

The Nature of Error Consistency in Apraxia of Speech and Aphasia with Phonemic Paraphasia

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

Doctor of Philosophy

University of Washington

2015

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Abstract

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Effective treatment programs for communication disorders are based on the underlying nature of the impairment; therefore, accurate diagnosis is critical. In some cases, however, reliable and valid methods of differentially diagnosing disorders with similar behavioral profiles are lacking. This is particularly true of acquired apraxia of speech (AOS) and aphasia characterized by frequent occurrences of phonemic paraphasia (PP). The differential diagnosis of AOS and aphasia with PP is challenging because both disorders result from left hemisphere stroke and share clinical characteristics. Therefore, the identification of characteristics that pattern uniquely to each disorder is important. One way in which to strengthen the current diagnostic process is to examine the validity of diagnostic criteria used to inform differential diagnosis. The current criteria proposed to differentiate AOS from aphasia with PP include: 1) slow speech rate characterized by prolonged segment and intersegment durations, 2) sound distortions, 3) distorted sound substitutions, 4) prosodic abnormalities, and 5) relatively consistent errors in regard to error location and error type. Of these characteristics, error consistency is the most controversial. Error consistency refers to whether or not errors are relatively consistent from trial to trial in regard to the location of errors within a word (e.g., word initial) and the type of errors produced (e.g., distortions vs. substitutions). Investigations comparing the nature of error consistency in AOS and aphasia with PP have

revealed conflicting results. These studies, however, differ in important methodological areas, making it difficult to draw conclusions about the nature of error consistency in these two populations. Furthermore, previous studies suggest that error consistency may be influenced by a number of variables, such as error rate, severity of impairment, and stimulus presentation condition. This study sought to further examine the nature of error consistency in a group of 10 individuals with AOS and concomitant aphasia and a group of 11 individuals with aphasia with PP. Specifically, this study examined group differences in the consistency of error location and error type during the repetition of two-, three-, and five-syllable words. The influence of error rate, severity of impairment, and stimulus presentation condition on measures of error consistency was also examined, as well as group differences in the types of errors produced. Results suggest that consistency of error location does not differentiate group performance, whereas the variability of error type does. In particular, individuals with AOS and aphasia demonstrate more variable errors compared individuals with aphasia with PP. Results also indicate that the consistency of error location is influenced by error rate and severity of impairment. Stimulus presentation condition, however, did not appear to influence group performance on either measure of error consistency. Lastly, results of an error type analysis show that individuals with AOS and aphasia demonstrate significantly more phonetic errors compared to individuals with aphasia with PP. In conclusion, results do not support the use of error consistency as a valid measure in which to differentiate individuals with AOS and aphasia from individuals with aphasia with PP.

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Acknowledgements

This dissertation was completed with grateful appreciation to the National Institute on Deafness and Other Communication Disorders of the National Institutes of Health (award number F31- DC 1394701).

Completion of this doctoral dissertation was possible with the support of several people. I would like to express my sincere gratitude to each of them. First and foremost, I am extremely thankful to my mentor, Dr. Diane Kendall. I have had the privilege of working with Diane for a number of years in different capacities, first as a masters student at the University of Florida – she was my instructor, clinical supervisor, and primary mentor on my masters thesis committee – and as a doctoral student at the University of Washington. From day one Diane inspired me to work hard, enjoy life, test boundaries, think deeply (and even deeper), and tackle life’s challenges with optimism and grace. I will be forever grateful to Diane for her guidance, encouragement, support, and for pushing me to do and be more than I ever knew possible. In addition to Dr. Kendall, I am thankful for the other members of my doctoral committee – Dr. Malcolm McNeil, Dr. Kristie Spencer, and Dr. Kathryn Yorkston – who have challenged, inspired, and educated me through their difficult questions, encouragement, and example of solid research practices. I am grateful to Dr. McNeil for his founding work in the area of apraxia of speech and for supporting my desire to explore and contribute to this important area of research. I will forever be indebted to Dr. Spencer for her patience, guidance, and support – both in my research and family life. I am thankful to Dr. Spencer for teaching me to be systematic in my thinking and for helping me narrow down my dissertation research focus (making this defense date possible). Lastly, I’m grateful to Dr. Yorkston, whose clinical research was my initial source of inspiration to become a researcher in the field of speech-language pathology.

Throughout my doctoral program I have had the opportunity to meet and collaborate with a number of individuals. These wonderful people not only helped me in my pursuit of a doctorate, but made the experience that much more enjoyable. First, I must thank Dr. Christina del Toro for being my “big sister” in the Aphasia Research Lab at the University of Florida and for continuing this role at the University of Washington. I am forever grateful to the members of the UW Aphasia Research Lab –

Christina del Toro, JoAnn Silkes, Rebecca Hunting Pompon, Irene Minkina, Liz Brookshire, Megan Oelke, and Wesley Allen – for their willingness to listen and talk through theory, methodology, and/or the pragmatics of data collection and analysis, as well as their continued friendship and support. I am thankful to my peers – Julie Feuerstein, Bonnie Lau, and Phil Weir –in the Department of Speech & Hearing Sciences for their friendship, numerous therapeutic coffee dates, and for sharing in this journey. I am also thankful to Dr. Mike Burns for his calming nature and insightful words of advice.

This work would not have been possible without the contributions of participants and their families and research assistants. I am incredibly grateful to the research participants and their families for contributing their time and efforts. I could not have completed this project without the hard work, focus, and attention to detail performed by the following research volunteers, Melissa Helen, Lisa Bojorback, Christi Quilligan, Trina Ross, Laura Cain, and Josie Stump. A special acknowledgement to Melissa Helen, who spent numerous hours thoroughly transcribing difficult speech samples with skillfulness and confidence, thank you for your time and dedication to this project.

Last, but most certainly not least, I am deeply grateful for the love and support of my parents, two younger sisters, and my husband. A special thank you to my Mom, Lorraine Bislick, who has been a source of inspiration through her own accomplishments, my biggest cheerleader, and a fountain of never ending love and encouragement. Finally, I am especially grateful to my loving, supportive, encouraging, and patient husband, Matthew Wilson, who has been by my side throughout my journey as a doctoral student. Thank you for believing in me and supporting my passion, for being my rock and source of comfort when things were difficult, for celebrating my accomplishments, and for always reminding me of the bigger picture.

CHAPTER 1

Introduction

Effective treatment of acquired communication disorders relies on an accurate diagnosis of the disorder so that individuals can be placed into appropriate treatment programs. Furthermore, the systematic development of effective treatment protocols relies on the inclusion of accurately diagnosed participants. Most often, a diagnosis becomes evident when a speech-language pathologist (SLP) perceptually identifies behavioral characteristics unique to a particular disorder. This diagnostic process, however, can be challenging when behavioral characteristics overlap across disorders. This is particularly the case with acquired apraxia of speech (AOS) and aphasia characterized by frequent occurrences of phonemic paraphasia (PP) (Haley, Jacks, de Riesthal, Abou-Khalil, & Roth, 2012; McNeil, Robin, & Schmidt, 1997, 2009; McNeil, Pratt, & Fossett, 2004). AOS and aphasia with PP share clinical characteristics and neurological locus of impairment, and consequently, researchers and clinicians demonstrate difficulty in distinguishing between these two disorders. The inability to accurately diagnose the presence of AOS is problematic for a number of reasons. First, misdiagnosis can lead to the inaccurate assignment of participants into experimental research studies exploring the underlying impaired mechanism in these disorders, resulting in confusing and inconsistent results. Second, misdiagnosis can lead to the inaccurate assignment of participants into experimental treatment studies, limiting the development of effective treatment protocols for AOS, as well as aphasia. Lastly, individuals who are misdiagnosed may receive inaccurate, thus ineffective treatments in clinical settings. Improvements in the differential diagnosis of AOS from aphasia with PP are warranted. Better characterization of these disorders may help to further differentiate AOS from aphasia with PP, refine current models of speech production, and influence treatment development.

Within this document AOS, aphasia, and the occurrence of PP are defined. Then, a brief history on AOS and error consistency – a controversial characteristic – is discussed. Next, models of speech production that provide support for the differentiation of AOS and aphasia with PP are described. Then, a review of the literature on error consistency, its role in the differential diagnosis of AOS and aphasia with PP, and variables thought to influence error consistency is provided. Finally, the current study including research questions, methodology, results, conclusions, and future directions are discussed.

CHAPTER 2

Background & Significance

Apraxia of Speech and Aphasia Characterized by Phonemic Paraphasia

AOS is a motor speech disorder that results from impairment in the ability to plan/program motor commands necessary for normal speech production (Duffy, 2005, 2013; Maas, Robin, Wright, & Ballard, 2008; Van der Merwe, 1997, 2009). The speech of individuals with AOS is primarily characterized by sound distortions, distorted substitutions, syllable segmentation, slow speech rate, and impaired prosody (McNeil et al., 1997, 2009; Haley et al., 2012; Staiger, Finger-Berg, Aichert, & Zeigler, 2012; Wambaugh, Duffy, McNeil, Robin, & Rogers, 2006). AOS typically results from left hemisphere cerebral vascular attack and often co-occurs with aphasia (Duffy, 2005, 2013).

Aphasia is an acquired linguistic processing disorder that affects all linguistic modalities, including expressive and receptive spoken language, as well as reading and writing (McNeil & Pratt, 2001). Individuals with aphasia demonstrate anomia or word finding difficulties, and semantic (cat → dog), phonologic (cat → mat), or mixed (cat → rat) paraphasias. Some individuals with aphasia demonstrate a greater occurrence of one error type over others (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997). In particular, aphasia with frequent occurrences of PP is believed to result from difficulties in activating and/or selecting the appropriate phonemes and phoneme sequences for speech production (Dell, 1988). Examples of PP include sequential phonologic errors, such as phonemic anticipations (e.g., barn door → darn door), perseverations (e.g., barn door → barn bore), and transpositions (barn door → darn bore) (Buckingham, 1992; Dell, 1988; McNeil et al., 2004; McNeil et al., 1997, 2009), and also nonsequential phonological errors (cat → mat). Sequential and nonsequential phonological errors, along with other characteristics of aphasia, may also be observed in individuals with AOS and concomitant aphasia. Shared behavioral characteristics and locus of impairment, along with the frequent co-occurrence of AOS and aphasia, contribute to the struggle involved in differentiating these disorders.

History of Apraxia of Speech

AOS was first described over four decades ago (Darley, as described in Duffy, 2005). Since that time the nature of the disorder, its diagnosis, and treatment have been the subject of much debate. The

controversy that historically surrounds the disorder may reflect the early definitions of AOS. Although AOS was recognized as a motor speech disorder, indistinct clinical characteristics and a scarcity of appropriate theoretical models created participant classification criteria that lacked sensitivity and specificity in diagnosing the disorder (Hula, 2008, McNeil et al., 2004). Consequently, past investigations aimed at identifying the clinical characteristics of AOS may have included inappropriate participants (e.g., individuals with aphasia with PP without AOS), thereby confounding outcomes (McNeil et al., 2004). Over the years our understanding of AOS has become more model-driven, modern experimental paradigms have been employed, and more data are available, leading to the modification of old diagnostic criteria (Ziegler, Aichert, & Staiger, 2012).

In the 1970's, the criteria used to diagnose AOS included 1) phonemic errors, with substitutions being the predominant error type, 2) trial and error groping, 3) inconsistent error type, 4) speech initiation difficulties, and 5) islands of error-free speech (McNeil et al., 1997). These characteristics consist of what are now considered to be nondiscriminatory characteristics shared by AOS and aphasia with PP, and are not specific enough to distinguish between these two populations. More recently, the diagnostic criteria for AOS have been significantly modified to more sensitively reflect a motor planning/programming deficit. The current discriminatory characteristics of AOS include 1) slow speech rate characterized by prolonged segment and intersegment durations, 2) sound distortions, 3) distorted sound substitutions, 4) relatively consistent errors in regard to error location and error type, and 5) prosodic abnormalities (McNeil et al., 1997, 2009; Wambaugh et al., 2006). Many of these characteristics have been considered typical of AOS for decades, such as prolonged segment and intersegment durations and prosodic abnormalities (Kent & Rosenbek, 1993; Odell, McNeil, Rosenbek, & Hunter, 1991; Strand & McNeil, 1996). The characteristic of distorted sound errors was established by investigations of individuals with relatively pure AOS (Odell, McNeil, Rosenbek, & Hunter, 1990; Square, Darley, & Sommers, 1982) and is supported by more recent studies of individuals with AOS and co-occurring aphasia (Haley, Bays, & Ohide, 2001; Mauszycki, Wambaugh, & Cameron, 2010a, 2010b, 2012; Shuster & Wambaugh, 2000). The characteristic of error consistency, however, is considered controversial and has been the topic of more recent debate (Haley, Jacks, Cunningham, 2013; Croot, 2002; Ziegler et al., 2012).

Error Consistency

The characteristic of error consistency reported in the AOS diagnostic criteria is made up of two components: 1) the “consistency” of error location, and 2) the “variability” of error type. Specifically, the consistency of error location refers to the consistency in which an error occurs on the same target sound within a word across repeated trials. The variability of error type refers to the variability in which the same exact error is made within the same location of a word across repeated trials (McNeil, Odell, Miller, & Hunter, 1995). Initially, error consistency was used as a clinical marker to aid in the differential diagnosis of AOS from dysarthria. In particular, Johns & Darley (1970) demonstrated that individuals with AOS produce more variable errors compared to individuals with spastic, flaccid, and/or mixed dysarthria. As a result of these findings, variable or inconsistent errors were considered a hallmark of AOS. This view, however, was later challenged when an influential study revealed that the errors produced by individuals with AOS might be more consistent than previously described (McNeil et al., 1995). In particular, McNeil and colleagues (1995) examined error consistency in a small number of carefully selected participants, including individuals with pure AOS, conduction aphasia (CA)¹, and ataxic dysarthria (AD). Findings revealed that individuals with pure AOS perform similarly to individuals with AD and produce relatively consistent errors, whereas individuals with CA were more variable in the type and location of their errors. These results motivated the change in AOS criteria from inconsistent errors to relatively consistent errors.

Since this time, subsequent investigations have examined error consistency in individuals with AOS and concomitant aphasia and yield conflicting results. Some studies suggest that errors are consistent in individuals with AOS (Mauszycki, Dromey, & Wambaugh, 2007; Mauszycki et al., 2010a, 2010b, 2012; Wambaugh, Nessler, Bennett, & Mauszycki, 2004), whereas others indicate that errors are more variable (Mauszycki & Wambaugh, 2006; Staiger et al., 2012). These studies, however, did not include a comparison group to examine the “relative” nature of error consistency. Therefore, these findings reflect the performance of individuals with varying degrees of AOS and concomitant aphasia alone, not in comparison to other populations (e.g., aphasia with PP). Furthermore, many of these

¹ The term CA describes individuals with aphasia who have impaired repetition that is characterized by PP and demonstrate relatively preserved comprehension (Goodglass, 1992). Over the years, as a result of our advancement in understanding aphasia, the field has moved away from using aphasia subtypes such as CA (Schwartz, 1984).

² Studies suggest that allophonic variations of participant production including an omission error may determine the locus of breakdown (phonetic vs. phonemic) (Buchwald & Miozzo, 2011, 2012; 4

investigations differ methodologically from McNeil et al. (1995), which likely contribute to differences in findings across studies (Shuster & Wambaugh, 2008).

In addition to the study by McNeil and colleagues (1995), only two other investigations have examined error consistency in AOS compared to aphasia with PP with the goal of examining group differences (Haley et al., 2013; Miller, 1992). These three investigations reveal conflicting results, possibly caused by differences in participant inclusion criteria and methodology. A detailed account of these studies and their findings will be discussed later under “Previous Investigations of Error Consistency”.

Models of Speech Production

Two models and a theoretical framework work provide support for the differential diagnosis of AOS from aphasia with PP and contribute to our understanding of error consistency in these two populations: Dell’s model of Interactive Activation (IA; Dell, 1986), the Directions into Velocities of the Articulators (DIVA) model (Guenther, 2006), and the Theoretical Framework of Speech Sensorimotor Control for Pathological Motor Speech Disorders (van der Merwe 1997, 2009). Although these models and this framework differ, they are also complementary. Dell’s model (1986) provides a detailed description of the linguistic network and support for the linguistic errors observed in individuals with aphasia with frequent occurrences of PP. The DIVA model (Guenther, 2006) provides insight into the development and execution of motor commands responsible for the production of fluid, rapid speech and support for the phonetic errors observed in individuals with AOS. Each of these models allow for speculation on error consistency patterns in aphasia and AOS, respectively. The Four-level Framework (van der Merwe, 1997, 2009) describes and differentiates communication impairments resulting from pathology at each level of the speech production system. Although not a testable model, this framework provides an overview of the entire speech production process, including both linguistic and motor components of speech production, and describes disorders that occur at different levels of breakdown. Below, each of the models and the framework will be reviewed in the context of how they account for the behavioral characteristics observed in AOS and/or aphasia. Finally, speculation regarding how these models provide support for error consistency in AOS and/or aphasia will be discussed.

Interactive Activation Model

Dell (1986) describes a two step-model of lexical retrieval constructed from speech error data. According to this model, the *lexical network* consists of three levels of representation: semantic, lexical, and phonemic, as well as “nodes” associated with each level. *Nodes* contain specific information associated with their corresponding level. For example, nodes at the semantic level represent semantic features, nodes at the lexical level represent words and morphemes, and nodes at the phonemic level represent phonemes marked by word position (e.g., onset, nucleus, and coda) and their associated features (e.g., place, manner, voice; feature order is not specified here). Information is transferred within the lexical network via connections within and between levels. Specifically, activation spreads to adjacent levels above and below, and can influence outcomes further down the processing stream. The level of activation varies depending on different linguistic factors, such as frequency of use or relatedness. For example, nodes that are more closely related (e.g., table and chair) have stronger connections compared to those that are not (e.g., table and guitar). All connections are facilitatory. The decay of activation results from distance and time (Dell, 1986; Levelt, 1999).

According to the model, speech production begins with the selection of semantic feature nodes that represent a concept. These feature nodes receive a “jolt” of activation that sends excitatory activation to related nodes at the lexical level (Dell, 1986). Activation then spreads from the activated lexical nodes to all the related phonemic nodes, while simultaneously spreading from the lexical level back up to the semantic nodes. The return of activation to the semantic level reinforces the selections made and in return boosts the activation of the word nodes at the lexical level that are also receiving feedback from the activated phonemic nodes. Typically, in a healthy network, the appropriate lexical node is the one most highly activated at the time of selection. The selected lexical node will send higher levels of activation to related phonemic nodes and eventually, the most highly activated phonemic nodes will be selected, linked to slots in a phonological frame, and via spread of activation connected to their corresponding feature nodes (Dell, 1986). The phonological frame contains information about the number of syllables, syllable stress pattern, and the sequence of consonants and vowels within each syllable (Dell et al., 1997). A subsequent version of the model includes an approach with several possible word shapes, this expansion accounts for the addition and omission of phonemes (Dell, 1988). After phonemic selection, or

phonemic encoding, information spreads to the articulatory network and is prepared for speech planning, programming, and execution. This model does not account for the processes involved in the articulatory network, only the retrieval of linguistic representations (Figure 1).

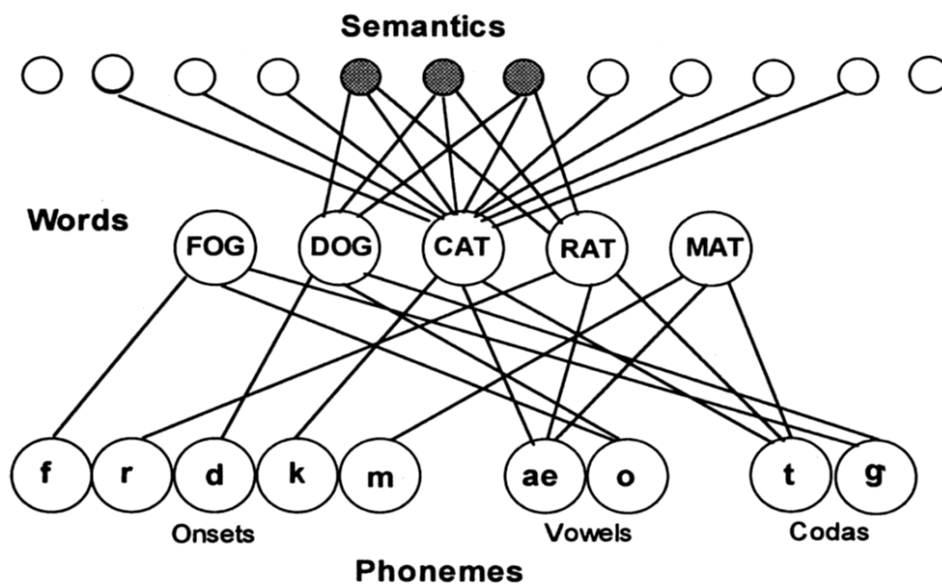


Figure 1: Illustration of the lexical network for the Interactive Activation Model. Connections are excitatory and bi-directional (figure taken from Dell et al., 1997).

For example, for the target word *cat*, the semantic nodes that represent *cat* are activated, such as *pet*, *four legs*, *fur*, *purrs*. The activated features spread activation to all related lexical nodes. Thus, the lexical node for *cat* will be activated, but so will nodes that share similar features, such as *dog*, *rat*, or *mat*. The lexical node for *cat* should be more highly activated than the nodes for *dog*, *rat*, or *mat* because it received activation from both shared and specific feature nodes (“four legs” versus “purrs”). This information spreads activation to all the phoneme nodes related to the activated lexical nodes. The phoneme nodes for *cat*, /k/, /a/, /t/, are activated, but so are the phoneme nodes for *dog*, *rat*, and *mat*. Activation from the phonemic level will flow back to the lexical level to further reinforce the selection of the target, *cat*. Once *cat* is selected, excitatory activation spreads to reinforce the selection of the phoneme

nodes for the target word, /k/, /a/, /t/, and sequence the selected phonemes into the phonological frame. Spread of activation will continue on to identify the feature nodes for each sound.

