This tile says /f/, what is working to make that sound? The participant says “lips and teeth”. The therapist says “correct” and asks what picture represents moving lips? The participant points to the mouth picture depicting /f/.
  - After the subject is able to correctly match graphemes to mouth pictures, graphemes are then used in production and perception tasks described above. For example, in a production graphemic task, the therapist would place the tile /p/ in front of the subject and ask them to produce that sound.
  - 100% KR is provided (E.g. “that is correct” or “that is incorrect”). Both correct and incorrect responses are reviewed using Socratic questioning (e.g. “What moved to make that sound?” “Is that sound noisy/quiet”)

### Stage II Treatment – Syllables

- Background for Stage II. The purpose of this stage is to extend skills acquired in Stage I to various phonemic combinations. Production, perception and graphemic tasks remain the same with the one difference that sounds are produced in combinations rather than isolation. Training progresses hierarchically (e.g. VC, CV, CVC, CCV, VCC, CCVC, CVCC, CCVCC). Upon mastery of 1-syllables, 2-syllable stimuli are composed using various combinations of 1-syllable stimuli. Only nonword sound combinations are used. Stage II is performance-based and the subject progresses through a hierarchy from 1-, 2- and 3-sound one-syllable words to 2-syllable combinations based on 80% accuracy over 3 treatment sessions.
- Perception Task
  - The therapist produces a sound combination. Depending where the subject is in the treatment hierarchy, the sound combination could be VC, or VCC-VC).
  - The therapist asks the participant to arrange pictures or graphemes to depict the target.
  - For example, if the subject heard VC (ip), they would select the graphemes /i/ and /p/.
  - Following each response, 100% KR is provided.
- Production Task
  - The therapist shows a mouth picture or grapheme tiles and asks the participant to produce those sounds individually, then blended. For example, the participant would say “/p/ /ee/ /f/” that says /peef/.
  - For both correct and incorrect responses, Socratic questioning is used. In this example, the therapist says “You said /peef/, does that match these letters?”
  - Next, the therapist changes one sound in the word (e.g. /peef/ changed to /feef/). The participant is cued to say the old word by touching each sound individually,

- then identifying the new sound and blending the new word (e.g. the old word says /p/ /ee/ /f/, /p/ was removed and /f/ was added, the new word says /feef/).
- Making one sound change is done for a series of 5-10 nonwords.
  - Subjects progressed through the treatment hierarchy based on performance. All subjects terminated therapy at 96 hours.