Dell's IA model is characterized by bi-directional spread of activation throughout the lexical network. As a result of this flow of information the need arises for storage or buffering during advanced planning across linguistic levels (Fry, 1969 as cited in Dell, 1986, p. 285). For example, a buffer stores higher-order information, such as semantic feature selections, while activation extends to phonologic representations. Moreover, the need for storage or a buffer may also be required at the phonological level to aid in the selection and sequencing of phonemes into the phonological frame (Dell 1986; Rogers & Storkel 1998, 1999; Shattuck-Hufnagle, 1979).

Dell (1986) and others (Crompton, 1981; Stemberger, 1983) purport that the smallest processing unit in the lexical retrieval network is the phoneme. Their claim is supported by evidence that sound level errors produced by healthy adults and individuals with aphasia are primarily phonological in nature, rather than phonetic or syllabic (Dell, 1986, 1988; Dell et al., 1997; Shattuck-Hufnagle, 1979). For example, sound level errors produced by healthy adults typically consist of phoneme deletions, additions, substitutions, or exchange errors (Blumstein, 1973; Dell, 1986; 1988; Dell et al., 1997; Shattuck-Hufnagle, 1979). These errors are nearly always phonotactically well formed (Buckingham & Kertesz, 1976; Lecours, 1982). The same can be said for individuals with aphasia with PP, but the difference between the errors produced by healthy adults and individuals with aphasia with PP is the frequency in which errors occur. This difference is attributed to the effects of brain damage, when the transmission of activation between levels and/or the representations within each level are disrupted (Dell et al., 1997).

The IA model (Dell, 1986) accounts for the occasional speech errors made by healthy adults and the more frequent errors produced by individuals with aphasia. Errors are proposed to result from interference or "noise" in the system and can occur at any level of the linguistic network – semantic, lexical, and/or phonologic. In particular, phonologic errors result from interference that leads to the activation of an incorrect phoneme during phonemic encoding (Dell, 1988). To account for phonemic errors, the model incorporates three sources of noise. The first is *spreading activation*, which permits competition during phonologic encoding. For example, recall all the other phonemes activated during "cat"

in the example above. If activation spread does not effectively go where it should, the phonological node for *mat* (versus *cat*) may receive an inaccurate jolt of activation leading to the selection of an incorrect phonological node (i.e., /m/). The second source of noise is the result of interference produced by the activation of previously spoken and upcoming words. For example, “barn door” may be produced as “darn door” because the initial consonant in “barn” was competing with the initial consonant in “door”. This type of error is referred as an anticipation error. A few other examples include perseveration (e.g., barn door → barn bore) and exchange *errors* (barn door → darn bore). Finally, unintended words can also be active during lexical retrieval by “extraneous cognition and perception” leading to competition during phonologic encoding (Dell, 1988, p. 131).

Overall, the IA model is valuable in differentiating AOS from aphasia with PP. Although the model does not extend to the level of articulation, it accounts for phonological paraphasias produced by individuals with aphasia with PP as impairment in phonological retrieval (Dell, 1988; Levelt, 1989; Shattuck-Hufnagle, 1979; and others), rather than an impairment of speech motor planning/programming.

Error Consistency. Dell's IA model allows for speculation regarding the consistency of error location and error type in individuals with aphasia with PP. The model accounts for the sound errors produced by individuals with aphasia with PP via the sources of interference or noise in the system, as discussed above (spreading activation, activation of previously spoken and upcoming words, and extraneous cognition and perception). As a result of interference and the combined effects of these different sources, the potential exists for a number of variations of phonological errors to occur within any given word, especially in an impaired network. For example, the word “telephone” could be produced correctly, it could be produced with an anticipatory (e.g., phelephone), perseverative (e.g., teletone) or transposition (e.g., pheletone) error, an omission (e.g., telepho) error, or an addition (e.g., telerphoney) error. In regard to error location, the model does not predict where an error will occur, but it does support the observation that anticipatory, perseverative, and exchange errors reflect competition of sounds located within the same word or syllable position (e.g., barn door → darn bore). Furthermore, participant data suggest that syllable- and word-initial consonants are much more likely to participate in phonological errors than final consonants, by a factor of about 5 to 1 (Stemberger, 1983, as reported in Dell, 1988).

Dell (1988) discloses that the model is unable to replicate this finding. Participant data also indicate that individuals attempt to correct errors on subsequent trials, but are not always successful (Dell et al., 1997, McNeil et al., 2009). Thus, some participants may be able to correct their errors, or produce a closer approximation of the target, suggesting more variable responses.

During phonological processing, the need arises for storage or maintenance of activation of phonological information to aid in the selection and sequencing of phonemes into the phonological frame (Dell 1986; Rogers & Storkel 1998, 1999; Shattuck-Hufnagle, 1979). The integrity of an individual's ability to store or maintain activation of phonological information (e.g., phonological buffer) may also influence error consistency. If unimpaired, activated phonemes may be maintained (or successfully stored) during consecutive productions of a stimulus. On the contrary, if activated phoneme nodes decay quickly or the ability to store phonological information is impaired, an individual may demonstrate relatively more variability across subsequent trials. Thus, the consistency in which errors are produced may not be homogenous across all individuals with aphasia.

Directions into the Velocities of The Articulators

The DIVA model (Guenther, 2006; Guenther, Ghosh, & Tourville, 2006; Tourville & Guenther, 2011) provides a computational and neuroanatomical account of speech acquisition and production. According to the model, the integration of auditory, somatosensory, and motor information is required for the development and execution of fluent speech. The model associates the information sources with specific neural anatomical locations that mediate their function (Guenther, 2006). Providing a neuroanatomical account for speech production is beneficial in that it enhances our knowledge of the neural structures involved in normal speech production and guides our understanding of the level and nature of breakdown observed in clinical populations. The DIVA model does not provide information about higher-level linguistic processes, such as semantics and syntax. Instead, it concentrates on the learning and interaction between “sound” representations (e.g., phonemes, syllables, and/or words) and motor commands.

The model consists of two control subsystems: *feedback control and feedforward control*. These control subsystems develop in infancy, during the time when babbling and early word imitation emerges

(learning is explained in more detail below). *Feedback control* is established first and aids in the development of feedforward control. Eventually, the feedforward control subsystem becomes independent, acting as the sole operator of the speech programming system with minimal reliance on feedback control. Feedforward control consists of learned feedforward motor commands responsible for the production of fluent speech. In addition to learned motor commands, the feedforward control subsystem consists of a speech sound map, articulator velocity and positions maps, and an initiation map. These maps refer to a group of neurons in the brain that are active during specific processes involved in the learning and production of speech. The left frontal operculum, cerebellum, thalamus, motor cortex, supplementary motor area, and the basal ganglia mediate the feedforward control subsystem (Figure 2).

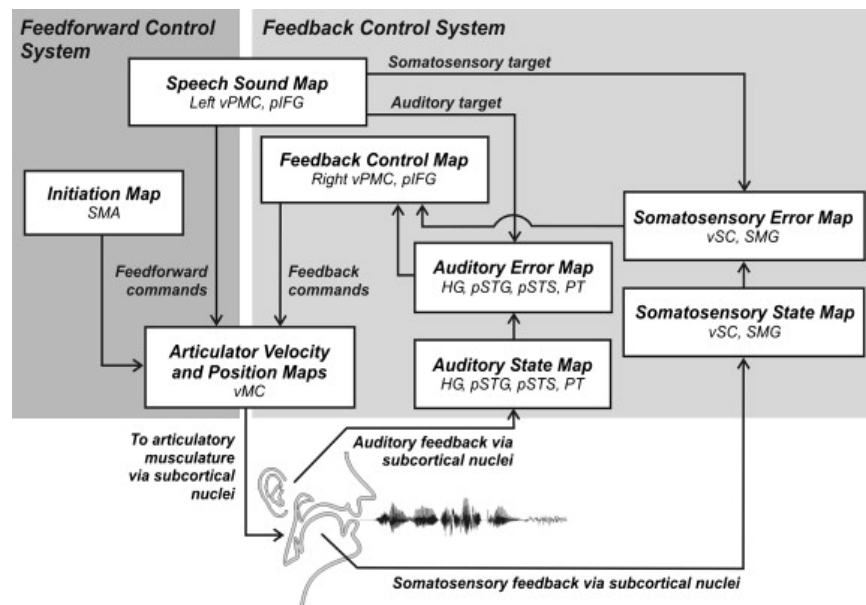


Figure 2: Schematic of the cortical components of the DIVA model. Each box in the diagram corresponds to a set of neurons, or map, and arrows correspond to synaptic projections that transform one type of neural representation into another (adapted from Guenther & Vladusich, 2012). Abbreviations: HG = Heschl's gyrus; pIFG = posterior inferior frontal gyrus; pSTG = posterior superior temporal gyrus; PT = putamen; SMA = supplementary motor area; SMG = supramarginal gyrus; vMC = ventral motor cortex; vPMC = ventral premotor cortex; vSC = ventral somatosensory cortex.

Feedback control consists of auditory and somatosensory feedback. The feedback control subsystem is responsible for the development of auditory and somatosensory target representations for sound production and perception, as well as error detection. Specifically, the model includes separate

auditory and somatosensory target maps, state maps, and error maps. These maps correspond to groups of neurons in the brain that represent the auditory or the somatosensory target representations for specific sounds (target maps), the sensory state of an active sound recently produced or perceived (state maps), and the error detected between the target representation and the active sound (error maps). The superior temporal cortex and parietal cortex mediate auditory and somatosensory control, respectively. The cerebellum and the thalamus are also active during these processes. Additionally, a feedback control map, located in the right frontal lobe, projects error correction information to the feedforward control system (Tourville & Guenther, 2011).

According to the DIVA model, the speech sound map mediates both feedforward and feedback control processes (Tourville & Guenther, 2011; Guenther & Vladusich, 2012). The speech sound map contains cells, or groups of neurons, that correspond to individual speech sounds. These sound representations are initially learned via interaction with the feedback control subsystem and are later involved in the feedforward of learned motor commands. The cells in the speech sound map represent phonemes, syllables, words, and phrases (Tourville & Guenther, 2011).

In an unimpaired, adult speaker, the process of speech production starts with the activation of a speech sound map cell in the left premotor and adjacent inferior frontal cortex. Once a cell is activated, the corresponding feedforward command projects to cells in the articulatory velocity map located in the motor cortex. It is here that the motor commands for articulator movements are encoded (Guenther & Valdusich, 2012). The transfer of information from the speech sound map to the articulator velocity map is comparable to phonetic encoding (i.e., the transfer of abstract representations to movement goals for the articulators; Levelt & Wheeldon, 1994; Levelt, Roelofs, & Meyer, 1999; Tourville & Guenther, 2011). When activated, a speech sound map cell will also project to the auditory and somatosensory target maps in the feedback control subsystem (Tourville & Guenther, 2011). These projections encode the sensory targets associated with the activated speech sound map cell and allow the system to monitor and make comparisons to the output (discussed below). Next, speech is consciously initiated and information is sent from the articulator velocity map (and articulator position map) to the articulatory musculature via the

subcortical nuclei for speech motor programming. The DIVA model does not provide a detailed account of speech execution.

After the message is articulated, auditory and somatosensory feedback is processed by the feedback control subsystem. If the produced stimulus falls within the acceptable range of expectations (i.e., a clearly recognized target), the auditory and somatosensory state maps send inhibitory inputs to auditory and somatosensory error maps, respectively. These error maps monitor speech production and represent the difference between expected and actual sensory states. If, however, a speech error occurs, such as a sound distortion, an error signal will be sent to the feedback control map (Tourville & Guenther, 2011). The feedback control map will convert the error signal into corrective commands, which will integrate with feedforward commands in the articulator position maps. The cells located in the articulator position maps correspond to the movement parameters of the articulators (Tourville & Guenther, 2011).

Learning. The connections between the speech sound map and the feedforward and feedback control subsystems are learned in infancy through exposure and practice. According to the DIVA model, learning how to speak begins first with babbling and then second with early word imitation. During the babbling stage, auditory and somatosensory feedback commands are tuned via random movements of the articulators, resulting in articulatory, auditory, and somatosensory activation. The interaction of articulatory, auditory, and somatosensory information is used to tune the connections between sensory error maps and the motor cortex. The sensorimotor transformation learned at this stage is vital to the learning of speech sounds during imitation (Figure 3a).

Early word imitation supports the model in learning specific sound representations. During this stage projections from the speech sound map to the articulator velocity map are tuned via exposure and practice. This process is likened to how a child learns to speak. For example, a child learns the auditory target for /ba/ through repeated auditory exposure to a correctly produced /ba/ from the child's parent or caregiver. As the child attempts to copy the parent's production, the auditory error map provides information used to create and update the feedforward motor commands for /ba/ so that future productions more closely match the stored auditory target. The projections from the speech sound map to the somatosensory target map are also tuned at this stage (Figure 3b). The cerebellum plays an

important role in this tuning process. Sensory error representations located in the cerebellum drive corrective motor commands and contribute to feedback-based motor learning (Tourville & Guenther, 2011). The corrective commands issued by the feedback control subsystem are stored in the cerebellar feedforward commands, to be used on the next speech attempt. Subsequent attempts to produce the sound result in improved feedforward commands (Guenther, 2006). Eventually, the feedforward command is capable of producing the sound without any auditory error (Spencer & Slocumb, 2007). When the sound can be produced in a fluent (e.g., smooth) and coordinated manner, the feedback control subsystem is no longer needed to guide speech production. From this point on, feedforward commands are sufficient to produce the sound on its own during normal speech. The cerebellum, however, continues to play an important role in the maintenance of learned feedforward motor commands and is hypothesized to guide the selection of motor commands (Tourville & Guenther, 2011).

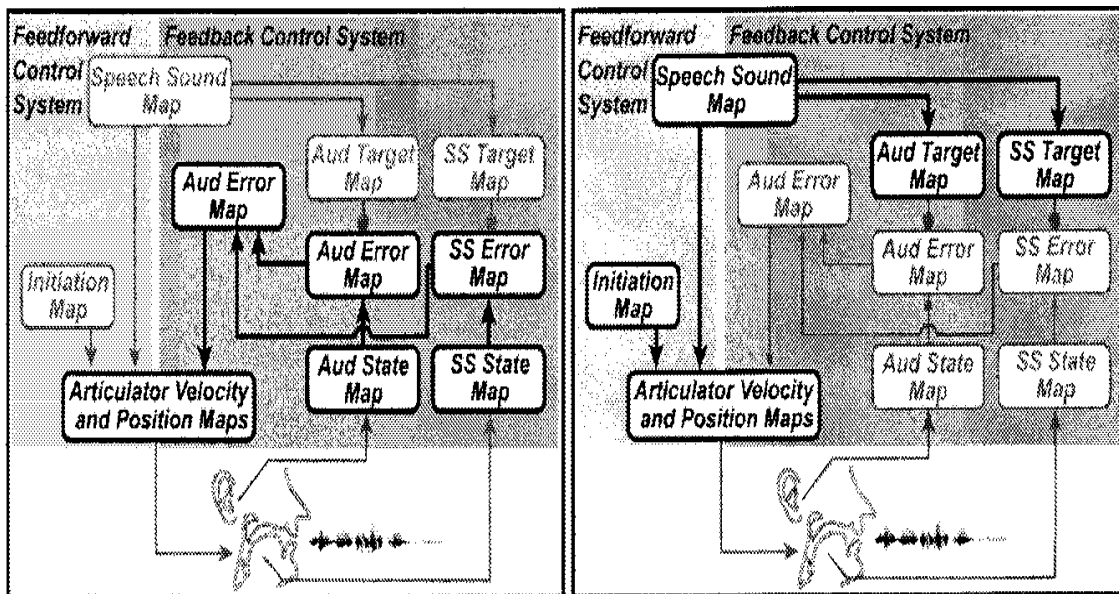


Figure 3a and 3b. The DIVA model and learning. The figure on the left represents learning during the babbling phase and the figure on the right represents learning during the imitation phase (figure taken from Tourville & Guenther, 2011).

A breakdown in the components of the feedforward or feedback control subsystems would result in impaired production or acquisition of speech. A small but growing body of literature has focused on the ability of the DIVA model to account for the speech characteristics of individuals with motor speech

disorders. Specifically, investigations have begun to explore the DIVA model's ability to account for acquired AOS (Jacks, 2008; Maas, Mailend, & Guenther, 2012, 2015; Mailend & Maas, 2013), but not the dysarthrias. Speculations about ataxic dysarthria have been made based on neuroanatomical and behavioral research (Tourville & Guenther, 2011).

In the extant literature, three different hypotheses have been proposed in regard to how the DIVA model may account for the behavioral characteristics of AOS. These hypotheses include: 1) impairment to the feedforward control subsystem (Jacks, 2008; Mass et al., 2012, 2015), (2) impairment to the feedback control subsystem (Ballard & Robin, 2007), and/or (3) damage to the speech sound map (Bohland, Bullock, Guenther, 2009; Mailend & Maas, 2013). Of these hypotheses, impairment to the feedforward control mechanisms is the strongest explanation for the characteristics observed in AOS. More specifically, feedforward control mechanisms are responsible for the production of rapid movements that are relatively consistent from trial to trial. Impaired feedforward control would result in inaccurate or "noisy" motor commands and increase reliance on feedback control (Jacks, 2008; Maas, et al., 2012; Tourville & Guenther, 2011). This reliance on feedback control is likened to that of speech development, where prior to the construction of feedforward commands, speech production is slower and more variable across repeated trials of the same behavior (Jacks, 2008; Schmidt & Lee, 2005). Thus, reliance on feedback control as a result of impaired feedforward commands for speech production may account for several of the characteristic features of AOS, "including inaccurate but perceptually recognizable movements" (e.g., sound distortions) and increased segment and intersegment durations resulting in slowed speech rate (Jacks, 2008). Slowed speech rate may also lead to the perception of abnormal stress and prosody. The degree to which the feedforward control subsystem is impaired would likely determine the extent to which the feedback control subsystem is involved, which may reflect the severity of speech impairment. For example, feedforward control may be more impaired in individuals who produce more frequent speech errors and impaired rate, compared to individuals with infrequent speech errors and subtle rate defects.

The hypothesis that AOS results from impairment to the feedback control subsystem suggests that inefficiencies arise in the integration of corrective auditory feedback and feedforward commands

(Ballard & Robin, 2007). Thus, impaired feedback control would result in an inability to learn new motor commands, make adjustments to feedforward commands based on changes in the environment, and correct errors. This hypothesis is unlikely to explain AOS for a number of reasons. First, this hypothesis indicates that feedforward control is intact in an adult speaker with acquired AOS, which does not explain the difficulty observed in the production of motor commands learned prior to brain damage. Second, there are many reports that treatment can lead to improved speech in AOS, suggesting that the construction of new motor commands or correction/updating of impaired commands is indeed possible (Bailey Eatchel, Wambaugh, in press; Wambaugh et al., 2006). Additionally, participant data indicates that individuals with AOS can make adjustments during articulatory perturbation, implying that individuals with AOS are able to update and modify feedforward commands based on changes in the environment (Jacks, 2008). It is important to note, however, that many individuals with AOS still demonstrate some difficulty post treatment. It is unclear if residual deficits can be improved upon with additional treatment(s) or if there is some degree of impairment to the overall development or modification of impaired feedforward commands post brain damage.

The hypothesis that AOS results from an impaired speech sound map suggests that difficulty occurs in the activation or retrieval of abstract sound representations (Bohland, et al., 2009; Maas, et al., 2012; Mailend & Maas, 2013). In the context of the DIVA model, difficulty activating the appropriate cell in the speech sound map would result in substitution errors, groping, and may also account for slowed speech as a result of delayed activation (Bohland et al., 2009; Mailend & Maas, 2013). This hypothesis does not provide clear support for sound distortion errors, although it has been argued that distortions and distorted substitutions may reflect the co-activation of multiple speech targets (see Goldrick & Blumstein, 2006; Mailend & Maas, 2013). Furthermore, this hypothesis does not differentiate the errors observed in AOS from those of aphasia with PP (in fact, the contents of the speech sound map can be likened to that of Dell's abstract phonological representations, therefore damage to the speech sound map is suggestive of aphasia with PP). A recent investigation by Mailend & Maas (2013) suggests that some speakers with AOS demonstrate difficulty in the retrieval of sound representations in addition to feedforward control, though findings may also reflect disruptions in phonological activation, i.e., aphasia.

Error Consistency. The hypothesis that AOS results from damage to the feedforward commands for speech production suggests that individuals with AOS demonstrate some degree of variability across trials (e.g., not 100% consistent). Impaired feedforward control results in “noisy” motor commands and an overreliance on feedback control, which is not as sophisticated as feedforward control and leads to slower and more variable speech production across repeated trials (Jacks, 2008; Mass et al., 2012, 2015; Schmidt & Lee, 2005). This hypothesis, however, does not explicitly address the consistency of error location in individuals with AOS. Extent of damage to the feedforward control subsystem may be one factor that contributes to the manner in which errors occur. For example, it may be possible to successfully access and implement some feedforward commands, therefore only relying on the feedback control subsystem for feedforward commands that cannot be successfully implemented. The complexity of the speech task, stimuli, or part of speech may also contribute to error consistency. For example, more complex sounds or words (e.g., singletons vs. clusters, monosyllabic vs. multisyllabic words) may tax the feedback control subsystem and result in more consistent and/or predictable speech errors. Previous studies suggest that individuals with AOS demonstrate more frequent errors on fricatives compared to stops (Johns & Darely, 1970; LaPointe & Johns, 1975; Odell et al., 1990), clusters compared to singletons (Aichert & Ziegler, 2008; Buchwald & Miozzo, 2012), trisyllabic compared to monosyllabic words (Marquardt, Schneider, & Jacks, 2010; Strand & McNeil, 1996), and word onsets compared to other locations within a word (Odell, 2002; Staiger et al., 2012). Thus, it is possible the consistency of error location is influenced by the complexity of the sound, word, or word position. With regard to variability of error type, the impaired feedforward hypothesis identifies sound distortion errors as the most frequently produced sound error in AOS, suggesting some consistency in error type patterns across repeated trials (participant data support this claim; Mauzycki et al., 2010a, 2010b, 2012; Odell et al. 1990; Shuster & Wambaugh, 2008).

Four-Level Framework of Speech Production

The Theoretical Framework of Speech Sensorimotor Control for Pathological Motor Speech Disorders (van der Merwe 1997, 2009) was constructed to explain and differentiate communication impairments resulting from pathology at each level of the speech production system. Van der Merwe (2009) combines brain imaging data with behavioral outcomes to describe the speech production

processes in its entirety, starting with the activation of abstract linguistic information and ending with the execution of successfully timed speech movements. Van der Merwe's description of higher-order linguistic mechanisms is not as detailed as Dell's (1986). However, the Four-level Framework highlights neural structures thought to be involved in the process, whereas the IA model does not. What sets van der Merwe's framework apart from the previously described models is its detailed description and separation of the processes involved in the organization and execution of speech. The framework includes four phases of speech production: (1) linguistic-symbolic planning, (2) motor planning, (3) motor programming, and (4) execution (Figure 4). See Appendix I for the full version of the framework (taken from van der Merwe, 1997).

A simplified view

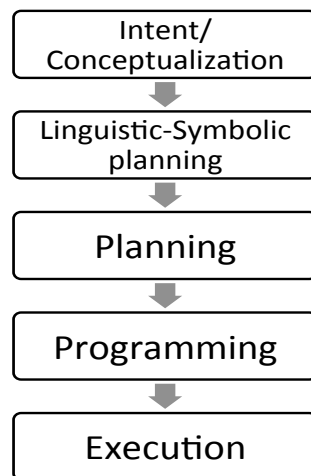


Figure 4. A simplified view of the Theoretical Framework of Speech Sensorimotor Control for Pathological Motor Speech Disorders.

The pre-motor phase, *linguistic-symbolic planning*, includes the organization of linguistic processes according to the linguistic rules of the language. At this phase, semantic, lexical, morphological, and phonological planning occurs. Similar to the IA model (Dell, 1986), van der Merwe's framework specifies phonemes as the smallest unit of the pre-motor phase and describes phonologic

planning as the selection and sequencing of phoneme combinations (Dell, 1986; van der Merwe, 1997, 2009; Shattuck-Hufnagle, 1979). This phonologic plan is “invariant,” in that any change during this process results in alterations to the meaning of the message (van der Merwe, 2009, p.8). For example, during phonological planning, the abstract phonemic representations for the target, *book*, are selected and sequenced correctly for production. If an error occurs and the incorrect phoneme is selected, (e.g., /b/ → /t/), the meaning of the word is changed, (e.g., /book/ → /took/). According to the framework, the temporal-parietal areas mediate linguistic symbolic planning. In particular, Broca and Wernicke areas are highly activated during this phase of speech production.

Following the linguistic-symbolic phase, abstract phonological representations are transformed into a code readable by the motor speech system. This transformation of information occurs across three subsequent phases: motor planning, motor programming, and execution. These phases represent a sensorimotor hierarchy in which movement control is exerted through highest, middle, and lowest levels. The highest level in the sensorimotor hierarchy is speech motor planning.

Speech motor planning begins by defining motor goals. Each speech sound (i.e., phoneme) has a core motor plan, which contains a number of motor goals. The motor goals for speech production are defined by their spatial and temporal properties. In other words, the motor goals specify place, manner, voice, and timing of movement for each sound. For example, the motor plan for the phoneme /b/ includes three individual motor goals: lip closure, velar lifting, and vocal fold closure. The combination of these motor goals makes up the core motor plan for this particular sound. Thus, motor planning is articulator-specific, not muscle-specific. In other words, the motor plan specifies the act to be performed by the articulators (e.g., lip rounding), but not the actual muscle movements (see Tourville & Guenther, 2011).

During speech development motor plans are attained and perfected via somatosensory and auditory feedback, and are stored in sensorimotor memory (van der Merwe, 1997; 2009) (similar to, but not as detailed as the DIVA model; Guenther, 2006). During speech production motor plans are recalled from sensorimotor memory and the different motor goals are identified and arranged sequentially. The selected motor plans are then modified according to the context in which the sound is to be produced. At this stage, the potential for co-articulation is created, as well as segmental duration and interarticulatory

synchronization. For example, the motor goals for /b/ in “book” versus /b/ in “big” differ in that the lips are more rounded for “book”. Adaptation of the core motor plan is based on internal feedback from stored representations learned through practice and exposure. Once completed, the articulator-specific motor plan is systematically relayed to the motor programming phase.

Broca’s and Wernicke’s areas, the prefrontal cortex, the motor association area, the supplementary motor area, and the parietal association areas are thought to mediate the transformation of abstract phonological representations into motor plans. The involvement of “non-motor” areas such as Wernicke’s area and the parietal areas likely indicate the communication of sensory information.

Motor programming involves the transformation of articulator-specific motor plans into muscle commands or programs that are sequenced before movement begins (Mardsen, 1984, as cited in van der Merwe, 2009, p. 6). At this stage, the tone, movement velocity, direction, force, and range of muscle movements are specified (i.e., movement parameters). For example, when reaching for a cup of coffee the motor plan for this action would include extending the arm toward the cup, opening the hand, and placing the fingers around the cup or perhaps around the handle, and then moving the arm back toward the body and lifting the cup to the mouth. The motor program for this action specifies the spatiotemporal properties of the muscles responsible for the movements of the arm, hands, and fingers. These specifications determine the rate of movement, muscle force required when gripping the cup, and the tension required to steady the cup as it is moved back towards the body and up to the mouth. The term motor programming (used here) shares similar characteristics with the responsibilities of the articulator velocity and position maps described in the DIVA model, as both determine movement parameters for the muscles of the articulators (Tourville & Guenther, 2011).

Motor programming is mediated bilaterally by mid-level neural structures, unlike motor planning, which is mediated by cortical association areas in the dominant hemisphere. According to van der Merwe (2009), the basal ganglia and the lateral cerebellum are especially important in motor programming. The behavioral characteristics observed in individuals with Parkinson’s disease and cerebellar disease, respectively, support this claim. The supplementary motor area and frontolimbic system are also involved

at this stage and are believed to play a role in the initiation and feedforward of co-occurring and successive motor programs.

After the articulator-specific motor plans have been retrieved and sequenced, and the motor programs for muscle movement have been selected, it is time to execute (articulate) the prepared message. During the execution phase plans and programs are transformed into actual movements. Specifications are relayed to the lower motor centers that control the joints and muscles through the final common pathway and are translated into timed commands for muscle movements. After the message is articulated external feedback, somatosensory and auditory information, is provided. External feedback can be used to update motor programs when necessary, as in the case of a speech error or changes to the external environment (i.e., jaw perturbation).

The motor cortex, lower motor neurons, peripheral nerves, and the motor units in the muscle mediate the *execution* of movements for speech production. Additionally, the supplementary motor area, cerebellum, basal ganglia, thalamus, and brainstem play a role in the control of movement. The supplementary motor area, cerebellum, and the basal ganglia also mediate motor programming. Thus, damage to one of these structures may indicate impairment at more than one level of the speech production hierarchy.

Through the delineation of the four phases of speech production (linguistic-symbolic planning, motor planning, motor programming, and execution), van der Merwe (1997, 2009) separates AOS from its clinical neighbors. The model suggests that impairment to linguistic-symbolic processes would result in aphasia, including disruptions in semantic, lexical, syntactic, morphological, and/or phonological planning. In particular, deficits at the level of phonological planning would occur from impairment in the selection and sequencing of the abstract phonological representations, resulting in frequent occurrences of phoneme substitutions and transposition errors (i.e., PP). Dell (1986, 1988) and colleagues (Dell et al., 1997) provide a detailed description of the errors produced by individuals with aphasia with PP, as discussed above. For example, individuals with aphasia with PP may select an incorrect phoneme during phonological planning (e.g., /phone/ → /pone/). Deficits in phonological planning may result in neologisms or real words, and can change the meaning and/or the intelligibility of the message being communicated.

Importantly, errors occurring at this phase result in sound substitutions not sound distortions. This framework supports that the characteristics observed in aphasia are linguistic, such as transposition errors, and do not overlap with the characteristics observed in AOS. It is important to point out, however, that in reality sound distortion errors are sometimes perceived as substitutions, making it difficult to discriminate between errors of motor and phonological planning, and complicating differential diagnosis (Duffy, 2005; Odell et al., 1990; Trost & Canter, 1974).

According to van der Merwe (1997, 2009), AOS occurs from deficits to the neural areas involved in speech motor planning. Disorders of motor planning may result in an inability recall the core motor plans and/or identify the motor goals for speech sounds, successfully sequence the movements for individual speech sounds and/or a group of sounds (syllable, words), adapt core motor plans, and control inter-articulatory synchronization. Impairment at this level may also result in an inability to successfully relay the articulator-specific motor plan to the motor programming phase. The behavioral characteristics thought to result from these inability include articulatory groping, effortful production, sound distortions, apparent substitutions, and slowed speech rate. Slowed speech rate may result from inter- and intra-syllabic pausing, and may also lead to perceived abnormal stress and prosody. In regard to distorted substitutions, van der Merwe (1997) suggests distorted substitutions result from deviant phonological selection at the linguistic-symbolic level with a subsequent problem at the motor planning stage. Thus, suggesting that distorted substitutions reflect a combination of linguistic and motoric deficits (e.g., AOS and co-occurring aphasia). Distorted substitutions have been observed in individuals with pure AOS (Odell et al., 1990), therefore indicating that these errors may reflect an extreme distortion that crosses phonemic boundaries and does not involve a linguistic selection error. The Four-Level Framework description of a disorder of motor planning does not incorporate true phonemic substitutions, nor does it allow for phoneme transposition errors. These errors are explained as a result of impaired phonological planning and support the claim that AOS does not result from linguistic deficits. It is important to recognize that these phases of speech production (i.e., linguistic-symbolic planning and motor planning) share neural structures, and therefore it is common for disorders of linguistic-symbolic planning and motor planning to co-occur.

The framework implies that damage at each phase of speech production may result in an isolated disorder resulting from that particular phase. This separation becomes especially difficult when talking about disorders of motor programming and execution as these phases of speech production are controlled by many of the same neural structures, such as the basal ganglia, lateral cerebellum, and the supplementary motor area. This overlap suggests the possibility of dual symptomology in some of the disorders that occur at the lower levels of the sensorimotor hierarchy. For example, hypokinetic, hyperkinetic, and AD, and in some cases spastic and UUMN dysarthria are considered disorders of motor programming and execution. Flaccid dysarthria, however, results from damage to the lower motor neurons, and is considered an isolated disorder of the execution phase.

Disorders of speech motor programming result from impaired programming of muscle tone, velocity, direction, and range of movement. Additionally, impairment in the ability to control initiation and feedforward of co-occurring and successive motor programs can occur at this phase. The behavioral characteristics of motor programming disorders include sound distortions, abnormal rate, and difficulty initiating movements for speech. Disorders of speech execution (i.e., flaccid dysarthria), result from weakness and reduced muscle tone. The behavioral characteristics of flaccid dysarthria depend on the severity and location of lower motor neurons damage. Some characteristics may include imprecise sound production, breathy voice, hypernasality, and the production of short phrases.

The primary characteristics of AOS are similar to the characteristics observed in disorders of motor programming and execution. In particular, AD and UUMN dysarthria are difficult to differentiate from AOS (Duffy, 2005, 2013). The differential diagnosis of AOS from AD is challenging because the disorders share many characteristics, including sound distortions, slow rate, and abnormal prosody. One characteristic they do not share however, are distorted substitutions (Duffy, 2005, 2013). Additionally, AD results from impairment to the cerebellum, whereas AOS typically results from left hemisphere cortical damage. AOS and UUMN dysarthria typically result from left hemisphere lesions and often co-occur (UUMN dysarthria can also result from right hemisphere damage; Duffy, 2005, 2013), but do not share a large number of deviant characteristics. When these disorders co-occur, however, it is difficult to attribute specific errors to one versus the other (Duffy, 2005, 2013).

The deviant characteristics shared by AOS and the dysarthrias result from impairment to different mechanisms and should at least have subtle differences (van der Merwe, 2009). For example, sound distortions in AOS likely result from failure to sequentially organize movements or successfully adapt the core motor plan for a sound. Sound distortions observed in disorders of motor programming likely occur from impaired programming of muscle tone, direction, or range of movement. Slowed rate in AOS may result from difficulty recalling the motor plans or motor goals for specific sounds, organizing movements within and between speech segments, and relaying structure-specific information to the motor programming system. Rate difficulties may increase or become more prominent in AOS when the motor planning load increases. For example, rate of speech production may be influenced by linguistic variables, such as word length or frequency of occurrence, automaticity of the stimuli, or stimulus modality (input). Rate abnormalities evidenced in the dysarthrias, however, are most often uninfluenced by linguistic variables, automaticity of the stimuli, or stimulus modality (Duffy, 2005, 2013; van der Merwe, 2009).

Error Consistency. Van der Merwe's framework (1997, 2009) does not address error consistency in aphasia or AOS. With regard to AOS, the framework states that errors result from inaccuracies in the selection or sequencing of motor goals, as well as inefficient adaptation of the motor plan relative to the context in which the sound is to be spoken. Thus, one variable that may influence the consistency of error location is task complexity or the context in which a specific sound is implemented (Aichert & Ziegler, 2008; Buchwald & Miozzo, 2012; Johns & Darely, 1970; LaPointe & Johns, 1975; Marquardt et al., 2010; Odell, 2002; Odell et al., 1990; Staiger et al., 2012; Strand & McNeil, 1996). For example, errors may occur more consistently to the /s/ in "stroll" than would occur to /s/ in "sun". The word onset for "stroll" requires more articulatory movements or articulatory specific motor goals, thereby increasing the motor planning load. According to the framework, the types of errors produced should reflect the nature of the impairment, e.g., motor planning impairment (sound distortions) versus linguistic-symbolic planning impairment (phoneme substitutions). Additionally, the framework acknowledges the co-occurrence of disorders, which would result in a greater variety of error types, ranging from sound distortions, distorted substitutions, and phoneme substitutions.

General Discussion of the Models

The IA and DIVA model and the Four Level Framework discussed above are valuable to the understanding and diagnosis of AOS and /or aphasia with PP. The IA and DIVA model provide detailed accounts speech production, one focusing on the linguistic network and the other on the acquisition and execution of motor commands, respectively. The Four-level Framework, on the other hand, provides an overview of all levels of speech production, from linguistic processing to articulation. Taken together the models and framework account for the behavioral characteristics observed as a result of breakdown at the respective levels of speech production. Although the IA and DIVA models do not directly address the nature of error consistency in aphasia with PP and AOS, they do provide insight and room for interpretation. Overall, the models suggest errors are somewhat variable in these populations. Although the models are unable to independently address the relative nature of error consistency in AOS compared to PP, there is some support that for the hypothesis that errors are relatively more consistent in AOS compared to aphasia with PP.

Previous Investigations of Error Consistency

Miller (1992)

In 1992 Miller examined error consistency in 30 monolingual English speakers who had suffered a single cerebrovascular accident. Participants included ten individuals with AOS (six with pure AOS), six individuals with a diagnosis of PP (four with pure PP), and six individuals with spastic and/or unilateral upper motor neuron (UUMN) dysarthria, without co-occurring aphasia or AOS. Assignment into diagnostic groups was made by two SLPs experienced in assessment of acquired neurogenic communication disorders. Diagnosis was made on a battery of tests, including oral physical examination, dysarthria assessment, and formal and functional aphasia batteries (listed below). Information regarding the severity of speech/language impairment in each group is not provided in the publication, but the authors report that there were no significant differences in severity of impairment across groups.

A diagnosis of AOS was determined in the presence of effortful speech, slowed rate, one-feature substitution errors, distortion errors, and the relative absence of anticipatory, perseverative, and transposition errors, as well as oral facial weakness and incoordination. A diagnosis of PP was

determined in the presence of fluent, non-effortful speech, as reflected by scores on the modified Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983) scales, a relative tendency to produce substitutions of more than one feature, and presence of anticipatory, perseverative, and transposition errors. Variability of errors was not a criterion for group assignment. As mentioned above, both the AOS and the PP groups contained subgroups of participants with and without concomitant aphasia. Individuals with spastic, UUMN dysarthria did not demonstrate co-occurring aphasia.

The experimental task included repetition of five mono-, bi- and trisyllabic words five times consecutively after the examiners model, during a single session. Participant responses were tape recorded and transcribed via narrow phonetic transcription. Final responses were analyzed and coded for distortions, one-feature substitutions, anticipations, perseverations, transpositions, substitutions differing from the target by more than one-feature, omissions, additions, and complex errors.

To determine error consistency, each segment was monitored across the five trials. If a word's status changed from correct to incorrect, incorrect to correct, or from one error type to another, a point was given for variability. If a segment was produced incorrectly in the same way on successive trials a point was given for consistency. For example, magazine → *magazine*, *gagazine*, *gagazine*, *nagazine*, *gagazine* would result in 3 points for variability and 1 for consistency. A series of statistical analyses were then carried out comparing individuals' and groups' consistency scores. The analysis was conducted on 22 of the original 30 cases. Responses from eight participants were not included in the final analysis, three participants made no errors, two produced no response and/or semantic errors only, and three produced mixed semantic and phonemic errors. The remaining participants included six individuals with dysarthria, ten individuals with AOS (6 with pure AOS), and six individuals with PP (only two of which demonstrated aphasia with PP). Consistency scores ranged from 50-98.8% (M = 74.93%) for individuals with dysarthria, 6.2-58.3% (M=31.37%) for individuals with AOS, and 0-47.7% (M=20.17%) for individuals with PP. Overall, a statistically significant difference was determined between the AOS and dysarthria groups, but not the AOS and aphasia groups. These results indicate that individuals with pure AOS and AOS with concomitant aphasia produce more variable errors compared to individuals with spastic and UUMN dysarthria, but perform similarly to individuals with PP with and without aphasia.

McNeil and Colleagues (1995)

In 1995, McNeil & colleagues examined the consistency of error location, variability of error type, and pattern of errors on successive attempts across repeated trials in individuals with acquired communication disorders with the goal of assessing group differences. Participants consisted of 16 carefully selected individuals, four with pure AOS, four with CA, four with AD, and four healthy controls. Diagnosis of AOS, CA, and AD was made perceptually based on participant performance on a number of speech and language tasks, including the Apraxia Battery for Adults (ABA; Dabul, 1979), verbal subtests from the Porch Index of Communicative Ability (PICA; Porch, 1967), a conversational speech sample from the Cookie Theft description from the Boston Diagnostic Aphasia Examination (BDAE; Goodglass & Kaplan, 1983), and each participant's repetition of his or her own utterances on the picture description task. Participants were diagnosed with AOS if their speech was characterized by effortful trial-and-error groping on the initiation of speech gestures, frequent single-feature sound substitutions, articulation and prosody as accurate on imitation as on spontaneous speech, variability of articulation and prosody on repeated trials of the same utterance, and a rating between 1 and 4 on articulation agility, phrase length, and melodic line tasks from the BDAE. Participants with AOS did not demonstrate weakness or incoordination of the speech musculature when used for reflexive or automatic acts, scored 22 or above on the Ravens Coloured Progressive Matrices (Raven, 1936), and scored above the 1st percentile for normal subjects on the average of subtests II, III, V, VI, VII, VIII, X, and XI of the PICA. Participants were diagnosed with CA if they demonstrated frequent sound substitutions that occurred more frequently in repetition than spontaneous speech, speech ratings between 4 and 7 on articulation agility, phrase length, and melodic line tasks from the BDAE, and did not demonstrate AOS. Lastly, participants were diagnosed with AD using Darley, Aronson, and Brown's (1969) perceptual criteria, had neurologic history and examination consistent with a lesion or disease involving the cerebellar system, and met the same criteria as the AOS participants on the PICA.

The experimental task included the repetition of bi-, tri-, and five-syllable words, taken from the ABA repetition subtest, three times consecutively after the examiners model, during a single session. Productions were audiotaped for offline perceptual analysis via narrow phonetic transcription. Final productions were analyzed for sound segment errors. No other information regarding error coding was

provided in the publication. To calculate the consistency of error location, responses in which errors occurred two - three times on the same sound segment across the three consecutive trials were included in the analysis (e.g., banana → *bananoo*, *bananoo*, *bananoo*; see Figure 5). To calculate variability of error type, "...the number of error types that differed from each other within the same location of a word [was] divided by the number of errors in that location" (McNeil et al., 1995, pg. 47; see Figure 6).

BANANA		b	a	n	æ	n	a		
1	bænænōū							✓	
2	bænænōū							✓	
3	bænænōū							✓	
<hr/>									
BUTTERFLY		b	ʌ	f	f	l	ɪ	ɪ	
1	bʌfɛblɪɪ							✓	
2	bʌfɛfɪɪ							✓	
3	bʌfɛfɪɪ							✓	

Figure 5. Example of the procedures used for measuring consistency of error location from McNeil et al. (1995). A check indicates presence of an error.

ASHTRAY		æ	ʃ	t	r	ɪ			
1	æʃ:trɪ		✓						
2	æstrɪ		✓	✓					
3	æʃtrɪ		✓						
<hr/>									
BANANA		b	a	n	æ	n	a		
1	bænænōū							✓	
2	bænænōū							✓	
3	bænænōū							✓	

Figure 6. Example of the procedures used for measuring variability of error type from McNeil et al. (1995). A check indicates presence of an error.

Results of consistency of error location showed that individuals with pure AOS (90%, range 80-94%) performed similar to individuals with ataxic dysarthria (87%, range 73-96%), whereas individuals with CA were more variable (64%, range 29-80%). Results regarding variability of error type revealed that individuals with pure AOS (13%, range 0-16%) performed similarly to individuals with AD (10%, range 8-13%), whereas individuals with CA were more variable (26%, range 0-45%). Findings suggest that

individuals with pure AOS and individuals with AD produce more consistent errors compared to individuals with CA.

The contrasting results reported by Miller (1992) and McNeil et al. (1995) are attributed to differences in participant populations and the analyses performed. The study conducted by Miller (1992) included individuals with AOS and aphasia, the co-occurrence of aphasia likely resulted in more variable performance compared to individuals with pure AOS. In addition, the analysis performed by Miller (1992) was not particularly specific to error location or error type, whereas the analysis performed by McNeil et al. (1995) was especially sensitive to the consistency of error location and variability of error type within a word across repeated trials.

Haley and Colleagues (2013)

A recent study by Haley and colleagues (2013) examined the diagnostic validity of error consistency in the differential diagnosis of AOS and aphasia with PP. In the context of a group design, error consistency was examined in 32 individuals with aphasia and “segmental speech errors” resulting from left hemisphere stroke, with the exception of one participant who suffered a gun shot wound to the left hemisphere (Haley, et al., 2013). All participants were administered a 43-item motor speech evaluation (Duffy, 2005; Wertz LaPointe, & Rosenbek, 1984) and participant performance on the following metrics were analyzed: phonemic errors, segmental distortions, segmental prolongations, and mean syllable duration for multisyllabic words. The authors report that distortion errors were surprisingly frequent and produced by the majority of participants in the study. Therefore, presence of distortion errors was not used to differentiate groups. It is important to note that no attempt was made to rule out UUMN dysarthria, a disorder typically resulting from left hemisphere stroke and characterized by articulatory imprecision (Duffy, 2005, 2013). In this population of 32 speakers with AOS and/or aphasia with PP it is possible there were cases of co-occurring UUMN dysarthria, which may account for the reported high frequency of distortion errors.

Based on participant performance the following four comparison groups were formed: 1) prominent speech sound errors and impaired prosody (9 participants), 2) speech sound errors and borderline prosody impairment (6 participants), 3) sound substitutions errors and normal prosody (11 participants), and 4) normal prosody and minimal articulatory errors (i.e., the minimally impaired group, 6

participants). Groups 1 and 2 include participants with profiles similar to AOS and group 3 included individuals with profiles similar to aphasia with PP. The authors report quantitative metrics used to form the four comparison groups were limited in number and may not have captured the complexity of the clinical presentations. Therefore, as a complementary analysis, the performance of five participants with a strong diagnosis of AOS and four participants with a strong diagnosis of CA was performed separately. Three experienced clinicians determined participant diagnosis via a rating scale procedure.

For the experimental task, all participants were asked to repeat four words and one phrase ranging from four- to eight – syllables five times consecutively after the examiners model during a single session. All productions were transcribed via broad phonetic transcription. Error coding was based exclusively on broad phonetic transcription, without consideration of distortion errors. Syllables, rather than sounds (see McNeil et al., 1995), were chosen as the unit of analysis.

Multiple analyses were performed to investigate error consistency, including consistency of error location and variability of error type (McNeil et al., 1995), and error token variability and total token variability (word-level measures of variability see Marquardt, Jacks, & Davis, 2004). A statistical analysis of variance (ANOVA) was completed separately for the four dependent variables (e.g., error consistency/variability measures), with participant group as the independent variable. An analysis of covariance (ANCOVA) was also performed to examine the effect of error frequency on group performance. Lastly, a correlation analysis was completed to determine the relationships among the error consistency measures.

Findings revealed that all four groups exhibited similar performance across all error consistency measures, with group four, the minimally impaired group, demonstrating more consistent errors for each measure. Correlation coefficients and an ANCOVA showed that all four measures were significantly mediated by overall error frequency.

Outcomes for the small comparison group (e.g., individuals with a salient diagnosis of AOS and concomitant nonfluent aphasia and CA) were similar to the results of the larger group comparisons for consistency of error location, but not for variability of error type. Specifically, individuals with a salient diagnosis of AOS and nonfluent aphasia demonstrated greater variability of error type (M=60%) compared to individuals with CA (M=30%). Furthermore, individuals with AOS and concomitant nonfluent

aphasia showed greater error token (M= 94%) and total token (M = 94%) variability compared to participants with CA (M = 43% for error token and M = 44% for total token variability). Again, error frequency influenced results; individuals with AOS and nonfluent aphasia had a generally higher error rate (M = 3.38 errors per word) compared to individuals with CA (M = 2.10). These findings suggest it is important to consider overall error rate and general frequency of sound errors in the examination of potential group differences for error consistency. Haley and colleagues (2013) surmise that error consistency is not a valid measure in which to differentiate individuals with AOS and concomitant aphasia from individuals with aphasia with PP.

The study by Haley et al. (2013) differed from McNeil et al., (1995) in a number of ways, including participant diagnoses, severity of impairment, and sensitivity of the analysis employed (e.g., transcription type and unit of analysis). As mentioned above, the inclusion of individuals with co-occurring AOS and aphasia likely influenced error consistency. Individuals with pure AOS demonstrate errors that reflect impaired motor planning, whereas the errors produced by individuals with AOS and concomitant aphasia reflect both motoric and linguistic deficits and a variety of sources of interference. Individuals with AOS and concomitant aphasia may produce a greater variety of errors, greater frequency of errors, and demonstrate greater overall impairment compared to individuals with isolated deficits.

The subtle differences in the types of errors produced in these two populations signify the importance of narrow phonetic transcription. Narrow phonetic transcription captures more detailed and subtle nuances of speech output (McNeil et al., 2009; Odell et al., 1991) and accounts for both phonetic and phonemic errors. Haley and colleagues (2013), however, employed broad phonetic transcription, which is less sensitive and only detects errors that affect the listener's perception phonemically rather than subphonemically (e.g., distortions). Although Haley et al. (2013) attempted to replicate the coding method used by McNeil and colleagues (1995), the authors performed the analysis at the level of the syllable rather than the constituent segments, again only capturing phonemic deviations from the target.

Further examination of error consistency is warranted for the purpose of better understanding the role of error consistency in the differential diagnosis of AOS and aphasia and aphasia with PP, as well as the nature of error consistency in each of these populations. Furthermore, individuals with AOS and

aphasia better represented the clinical population served by SLPs in clinical settings and are more difficult to accurately diagnose compared to individuals with isolated deficits.

Variables Thought to Influence Error Consistency

The literature suggests that the error patterns observed in AOS and aphasia may be influenced by a number of variables. In particular, the contrasting results of Miller (1992), McNeil et al. (1995), and Haley et al. (2013) indicate that methodological differences, such as differences in participant populations/characteristics and analysis may influence error consistency outcomes. Furthermore, variables such as error frequency (Haley et al., 2013), severity of impairment (Duffy, 2005, 2013; Haley et al., 2013; Shuster & Wambaugh, 2008) and stimulus presentation condition (Johns & Darley, 1970) may also influence participant performance. These variables should be taken into consideration when examining error consistency in individuals with AOS and aphasia.

Perceptual analysis

Narrow phonetic transcription is a sensitive and specific tool that aids in the distinction between AOS and aphasia. Unlike broad phonetic transcription, narrow phonetic transcription captures the subtle articulatory errors (e.g., distortions) produced by individuals with a motor planning impairment in addition to the more obvious phonemic errors produced by individuals with a linguistic impairment (McNeil et al., 1995; Odell et al., 1990, 1991; Shuster & Wambaugh, 2000). Many of the initial AOS investigations employed broad phonetic transcription to capture the speech characteristics of individuals with AOS and concomitant aphasia, and as result demonstrated a predominance of substitution errors for this population. In 1990, however, Odell and colleagues (1990) employed narrow phonetic transcription to examine consonant productions in individuals with pure AOS and revealed a predominance of distortion errors over any other error type. Years later, a series of investigations performed by Mauszycki et al. (2010a, 2010b, 2012) employed narrow phonetic transcription to examine the speech of individuals with AOS and concomitant aphasia and also found distortions to be the dominant error type. Thus, narrow phonetic transcription is a valuable and necessary tool when examining and characterizing the speech of individuals with motor planning/programming impairments, and, to an extent, aids in distinguishing between phonetic and phonemic errors.

Co-occurring Disorders

Co-occurring AOS and aphasia may also influence the consistency in which errors occur. For example, if individuals with pure AOS are relatively more consistent in the location and type of errors produced it is possible that level of consistency demonstrated by the motor planning/programming mechanism may be masked by the more variable error patterns reported in individuals with aphasia. The models and framework described above provide support for this hypothesis. Specifically, the errors produced by individuals with AOS and aphasia result from a variety of sources of interference (e.g., spreading activation, influence of previous stimuli, reliance on feedback control; Dell, 1998; Guenther, 2006) and a range of error types (phonologic, phonetic, and/or a combination of the two; van der Merwe, 1997, 2009). Thus, individuals with co-occurring AOS and aphasia may demonstrate more variable errors compared to individuals with pure AOS and/or aphasia with PP without AOS.

Error Frequency

Haley et al. (2013) revealed that error frequency mediated error consistency outcomes. Specifically, findings indicate that individuals with lower error rates demonstrate lower consistency of error location and lower variability of error type, compared to participants with more frequent errors. Error frequency is one method that has been employed in attempt to capture severity of impairment in individuals with AOS. Unfortunately, there is no gold standard in which to quantify severity of AOS, or differentiate severity of AOS and aphasia in individuals with co-occurring disorders. This method is motivated by the numerous treatment and experimental investigations that focus on accuracy of sound production in AOS (Wambaugh via conversation, 2013). The validity of this method, however, has not been examined.

Severity of Impairment

The influence of participant severity on error consistency is unclear in individuals with AOS and aphasia. Duffy (2005) suggests that more severely impaired individuals demonstrate higher error consistency compared to those with mild deficits. This may reflect an individuals' ability to detect or correct their errors. Specifically, individuals with mild deficits may be more able to detect and correct errors across multiple trials, compared to more severely impaired individuals, and therefore, demonstrate

less consistency. Conversely, results of a small single subject investigation by Shuster & Wambaugh (2008) indicate that errors may be less consistent in more severely impaired speakers. Haley and colleagues (2013) speculate that their findings reflect the performance of more severely impaired individuals (less consistent), whereas the results of McNeil et al. (1995) reflect the performance of individuals with mild deficits (more consistent).

Stimulus Presentation Condition

The task most often used to assess error consistency involves the repetition of multisyllabic words consecutively (e.g., in a blocked presentation condition), usually three to five times (e.g., animal, animal, animal). Little research has been done, however, to examine if the manner in which stimuli are presented influences error consistency in individuals with AOS and/or aphasia. In 1970, Johns and Darley were the first to examine the effect of stimulus presentation condition on error consistency in ten individuals with a diagnosis of AOS and concomitant aphasia and ten individuals with spastic, flaccid, and/or mixed dysarthria. Specifically, the authors examined the effect of stimulus presentation condition on the repetition of 30 word initial consonant clusters embedded in real CVC words. Three different presentation conditions were employed, two blocked conditions and one random condition. All 30 words were presented three times in each condition and participants were asked to repeat words after the examiner's model. The blocked conditions differed in that one condition included three consecutive repetitions of the target word with only one model from the examiner (1:3 ratio) and the other blocked condition consisted of three consecutive repetitions of the target word with each production occurring after the examiners model (3:3 ratio). In the random presentation condition, target words were also elicited three times, each after the examiners model (1:1 ratio), but in a random order. Error consistency was measured by accuracy of phoneme production for each group across presentation conditions (employing a different analysis than McNeil et al., 1995). Findings did not reveal a significant effect of presentation condition for either group. Individuals with AOS and concomitant aphasia, however, did appear to gradually adapt during the consecutive repetition of stimuli presented in the blocked condition (1:3 ratio). This finding may suggest that these participants were able to learn or correct their errors on subsequent trials during the blocked condition (1:3 ratio). Results should be interpreted with caution,

based on the inclusion criteria reported by these authors it is possible that the AOS group included individuals with aphasia without AOS, therefore, potentially confounding results.

Since Johns and Darley's (1970) initial investigation, only a few studies have examined the effect of stimulus presentation condition on word repetition in individuals with AOS and concomitant aphasia (Mauszycki et al., 2010a, 2010b, 2012; Wambaugh et al., 2004). The methodology employed in these studies differs from that of Johns and Darley (1970) and McNeil et al. (1995). In particular, these studies examined the repetition of a target sound embedded in different words (e.g., /b/ in bob, bib, bub, & bab), rather than the repetition of a specific word across multiple trials (e.g., banana, banana, banana). The series of reports by Mauszycki and colleagues (2010b, 2010b, 2012) analyzed the speech of 11 participants during the repetition of mono-, bi-, and trisyllabic words. Dependent measures included 1) the mean percentage of errors by sound and 2) the most dominant error type/category (e.g., distortions vs. substitutions). Results suggest that stimulus presentation condition has no effect on the frequency or type of errors produced by individuals with AOS and concomitant aphasia. In contrast, a study by Wambaugh et al. (2004), analyzed the speech of a single individual with moderate AOS and concomitant aphasia during the repetition of monosyllabic words. Dependent measures included 1) percent of target sounds in error, 2) error type/categories, 3) percent of same errors across sampling times, and 4) percent same words in error. Findings indicate that the types of errors produced were influenced by presentation condition. Specifically, the participant produced a greater variety of error types in the random condition compared to the blocked condition. In addition, the blocked presentation condition appeared to elicit more predictable patterns of incorrect or correct productions compared to the random condition.

There is some evidence from the limb motor learning literature which suggests that stimulus presentation may have an effect on the retrieval of motor plans/programs (Austermann Hula, Robin, Maas, Ballard, Schmidt, 2008; Hula, 2008; Maas et al., 2008; Schmidt & Lee, 2008). In a blocked condition, different targets are produced in separate, consecutive blocks (e.g., BBBB, AAAA, CCCC), requiring the execution of the same motor program for multiple consecutive trials. In a random presentation condition, however, stimuli are intermixed (e.g., ABAC, BCAC, CABA), requiring the retrieval and/or construction of a different motor program on every trial (Knock, Ballard, Robin, & Schmidt, 2000). Thus, it is possible that the condition in which stimuli are presented will have an effect on the retrieval or

construction of motor programs for speech production. The effect of stimulus presentation condition has not been examined in aphasia, though a similar argument can be made for the retrieval of linguistic information. In a blocked presentation condition the same activated linguistic information can be used for consecutive trials, whereas in a random condition the retrieval of different linguistic information is required on every trial. With respect to the IA model (Dell 1986) a random stimulus presentation condition may result in more variable productions across trials. Stimuli presented in a random condition require the reactivation of phonemes upon each production of the word. For example, if a participant is asked to repeat “telephone” five times in a random order the phonemes for “telephone” would need to be reactivated upon each repetition of the word. Phonological retrieval across each trial may be met with variable sources of interference (e.g., different preceding stimuli) and result in more variable productions. Conversely, stimuli presented in a blocked condition likely requires the activation of target phonemes one time and then activation is maintained (or the selected phonemes are stored) during consecutive repetitions, suggesting that errors in this condition may be more consistent. Individuals with impaired maintenance of activation (e.g., impaired verbal working memory), however, may produce less consistent errors in the blocked presentation condition, as activation quickly decays across trials (Martin & Gupta, 2004).

Overall, the effect of stimulus presentation condition on error patterns in individuals with AOS and aphasia remains unclear. Differences in participant performance across presentation conditions may provide insight into the impaired mechanism and identify group differences.

Research Questions

First and foremost this study is motivated by the difficulty involved in reliably differentiating individuals with AOS and aphasia from individuals with aphasia with PP, as well as inconsistencies in previous investigations examining error consistency in these populations. Therefore, the primary objective of the proposed study was to examine the nature of error consistency in two clinical populations: individuals with AOS and aphasia (Group A) and aphasia with PP (Group P). The influence of error rate, severity of impairment, and stimulus presentation condition on participant performance was also explored, as well as group differences in the types of errors produced. In the context of a two-group experimental design, the following primary and secondary research questions were addressed:

Research Question 1 – Error Consistency

Is there a significant between group difference in percent consistency of error location and variability of error type during repetition of bi-, tri-, and five syllable words in a blocked presentation condition?

Research Question 2 – Variables Thought to Influence Error Consistency Outcomes

Research Question 2a. Is there a significant within group effect of error rate, as measured by mean number of sound errors per word for each participant across all syllable lengths, on error consistency outcomes?

Research Question 2b. Is there a significant within group effect of severity of impairment, as measured by expert ratings (1=mild, 2=moderate, 3=severe), on error consistency outcomes?

Research Question 2c. Is there a significant within group effect of stimulus presentation condition (e.g., blocked vs. random), on the consistency of error location and variability of error type during the repetition of bi, tri-, and five syllable words?

Research Question 3 – Error Type

Is there a significant between group difference in the type of sound errors produced, as measured by group differences in the production of phonetic, phonemic sequencing, non-sequential phonemic substitutions, omission, and addition errors, during the repetition of bi-, tri-, and five syllable words?

See Table 1 for outline of research questions, tasks, outcome measures, and predictions.

Table 1. Research Questions, Tasks, Outcome Measures, & Predictions

Aim	Research Question	Task	Outcome Measure	Predictions
Primary Research Question	1. Is there a significant between-group difference in the consistency of error location and variability of error type?	Repetition of 15 (blocked condition) multisyllabic words 5 times	1. Percent consistency of error location 2. Percent variability of error type	Individuals with AOS should demonstrate greater consistency of error location and less variability of error type compared to individuals with aphasia with PP. The co-occurrence of aphasia with AOS, however, may lead to more variable performance.
	2a. Is there a significant within-group effect of error rate on measures of error consistency?	Repetition of 15 (blocked condition) multisyllabic words 5 times	Mean number of sound errors per word for each participant across all syllable lengths	It is predicted that error rate will influence performance. Individuals who produce high error rates will demonstrate higher consistency of error location and lower variability of error type.
Secondary Research Questions	2b. Is there a significant within-group effect of severity of impairment on measures of error consistency?	Discourse production (SRP) and ABA repetition task	Expert raters using 1-mild, 2-moderate, and 3-severe ratings. Experts must have consensus.	It is predicted that severity of impairment will influence individual performance. Individuals who are determined to be more severe will demonstrate higher consistency of error location and lower variability of error type.
	2c. Is there a significant within-group effect of stimulus presentation condition on error consistency?	Repetition of 30 (blocked vs. random conditions) multisyllabic words 5 times	1. Percent consistency of error location 2. Percent variability of error type	It is predicted that stimuli presented in the blocked condition will be more consistently in error than stimuli presented in the random condition for both groups. Individuals with aphasia with PP are predicted to demonstrate significantly greater variability in the random condition.
	3. Is there a significant between-group difference in the types of sound errors produced?	Repetition of 15 (blocked condition) multisyllabic words 5 times	Number of: 1. distortions, 2. distorted substitutions 3. distorted additions 4. substitutions 5. additions 6. omissions	It is predicted that groups will differ significantly in the occurrence of phonetic errors. Individuals with AOS and aphasia will produce a greater number of distortions, distorted substitutions, and distorted additions. No significant differences in the production of other sound errors are expected.

CHAPTER 3 Method

Study Design

The study involved a two-group experimental design to examine the relative consistency of error location and variability of error type in 21 individuals with aphasia characterized by PP, with and without AOS. The effect of stimulus presentation condition (blocked vs. random), error rate, and severity of impairment on the consistency of error location and variability of error type was also examined, as were group differences in the types of sound errors produced.

Participants

Ten individuals with acquired AOS and aphasia (Group A) and 11 individuals with aphasia without AOS (Group P) participated in this study. Group A included four males and six females, ranging in age from 45 to 71 (M= 61.8 years, SD = 8.2 years). Time post onset of stroke ranged from ten months to 230 months (M= 82.3, SD= 70.8). Group P included eight males and four females, ranging in age from 49 to 91 (M= 65.6 years, SD = 10.8 years). Time post onset from stroke ranged from seven months to 125 months (M= 67.6 months, SD = 36.2 months). See Tables 2-3 for participant demographics.

Lesion location information was collected for descriptive purposes, but was not part of the participant selection criteria. In Group A seven participants suffered an embolic cerebral vascular accident, one participant suffered a hemorrhagic and embolic cerebral vascular accident, and one participant suffered an unspecified encephalomalacia event. Detailed lesion information could not be obtained for one of the 10 participants (AOS4). In Group P, nine participants suffered an embolic cerebral vascular accident, one participant suffered a hemorrhagic cerebral vascular accident, and for one participant no acute hemorrhage or mass effect was identified on early CT scans (later scans were not available). Refer to Tables 2-3 for more detailed information regarding areas affected by cerebral vascular attack for each participant.

Selection Criteria. Participants were recruited through the University of Washington (UW) Aphasia Registry and Repository (IRB# 37400). All participants were at least six months post onset of left hemisphere stroke, right-handed, spoke English as their primary language, had completed high-school

education, passed an audiometric pure-tone, air conduction screening at 35 dB HL at 500, 1K, and 2K Hz for at least one ear or had reports of adequate hearing when aided (n=2), had normal or correct to normal visual acuity (20/20 – 20/40), as determined by a vision screen (Snellen chart), and scored above a 23/36 on the Raven's Coloured Progressive Matrices (RCPM; Raven, Raven, & Court, 1998), with the exception of participant PP12, who was only given one subtest of the RCPM and scored 7/12. See Tables 2-3 for participant demographics.

Table 2. Demographics for Group A

Participant	Age	Handedness	Education	MPO*	Gender	Location of Stroke
AOS1	61	R	18	172	F	Left M2 segment region infarct involving the lenticulostriate system, and also insular cortex.
AOS2	58	R	19	100	M	Left MCA infarct, involving frontal, temporal, and parietal lobes.
AOS3	68	R	23	50	M	Left MCA, involving the basal ganglia, adjacent insular cortex, left corona radiata, and left frontal lobe.
AOS4	67	R	18	230	M	Large left MCA
AOS5	51	R	14	40	F	Two Strokes: L MCA aneurysmal subarachnoid hemorrhage, including left circular sulcus, sylvian fissure, and operculum extending deep to the cavernous sinus region; and L MCA occlusive event with infarction of temporal, frontal, and parietal lobes.
AOS6	66	R	16	136	M	Encephalomalacia evident left frontal distribution consistent with sequelae of remote infarct.
AOS7	60	R	17	42	F	Large left M1 infarction, with extensive frontal and temporal lobe involvement, including the superior temporal gyrus, and extensive involvement of the insula.
AOS8	45	R	16	10	F	Moderate left MCA infarct, including the posterior inferior lateral left parietal lobe with mild involvement of the posterior insula and left caudate head.
AOS9	71	R	16	22	F	Left M2 region infarct with loss of left insular ribbon, loss of gray white differentiation in the left frontal operculum and left parietotemporal lobes.
AOS10	71	R	13	21	F	Left MCA infarct involving the temporal lobe, posterior frontal lobe, and anterior parietal lobe. Minimal periventricular deep white matter T2 signal alteration, extending to involved insular cortex and posterior temporoparietal cortex and inferior frontal cortex.
AVE (SD)	61.8 (8.2)	10 Right	17 (2.7)	82.3 (70.8)	4 M 6 F	

* MPO= months post onset

Table 3. Demographics for Group P

Participant	Age	Handedness	Education	MPO*	Gender	Location of Stroke
PP1	63	R	16	52	M	Left territory infarct involving the superior temporal gyrus, dorsal insula, and frontal operculum, extending deep rostrally to anterior periventricular white matter.
PP2	70	R	12	68	F	Two strokes: left MCA territory infarcts including the temporoparietal junction, coronal radiata, and subinsular region.
PP3	57	R	12	57	F	Large left MCA territory infarcts, including of much of the temporal and frontal lobes.
PP4	49	R	16	61	M	Small left cortical infarct, including lateral frontal lobe.
PP5	59	R	18	110	F	Left MCA territory infarct, including frontal, temporal, and parietal lobes, prior to aneurysm clipping.
PP6	57	R	16	65	M	Left hemorrhagic, including basal ganglia, post frontal, temporal, and parietal lobes.
PP7	66	R	14	125	F	Extensive Left MCA infarct; involving much of left basal ganglia and cortical grey matter.
PP9	75	R	18	123	M	No acute hemorrhage or mass effect identified on early CT scans. Left basal ganglia calcification is present. No other information provided.
PP10	72	R	13	23	F	Left MCA territory infarct, including posterior temporal and parietal lobes.
PP11	63	R	16	53	M	Left MCA infarct, involving anterior left parietal lobe extending to the level of the sylvan fissure, possible extension into the left temporal lobe.
PP12	91	R	18	7	F	Left superior temporal and anterior parietal infarcts with petenchial hemorrhage in the left superior temporal a lobe.
AVE (SD)	65.6 (10.8)	12 Right	15.2 (2.0)	67.6 (36.2)	8 M 4 F	

*MPO= months post onset

Exclusion Criteria. Participants were excluded from the study if they had a positive medical history of depression or other psychiatric illness, degenerative neurological illnesses, chronic medical illness, or presented with dysarthria.

Inclusion Criteria. Participants were included in the study if they demonstrated AOS and aphasia or aphasia without AOS. Included participants must demonstrate PP as a characteristic of their aphasia. To assess additional inclusion and exclusion criteria, participants underwent speech and language testing.

Aphasia was determined by the presence of language processing deficits as measured by participant performance on the language battery portion of the Comprehensive Aphasia Test (CAT; Swinburn, Porter, & Howard, 2004; see Tables 4 and 5 for participant performance). The language battery portion of the CAT includes written and spoken language comprehension, repetition, naming (score includes word fluency), picture description, and reading and writing (due to time limitations two participants did not complete the writing portion, one of these participants did not complete the reading portion either).

To further assess presence of phonological deficits the Standardized Assessment of Phonology in Aphasia (SAPA; Kendall et al., 2010) was administered. The SAPA is a tool used to identify phonological deficits in individuals with aphasia. The test is comprised of three subtests, reading aloud, auditory phonological processing, and repetition, parsing and blending. Included participants must demonstrate phonological processing deficits on subtests of the SAPA (see Table 4).

Presence of AOS was determined by primary characteristics observed during participant performance on subtests of the Apraxia Battery for Adults – Second edition (ABA; Dabul, 2000), including I: Diadokokinesis, II: Increasing Word Length, IV: Latency and Utterance Time for Polysyllabic Words, V: Repeated Trials Test, and finally a picture description task (See characterization of participants below for specific criteria).

To obtain a speech sample, other than picture description, the Story Retell Procedure (SRP) was administered, following the procedures carried out by McNeil and colleagues (2007).

Lastly, presence of dysarthria was assessed via physical examination of the jaw, lips, tongue, velopharyngeal function, and respiration and phonation (Yorkston, Beukelman, Strand, & Hakel, 2010) during a structural functional exam and participant performance on the ABA subtests. Of particular concern was the presence of unilateral upper motor neuron (UUMN) dysarthria, therefore, the following characteristics were used to determine presence and/or differential diagnosis of UUMN dysarthria from AOS: 1) unilateral central face and/or tongue weakness, 2) slow and sometimes irregular AMRs, 3) harsh, strained or hoarse-breathy dysphonia, 4) reduced loudness (sometimes), 5) articulatory imprecision (consistent) (Duffy, 2013). All tests and tasks were administered and scored by an ASHA-certified SLP

(the author). Expert raters were asked to confirm the author's assessment of dysarthria via audiovisual recordings of participant performance on subtests II and V of the ABA, as well as the picture description task. Expert rates were in 100% agreement with the author and each other. A few participants included in the study demonstrated mild asymmetry of the lips, but this asymmetry was determined to not influence speech production.

After completion of the initial testing phase, three out of the 25 participants were excluded from the study. One participant did not demonstrate aphasia, two participants with mild aphasia did not demonstrate characteristics consistent with a diagnosis of AOS or presence of PP, and one participant was dropped from the study after dementia had been suspected and later confirmed. Therefore, a total of 21 participants met inclusion criteria. Refer to Tables 4-5 for language assessment outcomes for the 21 participants.

Characterization of Participants. All 21 participants who met inclusion criteria during the initial testing demonstrated aphasia, of varying severity, characterized by PP. Next, participants were placed into one of two groups, Group A (AOS with aphasia), and Group P (aphasia with PP, without AOS). In addition to AOS, individuals in Group A also demonstrated PP. Three certified SLPs with 20 plus years experience in the field, proficient in the differential diagnosis of neurogenic communication disorders, served as expert raters for this study. Expert raters observed audiovisual recordings of participant performance on subtests II and V of the ABA, as well as the picture description task, to determine the presence of key characteristics for inclusion into either Group A or Group P. Consensus rating was employed, thus expert raters needed to be 100% in agreement on a participant's diagnosis; AOS with concomitant aphasia or aphasia without AOS, in order for that participant to be included in the study. Expert raters met consensus for all 21 participants.

Group A. To warrant a diagnosis of AOS the following behaviors must have been observed: (1) slow speech rate characterized by sound, syllable or word segregation and phoneme lengthening (2) sound distortions, (3) distorted sound substitutions, and (4) prosodic abnormalities (McNeil et al., 2009; Staiger et al., 2012; Wambaugh et al., 2006).

Group P. Participants in this group did not present with AOS, as determined by the criteria listed above, but did present with aphasia, as determined by impaired performance on the CAT and the SAPA, and occurrences of PP, as determined by non-distorted sequential and/or nonsequential phonemic errors.

Table 4. Group A: Participant performance on measures of aphasia, CAT (Swinburn et al., 2004) and SAPA (Kendall et al., 2010).

Group A	CAT subtests					SAPA			
	Spoken Language Comp. (66)	Written Language Comp. (62)	Repetition (74)	Naming (58) + Fluency	Reading (70)	Writing (76)	Reading (52)	Auditory Phonological Processing (65)	Repetition, Parsing, Blending (34)
AOS1	51	49	70	38 +18	70	69	43	55	26
AOS2	53	53	34	56 +14	29	71	20	49	5
AOS3	63	59	65	56 +16	56	73	38	61	24
AOS4	52	46	44	26 + 4	14	33	18	47	5
AOS5	41	44	59	28 +13	44	60	28	54	21
AOS6	56	55	63	46 +11	52	67	36	62	21
AOS7	63	58	71	57 +24	62	74	42	56	22
AOS8	59	58	22	18 +11	58	76	28	50	9
AOS9	62	60	68	38 +12	63	71	38	53	16
AOS10	60	56	57	52 +14	56	75	40	55	17
AVG (SD)	56 (6.7)	53.8 (5.4)	55.3 (15.8)	55.2 (16.8)	49.6 (16.9)	66.9 (12.1)	33.1 (9.0)	54.2 (4.8)	16.6 (7.7)

Table 5. Group P: Participant performance measures on of aphasia, CAT (Swinburn et al., 2004) and SAPA (Kendall et al., 2010).

Group P	CAT subtests					SAPA			
	Spoken Language Comp. (66)	Written Language Comp. (62)	Repetition (74)	Naming [(58) + fluency]	Reading (70)	Writing (76)	Reading (52)	Auditory Phonological Processing (65)	Repetition, Parsing, Blending (34)
PP1	60	58	64	52 +19	66	67	47	56	22
PP2	22	42	40	3 +3	29	24	15	38	10
PP3	30	45	42	21 +13	25	53	18	39	13
PP4	59	44	61	48 +19	67	71	43	53	11
PP5	47	45	56	34 +18	47	56	28	51	14
PP6	52	53	65	51 +23	62	71	33	60	20
PP7	55	50	46	40 +19	55	60	35	58	14
PP9	51	49	32	46 +20	59	64	47	42	96
PP10	42	42	57	23 +9	50	NA	40	59	26
PP11	38	33	26	4 +0	0	38	2	40	9
PP12	29	41	24	9 +3	NA	NA	10	22	0
AVG (SD)	44.1 (12.3)	45.6 (6.4)	46.6 (14.3)	43.4 (25.7)	46 (20.5)	56 (14.9)	28.9 (915.6)	47.1 (11.9)	21.4 (25.7)

It is important to note that six participants in Group A and seven participants in Group P partook in an intensive aphasia treatment program prior to participation in this study.

Outcome Measures

Four outcome measures (consistency of error location, variability of error type, error rate, and error type) were assessed by participant performance on the repetition of two-, three-, and five-syllable real words (see Table 6 for definitions and calculations for all outcome measures). Experimental stimuli are described below. Severity of impairment was determined by expert raters who evaluated speech production elicited via subtests of the ABA and a speech sample from the SRP.

Experimental Stimuli

Repetition stimuli were created with the aim to induce errors in individuals with AOS and aphasia with PP. Past studies indicate that clusters result in a greater occurrence of errors than singletons (Canter, Trost, & Burns, 1985; Odell, 2002; Odell et al., 1990) and multisyllabic words stimulate errors relative to monosyllabic words (Kohn & Smith, 1995; Marquardt et al., 2010; Odell et al., 1991; Strand & McNeil, 1996). Therefore, this study included 30 two-, three-, and five-syllable real words containing both consonant singletons and clusters. The stimuli were separated into two 15-word lists (List A and B; see Appendix II). Each word list contained the same number of two-, three-, and five-syllable real words and were matched for psycholinguistic variables, including frequency of use (List A, $M=2.9$, $SD=3.8$; List B, $M=2.8$, $SD=3.9$) age of acquisition (List A, $M=453.8$, $SD=68.1$; List B, $M=471.8$, $SD=86.3$), imageability (List A, $M=425.5$, $SD=78.6$; List B, $M=423.7$, $SD=100.1$), neighborhood density (List A, $M=1.47$, $SD=1.2$; List B, $M=1.5$, $SD=1.1$), and phonotactic probability (List A, $M=.0054$, $SD=.0027$; List B, $M=.0050$, $SD=.0023$). There were no significant differences between lists ($p > .05$). Secondary to matching lists on the psycholinguistics variables mentioned above, lists were matched for type of speech (nouns, verb, etc.), concreteness, familiarity, number of phonemes, and number of clusters. In addition, there was a strong attempt to balance voice, place, and manner. Lastly, word onset and overall syllable structure were found to be similar across word lists.

An adult male from the University of Washington Speech and Hearing Department was selected to act as the “model” for stimulus elicitation. His productions of the 30 multisyllabic words were audiovisually recorded. Three members of the University of Washington Aphasia Research Laboratory transcribed recordings for accuracy and intelligibility via broad phonetic transcription. Words were re-recorded as needed until 100% accuracy and intelligibility was reached. Re-recording was only required for two of the 30 productions.

Stimuli were elicited under two response conditions, blocked and random. The blocked-successive condition consisted of 15 multisyllabic words (List A). Participants were asked to repeat each word, after a model, five times consecutively as quickly and as clearly as possible. Following participant production, the next target was then presented. Each target was presented only one time during the entire protocol. Target words were randomized within the word list for each participant. The random-single condition consisted of 15 multisyllabic words (List B). Participants were asked to repeat the target word one time. Following participant production, the next target was then presented. Each target was presented five times during the entire protocol. Target words were pseudo-randomized within the word list for each participant so that the same word was never presented twice in a row.

In each condition (i.e., blocked, random) participants were asked to repeat 15 words five times, for a total of 75 repetitions in each condition. Overall, each participant was given the opportunity to produce a total of 150 repetitions. The order of presentation condition, blocked and random, was pseudo-randomized across participants. Half of the participants were exposed to the blocked condition first and half were exposed to the random condition first. Each condition was completed in a single session.

Experimental Procedures

Participants were seated in a quiet room, either located in the University of Washington Aphasia Laboratory or in the participants' home. A high quality, head-mounted microphone (Audio-Technica ATM75) and high quality video recorder (Canon VIXIA HF20) were utilized to capture the speech signals for perceptual analysis. A microphone-to-mouth distance of approximately five centimeters (two fingers widths) was maintained within and across participants to ensure uniform recording conditions. Audio-video recordings were chosen because video recording systems have some advantages over audio

recording systems. First, video recordings allow for the viewing of articulatory behavior (though not all articulatory behavior is linguistically significant); behaviors that otherwise might not be intelligible on audio media may become so when accompanied by visual information, e.g., articulatory groping. Additionally, in some cases visual information helps to better decode speech segments that may come across as unintelligible via audio media. The recorded speech data for each participant was stored in coded files.

Stimuli were presented to participants via a computer monitor and external speakers (Alesis M1ACTIVE 520). Participants were seated in front of the computer monitor and, for each trial, saw and heard a short video clip of a speaker's mouth producing the target word one time. This elicitation method was chosen because it allows the stimuli to be presented identically each time, therefore avoiding presenter mistakes that could induce participant errors (Staiger et al., 2012). Stimuli were played at a volume determined comfortable to the participant.

An ASHA-certified and licensed SLP (the author) conducted the experimental sessions. At times student research assistants aided in the data collection process under the supervision of the SLP. The tasks were explained to the participant during each condition. Once the participant was ready to begin three practice items were provided to make sure the participant understood the task, to test the volume/intensity, and to allow the participant to become familiar with the recorded voice and mouth image. Typically, the model was only provided one time to elicit a participants' response. The SLP played the model/recording a second time if a) the participant did not hear or understand the initial model, b) if the participant had been distracted during the time the model was played, or c) if there was extraneous noise during the first model. No feedback concerning the accuracy of productions was provided. General encouragers were the only form of feedback (e.g., "you're doing exactly what you're supposed to do", e.g., "great effort that was hard") provided to participants.

The experimental session took no longer than 60 minutes. On average, the session lasted about 40 minutes. Participants were given the option to take a break between conditions.

Outcome Measure Description

Four outcome measures, consistency of error location, variability of error type, error rate, and error type were determined by repetition of multisyllabic words five times. Specifically, the two measures of error consistency, consistency of error location and variability of error type were examined across the two presentation conditions (List A and B). Error rate and the types of sound errors produced (error type) were examined in the blocked condition only (List A). The blocked condition is considered standard of care, we were interested in variables that might influence error consistency outcomes in this condition and the types of sound errors elicited for each group. Severity of impairment was determined for each participant via consensus ratings by the three expert raters described above. Expert raters watch audiovisual recordings of each participant complete subtests II or III from the ABA-2 and a 25-35 second speech sample from the SRP. Raters used the following rating scale as a guide to rate severity: 1 = mild, 2 = moderate, and 3 = severe. Consensus rating was employed.

Outcome Measure Analysis

Consistency of error location was calculated to express the degree to which sound errors occur in the same target sound across five trials. High consistency of error location indicates that errors are located in the same place within a word and low consistency of error location indicates that errors occur in different locations within a word. Consistency of error location was determined as the number of instances in which the same sound segment was in error two times or more (Kendall, McNeil, Shaiman, & Simonian, 1999; McNeil et al., 1995). Consistency of error location was calculated by dividing the number of sounds consistently in error (within a word type) by the number of total sounds in error (Haley et al., 2013; McNeil et al., 1995; Szeles et al., 2011). The computation provided a percentage, with high values indicating more consistency and lower values indicating less consistency.

Variability of error type was calculated to express the degree to which sound errors differ from each other within the same location of a word. A low degree of variability of error type indicates that the type of errors made were relatively consistent, whereas a high degree of error type variability indicates that many different errors occurred from trial to trial in the same location of the word. Variability of error type was calculated by dividing the number of different errors that occurred within the same location of a

target word across trials over the total number of errors produced within the same location of the target word across trials (McNeil et al., 1995; Staiger et al., 2012). This computation provided a percentage, with high values indicating more variability and lower values indicating less variability.

Error rate was expressed as the mean number of sound errors per word for each participant (Haley et al., 2012).

Error type was expressed as the number of specified error types produced per participant. Errors were coded as follows: 1) distortions, 2) distorted substitutions, 3) distorted additions, 4) phonemic sequencing errors (i.e., perseverative, anticipatory, transposition errors), 5) nonsequential phonemic substitution errors, 6) omissions, 7) additions.

Table 5. Outcome Measures: Definitions and Calculations

Error Consistency:	Two measures: 1. Percent consistency of error location 2. Percent variability of error type
1. Consistency of error location	Degree to which sound errors occur in the same target sound across trials Calculated by dividing the number of sounds consistently in error (within a word type) by the number of total sounds in error
2. Variability of error type	Degree to which sound errors differ from each other within the same location of a word Calculated by dividing the number of errors that differ from each other within the same location of a target word across trials by the total number of errors produced within the same location of the target word across trials
Error Rate	Mean number of sound errors per word for each participant
Error Type:	Number of specified error types (6 types) produced by each participant
1. Distortions	An attempt at the target phoneme that does not cross the phoneme boundary, produced with perceptible place, timing, manner, or voice deviation from accurate production
2. Distorted substitutions	A production that not only crosses phoneme boundaries of the target phoneme, but is also distorted
3. Distorted additions	An inserted <i>non-target</i> phoneme that is distorted
4. Sequential substitutions	Phonemic perseveration, anticipation, and transposition errors
5. Nonsequential substitutions	Phonemic errors other than sequential errors, influenced by factors outside the target word
6. Addition	An inserted <i>non-target</i> phoneme that is phonetically accurate (not distorted)
7. Omission	A deleted phoneme

Transcription. All speech samples were analyzed perceptually offline utilizing narrow phonetic transcription (Shriberg & Kent, 2003) via audiovisual recordings. The participants' first full realization of the target word, for each trial, was selected for analysis. The selected audiovisual segments were spliced, saved separately from the recorded session, and pseudo-randomized so that speech samples of the same target would not be listened to consecutively. These procedures were followed to avoid possible perceptual influences during transcription, such as listening to multiple productions of the same target word in a row, or listening to the model that preceded the speech sample. For example, in the blocked condition participants were asked to repeat the target five times consecutively, "blessing, blessing, blessing, blessing, blessing". If all five productions were presented in a row during transcription the first production may influence perception of following productions. In the random condition participants were asked to produce a stimulus one time after the model (similar to the first repetition of each word in the blocked condition). If the model is presented prior to the production during transcription the model may influence what is perceived as an acceptable production.

Two groups of two trained transcribers worked to transcribe participant responses via *narrow phonetic transcription* (Shriberg & Kent, 2003). In each group the first transcriber, TS1, was responsible for creating the original transcription and the second transcriber, TS2, was responsible for "checking" the original transcription. The method of checking is explained in more detail below.

Transcribers sat at a computer in the Aphasia Laboratory, using headphones they (Sennheiser HD 280 Pro) listened to speech samples via Adobe Premiere Elements 13 (Adobe Systems Incorporated, 2015). Transcribers were informed to use the auditory information first and use visual information for clarification when necessary. Following the recommendations by Shriberg & Kent (2003), *previewing* was performed on speech samples from each participant before transcription began. Previewing allowed for volume/intensity adjustments, audio adjustments if background noise was present, desensitization to any biasing variables (e.g., dialect), and familiarization to the speech tempo of the participant.

After previewing, accuracy judgments were made. Transcribers listened to a speech sample one time and decided if the production was correct or incorrect. If correct, a box with a "C" was checked on

the score form and the transcriber moved on to the next speech sample. If incorrect, the transcriber transcribed the production using narrow phonetic transcription.

Participant responses were considered correct if they met one of two criteria: 1) perceived as correct, based on transcriptions from the Cambridge Dictionaries Online (Cambridge University Press, 2015) and the Merriam-Webster Online Dictionary (Merriam-Webster, Incorporated, 2015); or 2) considered an acceptable allophonic or dialectal variation of the target, e.g., /fotogræf/, /fotəgræf/, /forəgræf/.

Participant responses that consisted of a non-response, semantic error, or were difficult to reliability lineup with the target word to analysis error consistency (e.g., words had less than 30% of the target phonemes, but match the number of target syllables, or words had less than 40% of the target phonemes and did not match the number of target syllables) were not included in the data analysis.

Transcription checking. The second transcriber in each group, i.e., TS2, performed checking. TS2 listened to speech samples, as described above, while viewing the original transcription and checked whether or not they were in agreement with the original transcription created by TS1. If there were disagreements, notes were made, and consensus checking between TS1 and TS2 was employed. The following steps suggested by Shirberg and Kent (2003) were followed:

1. Replay the words on which transcribers disagreed. During each replay, both transcribers attempt to “hear” the other’s transcription, in addition to attempting to confirm his/her own transcription.
2. After the replays and second transcriptions, transcribers apply one of the 17 consensus rules (e.g., “If one transcriber immediately “hears” and accepts the other transcriber’s transcription as more accurate, select that transcription as the correct transcription”) (See Appendix III for 17 consensus rules, originally reported in Shirberg, Kwiatkowski, & Hofmann, 1984).

Error Coding. After speech samples were transcribed and checked, the final transcription was analyzed for predetermined error categories by one of the transcribers mentioned above. Error categories

included distortions, distorted substitutions, distorted additions, omissions, additions, phonemic perseverative, anticipatory, and transposition errors (sequencing errors), and other non-sequential substitution errors. Other behavioral characteristics, such as prosodic abnormalities and articulatory groping were noted, but not included in this analysis.

Distortion errors are defined as an attempt at the target phoneme that did not cross the phoneme boundary, produced with perceptible place, timing, manner, or voice deviation from accurate production. A distorted substitution error is defined as a production that not only crossed phoneme boundaries of the target phoneme, but was also distorted (van der Merwe, 1997, 2009). A distorted addition is defined as an inserted non-target phoneme that is distorted. Phonemic substitution errors make up two categories, sequential errors and non-sequential errors. Sequential phonemic errors include phonemic perseveration errors, anticipation errors, and transposition errors; these errors are influenced by the context in which the word is produced. Non-sequential substitution errors are defined as phonemic errors influenced by factors outside the target word (Mackay & James, 2004). An addition error is defined as an inserted non-target phoneme that is phonetically accurate. Lastly, an omission error is defined as a deleted phoneme that may result from breakdown at the level of phonological retrieval or motor planning².

Data Transfer. After coding was completed, a student research assistant transferred the hard copy information to an organized workbook in Microsoft Excel. Symbols in Microsoft Excel and an online IPA keyboard (Szynalski; <http://ipa.typeit.org/>) were used to input diacritic markers. This workbook was set up to mimic the original score forms, but with the addition of extra cells and macros to report and compute outcome measures (specified above). Data transfer was supervised and thoroughly checked by the author for accuracy.

² Studies suggest that allophonic variations of participant production including an omission error may determine the locus of breakdown (phonetic vs. phonemic) (Buchwald & Miozzo, 2011, 2012; Buckingham, 1986; Dell, 1988)

Blessing	'b		l	ε	s	l	ŋ
1	'b	ə	l	ε	s	l	ŋ
2	'b	ə	l	ε	s	l	ŋ
3	'b		l	ε	s	l	ŋ
4	'b		l	ε	s	l	ŋ
5	'b	ə	l	ε	s	l	ŋ

Figure 7. Participant data after it has been transferred to a digital format. An example of consistent error location and error type.

Announcement	ə	'n	aʊ	n	s	m	ə	n	t
1	ə	'n	aʊ	n	s	m	ə	n	t
2	ə	'n	aʊ	n	s	w	ə	n	t
3	ə	'n	aʊ	n	s	w	ə	n	
4	ə	'n	aʊ	n	s		ə	n	t
5	ə	'n	aʊ	n	s		l	ŋ	

Figure 8. Participant data after it has been transferred to a digital format. An example of more variable location and error type.

Statistical Analysis

SPSS (IBM Corp., 2012) was used to complete data analysis for each research question. Independent-samples T-tests were performed separately to answer the primary research question, group and secondary research questions 2b-3. Analyses of variance (ANOVAs) were completed to answer research question 2a. P-values were set at 0.05; all comparisons were motivated by a priori hypotheses. The probability of a Type I error is smaller than if the comparisons are arrived a post hoc, thus corrections for multiple comparisons were not performed (Ash et al., 2011). See Hurlbert and Lombardi (2012) for additional support for not using multiple comparison procedures.

Reliability

To establish inter- and intra-rater reliability for narrow phonetic transcription, 15% of each participant's responses were randomly selected for re-analysis by the two groups of transcribers described above. Transcribers that did not create or check the original transcription for a participant performed inter-rater reliability. Cohen's Kappa, an inter-rater agreement statistic, was used to compute reliability in SPSS (IBM Corp., 2012). Cohen's Kappa is specifically used for categorical data and controls

for the agreement you would expect to get based on chance alone. Inter-rater reliability for item-to-item agreement for narrow phonetic transcription was .74, including a limited number of transcription differences considered functionally equivalent (e.g., /d/; partially devoiced /d/ vs. /t/; a partially voiced /t/). This value denotes substantial agreement (Landis & Koch, 1977). Intra-rater reliability for item-to-item agreement for narrow phonetic transcription was .80, which also denotes substantial agreement.

To establish inter- and intra-rater reliability for error type, 15% of each participant's erred responses were randomly selected for re-analysis. Cohen's Kappa, was used to compute reliability in SPSS. Inter-rater reliability for coding was performed by a fifth transcriber who had not been involved in the transcription process, but was trained on narrow phonetic transcription and error types. Inter-rater reliability for item-to-item agreement for error type coding was .95 – almost perfect agreement (Landis & Koch, 1997). Intra-rater reliability for item-to-item agreement for error type coding was .99.

To establish intra-judge reliability for severity of impairment, one participant video from each group was repeated (~ 10% reliability). Intra-rater reliability for severity of impairment was 100%.

CHAPTER 4 Results

General information

All 21 participants completed both blocked and random conditions attempting to repeat the 30 multisyllabic words five times (a possible 150 productions per participant, totaling 3,150 productions total). A total of 244 responses were excluded from the study. These exclusions consisted of semantic errors, non-responses, and/or could not be reliably lined-up with the target word to analyze error consistency. Therefore, a total of 2,906 responses were included in the analyses reported below.

All participants made errors in the blocked and random conditions. Independent-samples t-tests were conducted to compare Group A and Group P on total errors in each presentation condition (blocked and random; see Figure 9). There were no significant differences between Group A ($M = 157.20$, $SD = 91.91$) and Group P ($M = 148.18$; $SD = 106.66$) in the number of errors produced in the blocked condition; $t(18.956) = .208$, $p = .837$. There were no significant differences between Group A ($M = 144.70$, $SD = 95.63$) and Group P ($M = 91.36$, $SD = 68.00$) in the number of errors produced in the random condition; $t(16.108) = 1.460$, $p = .164$. There were also no significant differences in the number of errors produced in the blocked and random conditions for Group P; $t(16.973) = 1.490$, $p = .155$. As indicated by the large standard deviation scores, a range of participant ability was present in each group and condition.

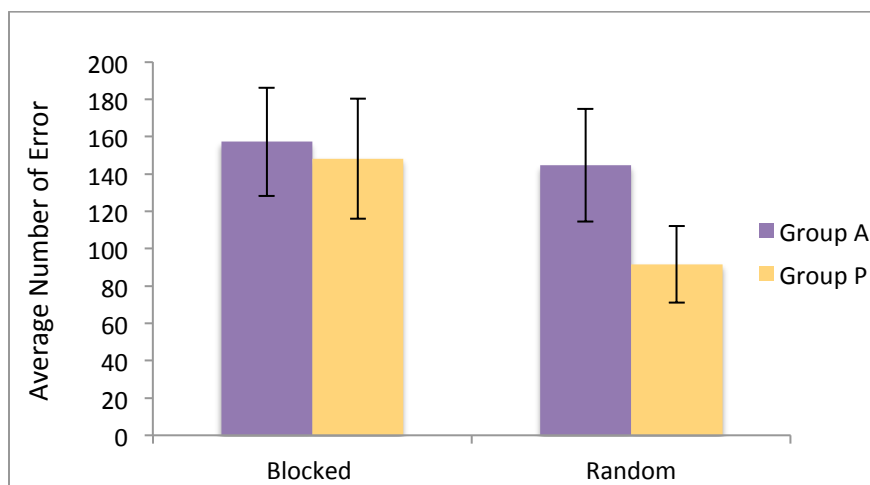


Figure 9. Average Number of Errors Per Participant for Each Group

Research Question 1 – Error Consistency

An independent-samples t-test was conducted to Group A and Group P on two measures of error consistency (percent consistency of error location and percent variability of error type) during repetition of multisyllabic words in a blocked presentation condition. There were no significant difference between Group A (M= 69.91%, SD= 12.54) and Group P (M= 66.76%, SD= 24.33) in percent consistency of error location; $t(15.25) = .378$, $p = .710$ (See Figure 10). There was a significant difference between Group A (M= 44.87%, SD= 11.51) and Group P (M= 29.88%, SD= 15.89) in percent variability of error type; $t(18.15) = 2.492$, $p = .023$ (See Figure 11). Figures 12 and 13 illustrate individual performance error location and type, respectively. In particular, for the consistency of error location Group P demonstrated a wide range of individual variability compared to Group A. These results suggest that one measure of error consistency, consistency of error location, does not differentiate group performance, whereas the variability of error type does. Specifically, individuals with AOS and aphasia demonstrate greater variability of error type during the repetition of multisyllabic words presented in a blocked condition compared to individuals with aphasia with PP.

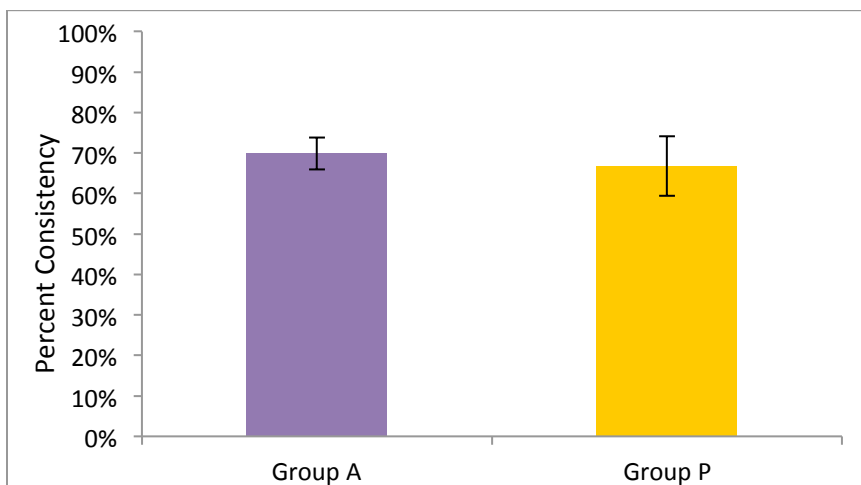


Figure 10. Group Performance on Consistency of Error Location in the Blocked Condition

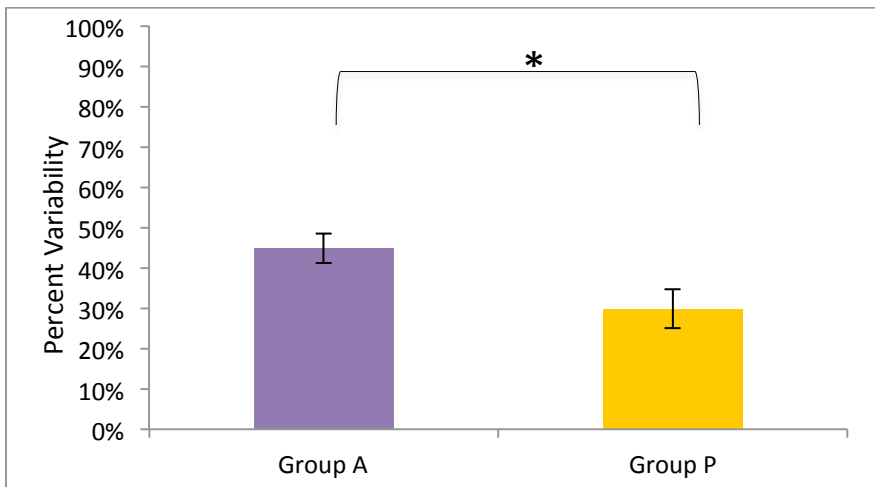


Figure 11. Group Performance on Variability of Error Type in the Blocked Condition

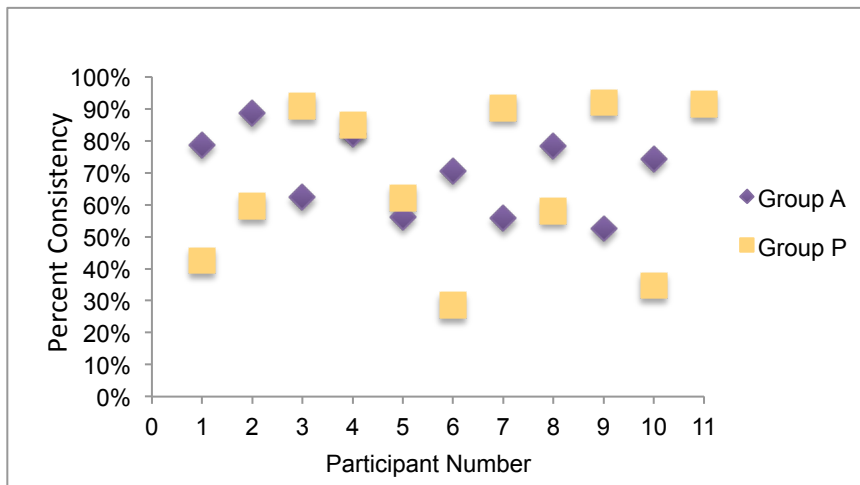


Figure 12. Individuals Performance on Consistency of Error Location in the Blocked Condition

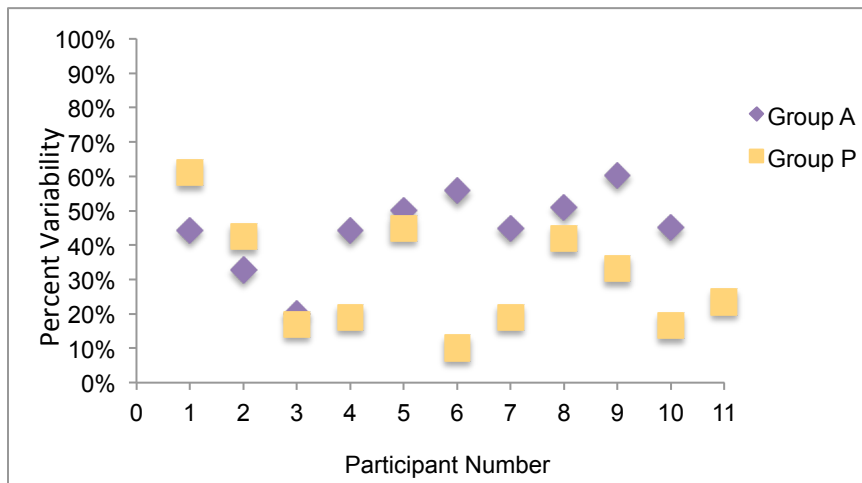


Figure 13. Individuals Performance on Variability of Error Type in the Blocked Condition.

Research Question 2 – Variables Thought to Influence Error Consistency

Outcomes

Research Question 2a – Error Rate

Across groups, the overall error rate ranged from 0.25 to 5.62 incorrect sounds per word. There were no significant differences between Group A ($M= 2.08, SD= 1.32$) and Group P ($M= 2.39, SD= 1.71$) on error rate $t(1.086)= -.469, p = .645$. Low and high error rate groups were created to examine the influence of error rate on measures of error consistency in each group. Error rate groups were created because an analysis of covariance (ANCOVA) was not supported due to the small sample size in Group A and Group P. Low and high error rate groups were created by averaging the mean error rate for all participants in each group, in the blocked condition, and dividing by two. Scores that fell below this number were considered to be relatively low error rates and scores that fell above this number were considered to be relatively high error rates.

Independent-samples t-tests were conducted to examine the influence of error rate on two measures of error consistency, percent consistency of error location and percent variability of error type, within groups, in the blocked presentation condition. For Group A, there were no significant differences

between the low error rate group (n= 2; M= 58.98%, SD= 4.75) and the high error rate group (n= 8; M=72.65%, SD= 12.50) on percent consistency of error location; $t(5.222)= 2.461$, $p = .055$ (See Figure 14). There were no significant differences between the low error rate group (n= 2; M= 32.49%, SD= 17.66) and the high error rate group (n= 8; M=47.97%, SD= 8.42) on percent variability of error type; $t(1.117)= 1.206$, $p = .424$ (See Figure 13). For Group P, there was a significant differences between the low error rate group (n= 3; M= 35.22%, SD= 6.98) and the high error rate group (n= 8; M=78.58%, SD= 15.67) on percent consistency of error location; $t(8.271)= 6.331$, $p = .000$ (See Figure 15). There were no significant differences between the low error rate group (n= 3; M= 29.26%, SD= 27.78) and the high error rate group (n= 8; M=30.11%, SD= 11.84) on percent variability of error type; $t(2.279)= -.051$, $p = .963$ See Figure 14). Results suggest that individuals in Group P who produce higher error rates produce more consistent errors, in regard to error location, compared to individuals with lower error rates, during the repetition of multisyllabic words presented in a blocked condition. A similar trend was observed for Group A, though this finding is not significant. Results also indicate that variability of error type is not influenced by error rate for either group.

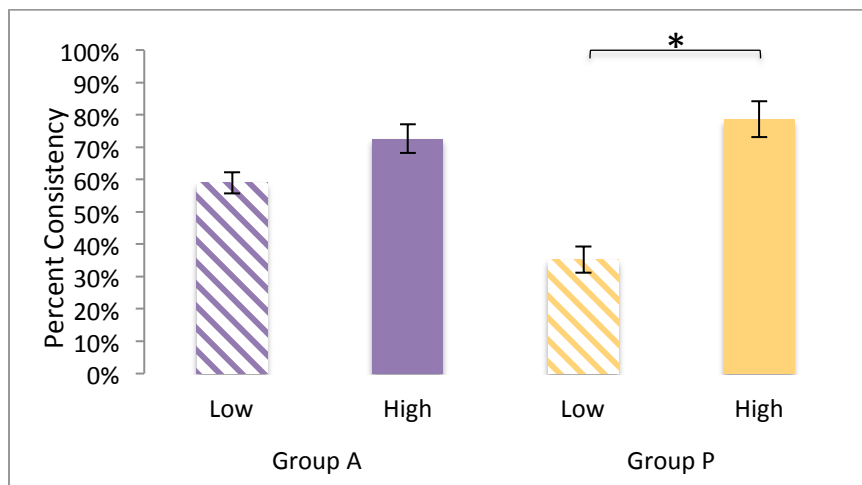


Figure 14. Influence of Error Rate on Consistency of Error Location

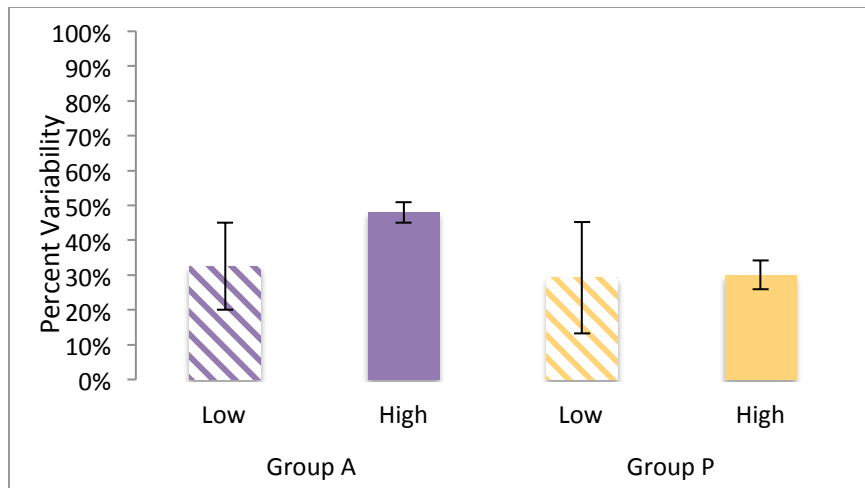


Figure 15. Influence of Error Rate on Variability of Error Type

Research Question 2b – Severity of Impairment

The result of expert raters severity ratings on severity of impairment for Group A yielded three groups: mild (n=3), moderate (n=5), and severe (n=2). Severity ratings for Group P yielded two groups: mild (n=5) and moderate (n=6).

For Group A, one-way ANOVAs were performed to examine the influence of severity of impairment on two measures of error consistency (percent consistency of error location and percent variability of error type) in the blocked presentation condition. For consistency of error location there was a nonsignificant main effect of severity, $F(2, 9) = 4.472$, $p = .056$ (See Figure 16). For variability of error type there was a nonsignificant main effect of severity, $F(2, 9) = .145$, $p = .868$ (See Figure 16). Independent-samples t-tests were conducted to further examine the influence of severity of impairment on consistency of error location in Group A. There was a significant difference between the mild ($M=56.83\%$, $SD= 5.01$) and moderate severity groups ($M=73.65\%$, $SD= 11.87$) in percent consistency of error location; $t(5.720)= -2.781$, $p = .034$. There was a significant difference between the mild and severe severity groups ($M= 80.19\%$, $SD= 2.71$) in percent consistency of error location; $t(2.989)= -6.730$, $p = .007$. There was no significant difference between the moderate and the severe group in percent consistency of error location; $t(4.784)= -1.158$, $p = .301$. These results suggest that severity of AOS and co-occurring aphasia influence the consistency of error location. Specifically, individuals with a diagnosis

of moderate or severe impairment demonstrate higher consistency of error location compared to individuals with a mild impairment.

For Group P, independent-samples t-tests were conducted to examine the influence of severity of impairment on two measures of error consistency, percent consistency of error location and percent variability of error type, within groups, in the blocked presentation condition. There were no significant differences between the mild (M= 62.13%, SD= 29.90) and moderate severity groups (M=72.31%, SD= 17.08) on percent consistency of error location; $t(19.790) = -7.07, p = .500$ (See Figure 15). There were no significant differences between the mild (M= 26.52%, SD= 18.57) and moderate severity groups (M=33.90%, SD= 12.79) on percent variability of error type; $t(0.301) = -.777, p = .457$ (See Figure 17). These results suggest that severity of aphasia alone does not influence consistency of error location or variability of error type in this group of individuals.

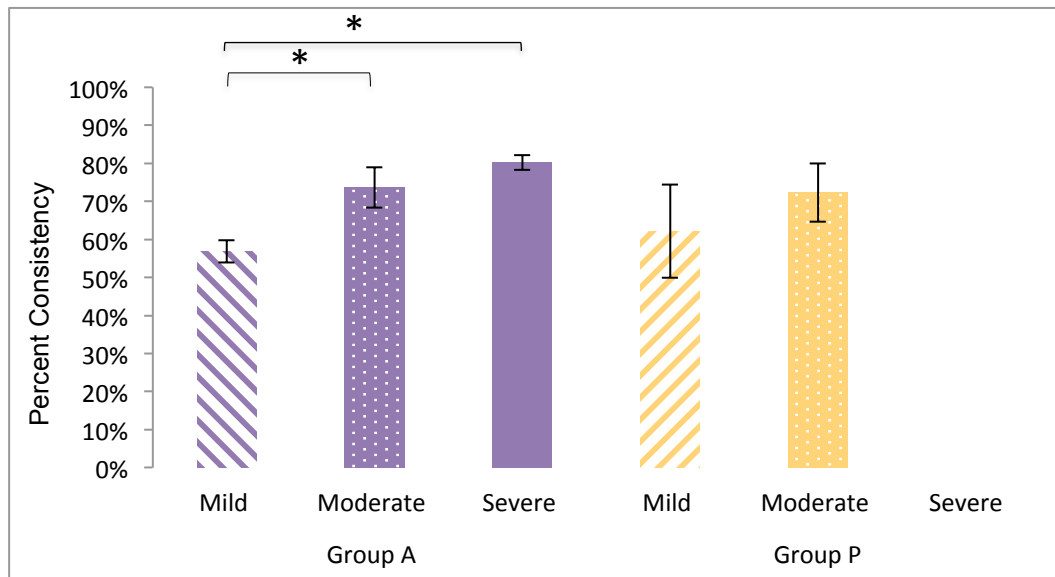


Figure 16. Influence of Severity of Impairment on Consistency of Error Location

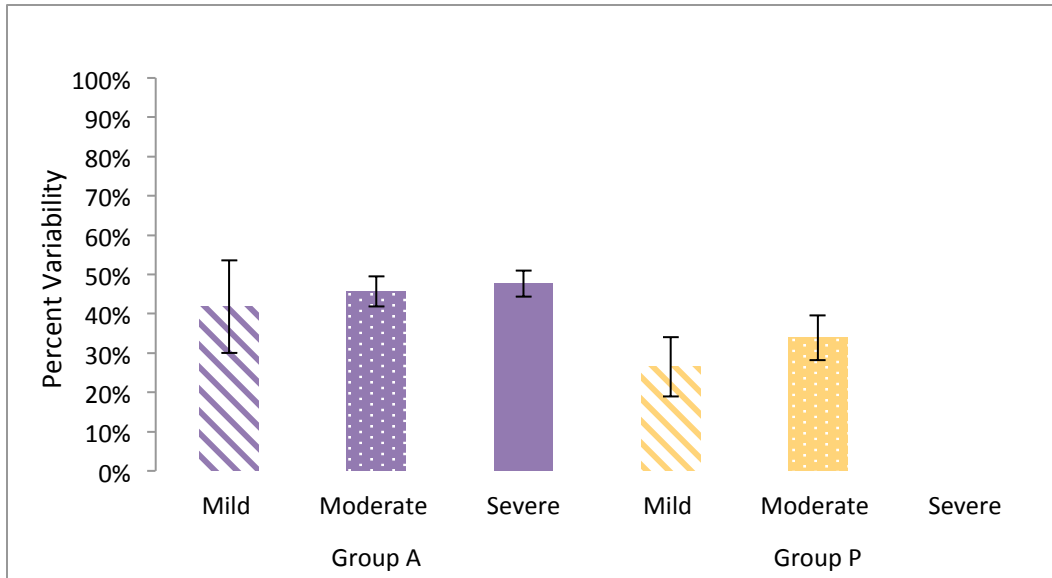


Figure 17. Influence of Severity of Impairment on Variability of Error Type

Research Question 2c – Stimulus Presentation Condition

Two, two-way repeated measures factorial ANOVAs were performed to examine the effect of group and stimulus presentation condition on error consistency (consistency of error location and variability of error type) during the repetition of multisyllabic words. There was a nonsignificant main effect of group, $F(1, 9) = 2.039$, $p = .187$, condition, $F(1, 9) = .556$, $p = .475$, and interaction between the effect of group and condition on consistency of error location, $F(1, 9) = 1.356$, $p = .273$ (See Figure 18). Figures 19 and 20 illustrate individual performance on consistency of error location in blocked and random conditions. In particular, Group P demonstrates a more variable pattern of performance across presentation conditions (Figure 20). An independent-samples t-test was conducted to examine between group differences on the consistency of error location for the random condition. There was not a significant difference between Group A ($M=71.37\%$, $SD= 11.74$) and Group P ($M=59.20\%$, $SD= 18.02$) in percent consistency of error location for the random condition; $t(1.909)= 1.848$, $p = .082$. For variability of error type, there was a nonsignificant main effect of group, $F(1, 9) = 1.239$, $p = .295$, condition, $F(1, 9) = 1.685$, $p = .227$, and interaction between the effect of group and condition, $F(1, 9) = 3.000$, $p = .117$ (See Figure 21). Figures 22 and 23 illustrate individual performance on variability of error type in blocked and random conditions. Once again, Group P demonstrates a more variable pattern of performance across

presentation conditions (Figure 23). Overall, results suggest that presentation condition does not affect error consistency performance in individuals with AOS and aphasia and individuals with aphasia with PP.

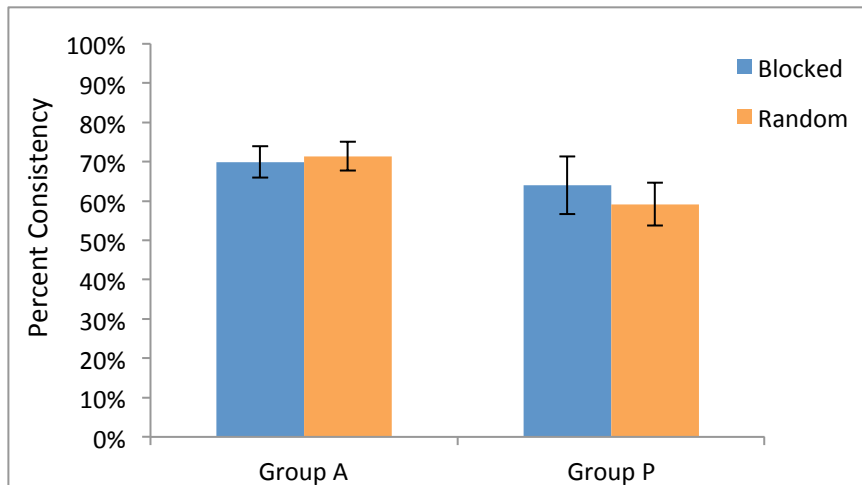


Figure 18. Effect of Group and Stimulus Presentation Condition on Consistency of Error Location

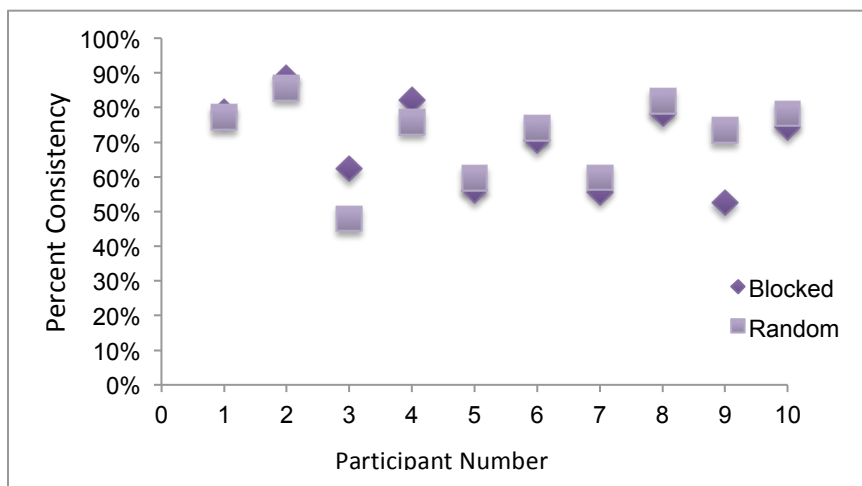


Figure 19. Group A: Individual Performance on Consistency of Error Location in Blocked and Random Presentation Conditions

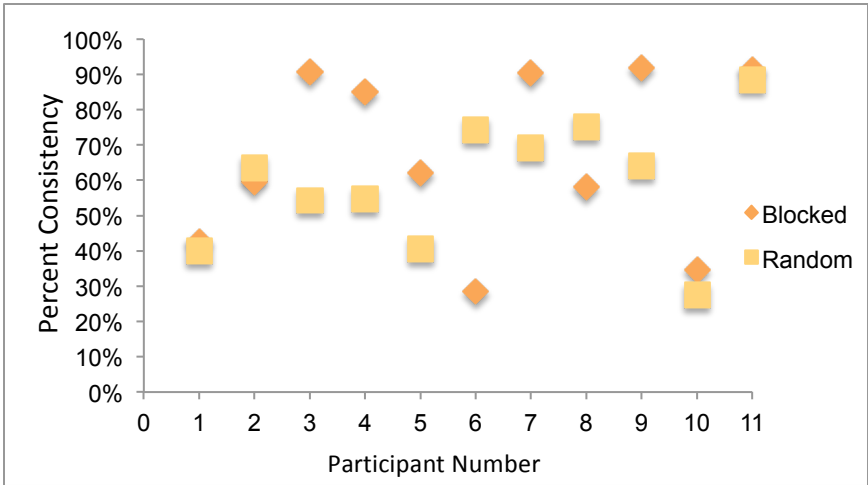


Figure 20. Group P: Individual Performance on Consistency of Error Location in Blocked and Random Presentation Conditions

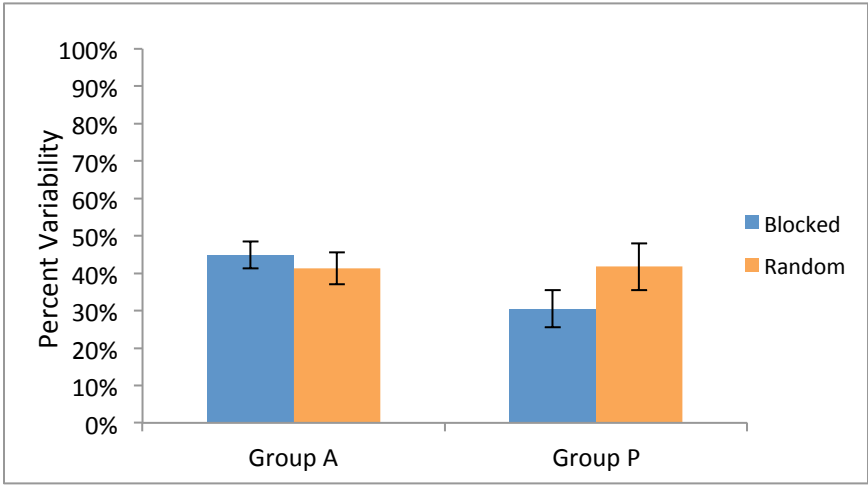


Figure 21. Effect of Group and Stimulus Presentation Condition on Variability of Error Type

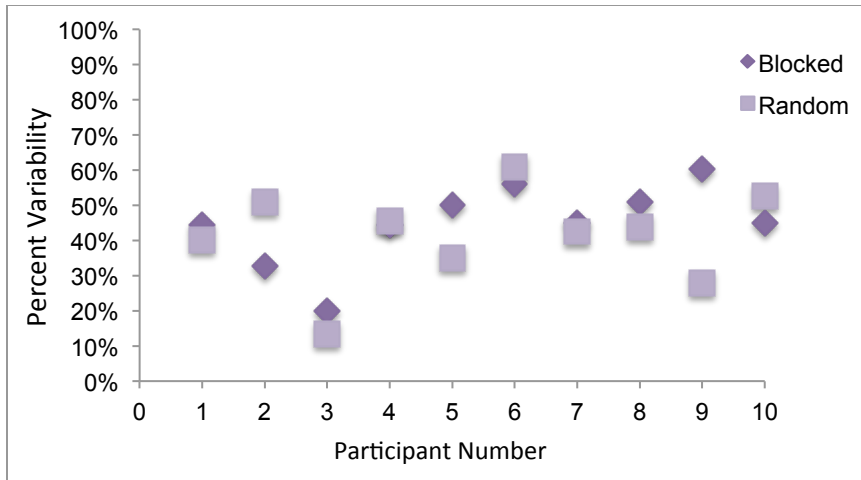


Figure 22. Group A: Individual Performance on Variability of Error Type in Blocked and Random Presentation Conditions

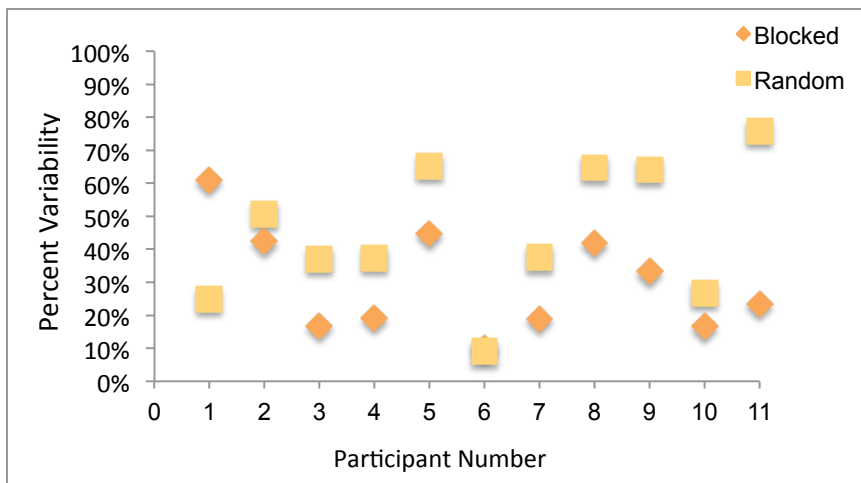


Figure 23. Group P: Individual Performance on Variability of Error Type in Blocked and Random Presentation Conditions

Research Question 3 – Error Type

An independent-samples t-test was conducted to compare Group A and Group P on the production of six error types (including distortions, distorted substitutions, distorted additions, substitutions, additions, and omissions) during repetition of multisyllabic words. Due to the small occurrence of sequential substitution errors in Group A and Group P, as well as a lack of between group

differences, the sequential substitution category was collapsed with the nonsequential substitution category, now referred to as “substitutions” (See Figure 24).

Distortions. In the blocked condition there was a significant difference between Group A (M= 32.50, SD= 16.22) and Group P (M= 15.36, SD= 7.22) in the occurrence of distortion errors; $t(12.20) = 3.08, p = .009$.

Distorted Substitutions. There was a significant difference between Group A (M= 17.00, SD= 9.90) and Group P (M= 5.09, SD= 4.28) in the occurrence of distorted substitution errors; $t(12) = 3.52, p = .004$.

Distorted Additions. There was a significant difference between Group A (M= 15.80, SD= 9.77) and Group P (M= 3.82, SD= 2.82) in the occurrence of distorted addition errors; $t(10.36) = 3.74, p = .004$.

Substitutions. There were no significant differences between Group A (M= 45.50, SD= 34.91) and Group P (M= 54.09, SD= 46.93) in the occurrence of substitution errors; $t(18.33) = -.479, p = .638$.

Additions. There were no significant differences between Group A (M= 23.90, SD= 21.34) and Group P (M= 28.09, SD= 29.67) in the occurrence of addition errors; $t(18.11) = -.374, p = .713$.

Omissions. There were no significant differences between Group A (M= 22.50, SD= 29.40) and Group P (M= 41.73, SD= 36.68) in the occurrence of omission errors; $t(18.73) = -1.33, p = .199$.

These results suggest that groups differed with regard the frequency in which sound distortions, distorted substitutions, and distorted additions occur. Specifically, Group A produced significantly more phonetic errors compared to Group P. Groups did not differ in the production of substitutions, omissions, and additions.

Percent error type. For Group A, in the blocked condition, nonsequential substitutions were the most prominent error type (23.09%; M = 36.30, SD = 30.31), followed by distortions (20.67%; M = 32.50, SD = 15.39), additions (15.20%; M=23.90, SD = 20.27), omissions (14.31%; M = 22.50, SD = 27.89), distorted substitutions (10.81%; M = 17.00, SD = 9.39), distorted additions (10.05%, M = 15.80, SD = 9.27), and sequential substitutions (5.85%, M = 3.07, SD = 3.00). For Group P, in the blocked condition,

nonsequential substitutions were also the most prominent error type (32.45%; M = 48.09, SD =42.41), followed by omissions (28.16%; M = 41.73, SD =34.67), additions (19.00%; M = 28.09, SD = 28.29), distortions (10.31%; M = 15.27, SD = 6.90), sequential substitutions (4.05%; M = 2.00, SD =2.47), distorted substitutions (3.5%; M = 5.18, SD =4.20), and distorted additions (2.6%; M = 3.82, SD =2.70).

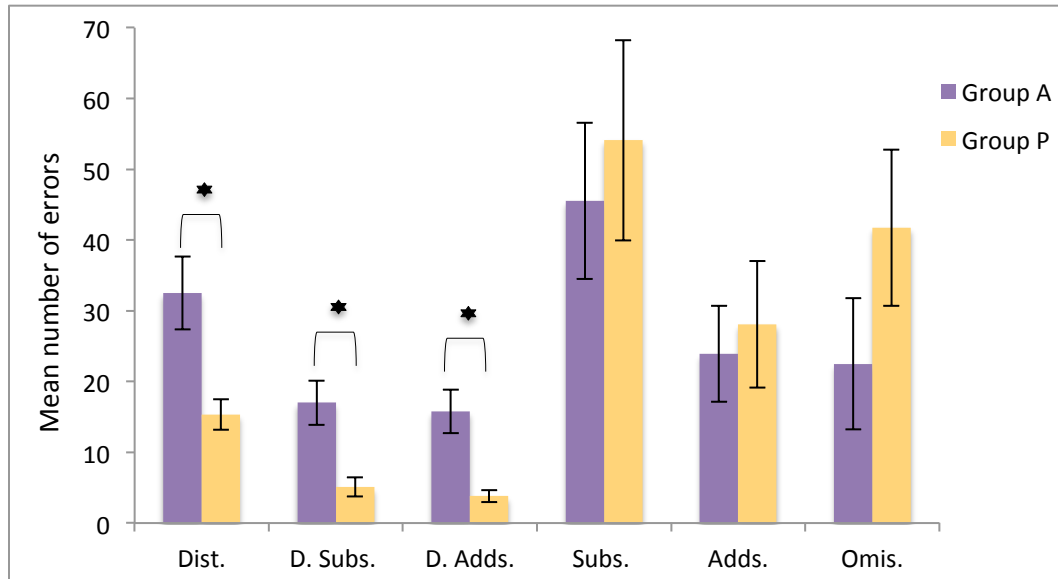


Figure 24. Group Differences in the Types of Errors Produced in the Blocked Condition

Post-Hoc Analyses

After reviewing the results of the current study with the committee two post-hoc analyses were suggested: 1) calculating error consistency for motor and linguistic errors separately, and 2) alternative calculations for consistency of error location and variability of error type. Preliminary results are reported in this section. See Appendix III for rationale, methods, and areas of concern.

Post-Hoc 1 (Preliminary Results)

The first post-hoc analysis attempts to isolate motor planning/programming errors from linguistic processing errors by calculating error consistency for motor and linguistic data separately. Difficulty arises in the ability to reliably separate motor from linguistic errors. Therefore, only errors that are most reliably attributed to breakdown at each level were selected for this analysis and include sequential substitution

errors, such as anticipations, perseverations, and transposition errors (impaired phonological retrieval) and distortion errors (impaired motor planning/programming).

The same calculations described above for the consistency of error location and variability of error type was performed on this data. Independent-samples t-tests were performed to examine between group differences in performance on the consistency of error location and variability of error type for sound distortion errors and sequential phonemic errors, separately. For sound distortion errors, there were no significant differences between Group A (M= 49.09%; SD= 17.52) and Group P (M= 44.45%; SD= 24.89) on consistency of error location; $t(17.950)=0.498$, $p= 0.630$. There were no significant differences between Group A (M= 18.43%; SD= 23.75) and Group P (M= 10.70; SD= 12.40%) on variability of error type; $t(13.852)=0.901$, $p= 0.383$. For sequential phonemic errors, there were no significant differences between Group A (M= 55.66%; SD= 35.55) and Group P (M= 39.40%; SD= 47.30) on the consistency of error location; $t(18.396)=0.896$, $p= 0.382$. Independent-samples t-test for the variability of error type could not be computed for either group because neither group demonstrated variability (M= 0.00%) (*these results likely reflect the small sample from which sequential errors were examined*). Since no between group differences were observed, groups were collapsed and independent-samples t-tests were performed to examine differences between error types (i.e., sound distortions and sequential phonemic errors), across groups, on the consistency of error location and variability of error type for individuals with aphasia with and without AOS. There was no significant difference between sound distortion errors (M= 46.66%; SD= 21.29) and sequential phonemic errors (M= 47.14%; SD= 41.91) on the consistency of error location; $t(29.680)=-0.047$, $p= .0963$, and a significant difference between sound distortion errors (M= 14.77%; SD= 19.13) and sequential phonemic errors (M= 0.00%; SD= 0.00) on variability of error type; $t(18.00)=3.365$, $p= 0.003$. Preliminary results suggest that distortion errors demonstrate greater variability of error type compared to sequential substitutions in participants with AOS and aphasia and aphasia with PP (*results should be interpreted with extreme caution as they are based on a small sample of specific error types pulled from a larger sample of sound errors*).

Post-Hoc 2 (Preliminary Results)

Two alternative calculations were employed to examine the consistency of error location and variability of error type in Group A and Group P. All error types were included (unlike Post Hoc – 1). Consistency of error location was calculated as the number of matches (errors that occurred in the same location) over the number of possible matches (See Figure 25 in Appendix IV). For variability of error type, change scores were calculated to capture the extent in which new error types occurred across trials. Specifically, this analysis compares the first production to subsequent productions (e.g., 2nd – 5th productions) with the goal of capturing the extent of change, in regard to error type categories, from the first production onward (See Table 7 in Appendix IV). If the same error is produced across productions then no points are given. However, if a new error type arises one point is given per occurrence of each new error type that surfaces on subsequent trials.

Independent-samples t-tests were conducted to examine between group differences in performance on the consistency of error location and variability of error type. There were no significant differences between Group A (M= 0.30; SD= 0.15) and Group P (M= 0.37; SD= 0.19) on consistency of error location; $t(18.580)=-0.942$, $p= 0.358$. There were no significant differences between Group A (M= 1.88; SD= 0.64) and Group P (M= 1.67; SD= 0.66) on variability of error type; $t(18.919)=0.749$, $p= 0.463$. Preliminary results for consistency of error location are in agreement with previous findings and indicate that individuals with AOS and aphasia perform similarly to individuals with aphasia with PP. The results of error type indicate that change scores do not differentiate between groups.

CHAPTER 5

Discussion

The primary aim of the present study was to examine the nature error consistency in two clinical populations: individuals with AOS and aphasia and individuals with aphasia with PP – while replicating the procedures performed in an influential study by McNeil et al. (1995). To that end, the consistency of error location and variability of error type was examined for 21 participants during repetition of 30 multisyllabic words across five trials. The influence of error rate, severity of impairment, and stimulus presentation condition on group performance was also explored, as well as group differences in the types of errors produced. This study aims to inform clinical diagnostic procedures to improve the differential diagnosis of AOS and aphasia with PP, as well as contribute to models of speech production. The following discussion will address the results of each research question separately. An integrated discussion of the results is provided under “General Discussion.”

Research Question 1: Error Consistency

The first research question addressed group differences in the consistency of error location and variability of error type during the repetition of multisyllabic words presented in a blocked presentation condition. The results show that groups performed similarly on the consistency of error location, but differed on the variability of error type. Specifically, Group A demonstrated greater variability of error type compared to Group P. These findings differ from the findings of McNeil et al. (1995), but are similar to those reported by Haley and colleagues (2013), specifically for the small comparison group including individuals with AOS and nonfluent aphasia and individuals with CA (see the description of Haley et al., 2013 under “Previous Investigation of Error Consistency”).

The finding that Group A and Group P performed similarly in the consistency in which errors occurred across multiple trials may be attributed to the co-occurring aphasia in Group A. Individuals in both groups exhibited aphasia with PP, implying that the same mechanisms are involved with regard to linguistic breakdown. Both groups demonstrate impaired phonological activation resulting from interference (spreading activation, preceding words, extraneous cognition and perception; Dell, 1986, 1988). These findings are similar to those reported by Haley et al., 2013, which compared similar patient

populations (AOS with aphasia and aphasia with PP). Furthermore, Group P performed similarly to participants with CA in the 1995 study by McNeil and colleagues. In addition, the stimuli employed in this investigation were created with the aim to elicit errors in individuals with AOS and individuals with aphasia. For example, previous investigations suggest that clusters result in greater occurrence of errors than singletons in individuals with AOS (Canter et al., 1985; Odell, 2002; Odell et al., 1990) and individuals with aphasia with PP (Canter et al., 1985; Caplan, 1986), and multisyllabic words stimulate errors relative to monosyllabic words in AOS (Marquardt et al., 2010; Strand & McNeil, 1996) and aphasia with PP (Kohn & Smith, 1995; Odell et al., 1991). Thus, the complex stimuli employed in this study may have influenced more consistent error production in both groups. It is important to note that Group P demonstrated a wider range of variability across participants compared to Group A on the consistency of error location (29-92% and 62-89%, respectively; Figure 12). This variability can be explained by error rate and is addressed in Research Question 2. In particular, the three participants that performed in the lower range (between 29% and 41% consistency of error location) are the same three participants in the lower error rate group.

The finding that Group A demonstrated greater variability of error type compared to Group P is also attributed to the co-occurring aphasia in Group A. The errors produced by individuals with AOS and aphasia result from damage to two different mechanisms (linguistic processing and speech motor control) and reflect a variety of sources of interference (e.g., spreading activation, previous spoken words, extraneous cognition and perception, variability of feedforward control, and motor complexity) (Dell, 1986, 1988; Guenther, 2006; van der Merwe, 1997, 2009). Thus, individuals with AOS and aphasia have the opportunity to produce a greater range of errors compared to individuals with aphasia without AOS. Individuals in Group A demonstrated a variety of error types, including phonetic errors (e.g., sound distortions), phonemic errors (e.g., sequential and nonsequential substitution errors), a combination of the two (e.g., distorted anticipatory errors), and errors that can be attributed to disruption at either level of the speech production network (e.g., omissions and additions). These findings are supported by previous investigations (Haley et al., 2013) and van der Merwe's Four-level framework (1997, 2009).

Taken together, these results suggest that the characteristic of error consistency, especially consistency of error location, is not beneficial to the differential diagnosis of these two clinical populations. These findings and recommendations are consistent with those of Haley et al. (2013).

Research Question 2: Variables Thought to Influence Error Consistency

The extant literature suggests that error patterns in individuals with AOS and aphasia may be influenced by a number of variables. Specifically, there is evidence that error rate (Haley et al., 2013), severity of impairment (Duffy, 2005; Shuster & Wambaugh, 2008), and stimulus presentation condition (Wambaugh et al., 2004) influence error consistency outcomes in individuals with AOS and individuals with aphasia. These studies, however, have revealed conflicting results that are likely attributed to methodological differences in participant inclusion criteria, definition and assessment of error consistency, and analysis.

Research Question 2a – Error Rate

In this study, individuals in Group P demonstrated greater consistency of error location when they produced higher error rates. A similar, but nonsignificant trend ($p=.055$) was observed for Group A. This finding may reflect an inability to adapt or correct errors across multiple trials as error rate increases. In other words, individuals with low error rates may be less consistent in the location of their errors because they are more likely to correct errors (or attempt to correct errors) over multiple trials compared to individuals with higher error rates. According to Dell and colleagues (1997), the primary difference between the errors made by normal healthy adults and those produced by individuals with aphasia is the frequency in which errors occur. This difference is thought to reflect the effects of brain damage and damage to the linguistic network. Thus, the frequency of PP in aphasia may be suggestive of the extent of damage to the linguistic network – phonological processing in particular. These results are consistent with the findings of Haley et al., 2013, where participants who demonstrated less frequent errors (the “minimally impaired group”) were less consistent in the location of their errors compared to three other groups that produced significantly more frequent errors.

Error rate did not influence variability of error type in either group. These results may be due to the small number of participants included in each error rate group and the disproportionate comparisons

between low and high error rate groups (e.g., Group A: low error rate, n=2, high error rate n=8).

Additionally, a large amount of individual variability was observed for the low error rate groups in Group A and Group P (see Figure 15).

Research Question 2b – Severity of Impairment

Individuals in Group A were more consistent in the location of their errors as severity ratings increased, suggesting that severity of impairment influences the consistency of error location in individuals with AOS and aphasia. Feedback from expert raters suggests that severity ratings for Group A reflect the combined effects of motor and linguistic impairment. After rating each participants communication impairment raters were asked what factors contributed to the given severity rating. For individuals in Group A, a rating of *mild* was primarily supported by the following characteristics: *deficits in speech rate, prosody, and articulation*. A rating of *moderate* was primarily supported by: *impaired prosody, frequent speech errors (distortions, distorted substitutions, schwa insertion), impaired linguistic content, empty or non-specific speech, agrammatism*. A rating of *severe* was supported by: *extreme slow rate, segmented speech, lengthened segments, impaired prosody, motorically battered (i.e., impaired) speech output, distortions, lack of linguistic content, lexical retrieval issues, long pauses, repeated attempts*. Specifically, responses from expert raters suggest that a mild severity rating is attributed to motor characteristics (e.g., slow speech rate, abnormal prosody, sound distortions), whereas more severe ratings were attributed to a combination of linguistic characteristics (e.g., empty or non-specific speech, agrammatism, anomia) and more significant motor characteristics (e.g., extreme slow rate, segmented speech, lengthened segments, impaired prosody, motorically battered speech output). Thus, it appears that as motor and linguistic deficits become more prominent severity ratings and consistency of error location increase. This finding may indicate an inability to adapt or correct errors over consecutive productions as severity increases.

The hypothesis that AOS results from impaired feedforward control is consistent with the behavioral characteristics reported by expert raters (e.g., distortions, slowed rate, impaired prosody). The observation of more significant motor speech impairment in individuals with moderate and severe severity ratings (e.g., motorically impaired speech output) may indicate more substantial damage to the

feedforward control subsystem, and therefore, increased reliance on feedback control. The consistency of error location may have been influenced by the combined effects of increased reliance on feedback control and the complexity of the stimuli employed in this study, as complex stimuli may tax the feedback control subsystem. Thus, as reliance on feedback control increases, complex stimuli are more likely to facilitate breakdown, leading to more consistent and predictable errors.

Severity of impairment did not influence the consistency of error location in Group P, these results may be indicative of the limited range of severity within this group (e.g., mild and moderate only). In addition, it is possible that a greater range of severity was perceived in Group A because individuals demonstrated both motor and linguistic deficits. For individuals in Group P a rating of *mild* was primarily supported by the following characteristics: *good to mildly slow rate, good prosody, good articulation, no distortions, good linguistic content, some pausing or linguistic delay, infrequent substitution errors*. A rating of *moderate* was characterized by *infrequent articulation errors, impaired linguistic retrieval, long pauses, empty or non-specific speech, poor verbal working memory on repetition tasks, repeated attempts, syllable deletions, increased errors with increased word length, agrammatism*. Interestingly, for Group P raters were more likely to comment on participant strengths before they addressed weaknesses, especially for the mild group. Some of these characteristics are shared with those reported for Group A, but characteristics such as poor verbal short-term memory and syllable deletions were not observed in Group A. The occurrence of poor verbal short-term memory and syllable deletions may have been overlooked or masked as a result of the motor impairment in Group A – or on the other hand may be truly unique to the participants included in Group P. It is important to note that once again individuals in Group P demonstrated a wide range of individual variability, especially those given a severity rating of mild.

Severity of impairment did not influence variability of error type for either group. In other words, as severity ratings increased, there was no significant change in the variability in which error types occurred across trials. Results suggest that the extent of damage to the linguistic and/or motor speech network does not influence error type patterns.

Research Question 2c – Stimulus Presentation Condition

Stimulus presentation condition did not influence participant performance on measures of error location or type for both Group A and Group P. It was hypothesized that errors would be more consistent in the blocked presentation condition compared to the random presentation condition, especially for Group P. The results of this study are not consistent with this hypothesis. In particular, stimulus presentation condition may not influence error consistency in individuals with AOS and aphasia (as reported by Mauszycki et al., 2010a, 2010b, 2012). With regard to aphasia with PP, the group data indicate that presentation condition may not influence error consistency. However, when examining individual performance, it appears that presentation condition may influence error consistency in some individuals.

Results from the limb motor learning literature suggest that stimulus presentation condition may influence the way in which motor plans/programs are retrieved (Schmidt & Lee, 2005). It is hypothesized that stimuli presented in a blocked condition require the execution of the same motor program for across consecutive trials (e.g., BBBB, AAAA, CCCC); however, stimuli presented in a random presentation condition require the retrieval and/or construction of a different motor program on every trial (e.g., ABAC, BCAC, CABA; Knock et al., 2000). Thus, it is plausible that stimulus presentation condition may influence the retrieval of motor plans/programs for speech production in individuals with AOS. The results of this investigation, however, indicate that this was not the case. For these participants, findings show that stimulus presentation condition did not influence error consistency outcomes in Group A. In fact, the individual data, Figures 19 and 22, show very little deviation in participant performance across presentation conditions.

In the context of the DIVA model, results suggest that stimulus presentation condition does not impact feedback or feedforward control subsystems. Specifically, for individuals with AOS, access to feedforward commands and/or the performance of the feedback control subsystem is not influenced by stimulus presentation condition. Instead, the occurrence and manner in which errors occur in AOS may be influenced by task complexity, where more complex stimuli tax the unsophisticated feedback control subsystem and result in more predictable errors.

The IA model (Dell, 1986) suggests that stimulus presentation may influence the retrieval of phonemes for speech production. More specifically, stimuli presented in a blocked condition require the maintained activation of the same phonological representation across consecutive trials, whereas stimuli presented in a random condition require the activation of new phonological representations on every trial. Therefore, stimulus presentation condition may influence linguistic retrieval in individuals with aphasia. It was hypothesized that a blocked presentation condition would promote more consistent productions on consecutive trials compared to a random presentation condition because the activation of new phonological representations provides opportunity for more variable productions (correct vs. incorrect) and sources of interference on each trial of the same stimulus (i.e., opportunity for different errors). The individual data for Group P, Figures 20 and 23, illustrate some individuals demonstrate more consistent performance in the blocked condition, for both error location and type, compared to the random condition. As previously mentioned, the individual variability observed in Group P may be reflective of heterogeneity of aphasia included in this study. A closer look at individual performance would be beneficial to understanding underlying mechanisms that contribute to the heterogeneity within this population.

Research Question 3 – Error Type

Group A produced a significantly greater number of phonetic errors compared to Group P. In particular, individuals in Group A produced significantly more sound distortions, distorted substitutions, and distorted additions compared to individuals in Group P. This finding is consistent with previous literature which identifies AOS a motor speech disorder characterized by sound distortions, distorted substitutions (Haley et al., 2001; Mauszycki et al., 2010a, 2010b, 2012; Odell et al., 1990; Odell et al., 1991; Shuster & Wambaugh, 2000; Square et al., 1982), and distorted additions (Duffy et al., 2015). There is theoretical support for the frequent occurrence of these phonetic errors in AOS. The DIVA model and the Four Level Framework support the occurrence of distortion errors as a characteristic of impaired feedforward control or motor planning, respectively (Guenther, 2006; van der Mewer, 1997, 2009). Sound distortion errors may reflect small spatiotemporal errors caused by the imprecision of feedback control (Jacks, 2008; Maas, et al., 2012; Tourville & Guenther, 2011), an inability to successfully recall the core motor plans/or identify motor goals, successfully sequence the movements for speech sounds, adapt core motor plans, and control inter-articulatory synchronization (van der Merwe, 1997, 2009). Distorted

substitution errors may reflect an extreme distortion error or the combination of linguistic and motor disruption (van der Merwe, 1997; 2009). Finally, distorted additions may also reflect the combination of linguistic and motor disruption, intrusive schwas, and/or be a product of groping (Clark, Hanley, Duffy, Strand, & Josephs, 2015).

Although Group A produced significantly more phonetic errors; phonetic errors also occurred in group P. This finding is not surprising, as neurologically healthy adults occasionally produce phonetic errors, most often during difficult speech tasks, such as tongue twister paradigms (Goldrick & Blumstein, 2006; McMillan & Corley, 2010), speeded repetition of word pairs (Goldstein, Pouplier, Chen, Saltzman, & Byrd, 2007), and SLIP (spoonerisms of laboratory induced predisposition; Pouplier, 2007). Thus, it is likely that factors contributing to phonetic breakdown in individuals without motor speech impairments include fast or rushed speech rate and /or phrases with competing or similar sounds. In the current study, participants were asked to repeat complex multisyllabic words five times in a row “as quickly and as clearly as possible.” This elicitation method, in combination with complex stimuli, may have facilitated the occurrence of phonetic errors in Group P. In addition, some individuals in Group P demonstrated significant effort during speech tasks, resulting in straining upon production. This effort may reflect difficulty in word finding and/or anxiety. Two participants demonstrated excessive effort as a result of anxiety during speech production tasks. For one participant anxiety was only associated with testing and the experimental task, and was not observed during spontaneous speech or more relaxed situations. For the other participant, the observed effort was not only associated with performance anxiety, but became more obvious during word finding difficulties as the participant would become extremely frustrated and would strain her voice while repeating stereotypies (this participant had a medical history of anxiety, but was medicated during the study). Data from these two participants may have added significantly to the phonetic errors observe in Group P. Although the purpose of the current study was to examine group data, further examination of individual data is warranted.

Group A and Group P did not differ in regard to the number of phonemic errors, which included clean sound substitutions (sequential and nonsequential), additions, and omissions. This finding is not surprising considering that individuals in both groups demonstrated aphasia characterized by PP

(although the frequency of PP different across participants within each group). Furthermore, nonsequential substitution, omission, and addition errors have been observed in individuals with pure AOS (Itoh, Sasanuma, & Ushijima, 1979; Odell et al., 1990, 1991). Therefore, these errors may reflect impaired planning/programming as well as impaired linguistic processing.

General Discussion

The IA model (Dell 1986, 1988), DIVA model (Guenther, 2006), and the Four-level Framework (van der Merwe, 1997, 2009) provide strong support for the speech characteristics observed in individuals with AOS and aphasia with PP, such as sound distortion errors, distorted substitutions, and phonemic paraphasias. In particular, the Four-level Framework provides convincing evidence for the differentiation of these two disorders based on their speech characteristics and locus of impairment. According to the framework, individuals with AOS demonstrate speech characteristics associated with impaired linguistic-symbolic planning (phonemic errors) and motor planning (sound distortions). Although not as comprehensive as the Four-level Framework (with respect to explaining disordered populations), the IA model and the DIVA model support the claims made by this framework in regard to the differentiation of AOS from aphasia with PP. Furthermore, the Four-level Framework addresses the combined effects of linguistic and motor impairment (e.g., co-occurrence linguistic and motor errors.), thereby supporting the co-occurrence of AOS and aphasia with PP. The results of the error type analysis in this study support these claims. The Four-level Framework does not speak to the nature of error consistency in these populations, but the IA and DIVA models provide some insight into this phenomenon. More specifically, the models indicate that errors in AOS and aphasia with PP are variable (i.e., not 100% consistent), which is supported by the results of this study and previous investigations (Haley et al., 2013; McNeil et al., 1995; Miller, 1992). Because neither model addresses both linguistic or motor processes involved in speech production, they are unable to address the relative nature of error consistency in AOS compared to aphasia with PP, as well as the nature of error consistency in AOS and concomitant aphasia.

The results of this investigation show that individuals with AOS and aphasia and individuals with aphasia with PP perform similarly in the consistency of error location. As mentioned above, these findings are attributed to the effects of impaired phonological retrieval in each group and are supported by the

literature (Haley et al., 2013; Miller, 1992). Findings suggest that error rate is an influential factor in error consistency in individuals with aphasia with PP. Specifically, as error rate increases, so does the consistency of error location. In the context of the IA model, phonologic errors result from noise and interference that leads to the activation of an incorrect phoneme during phonemic encoding (Dell, 1988). Thus, higher error rates indicate greater noise/interference in the phonological network, and the greater the noise/interference the greater the consistency of error location. Therefore, high levels of noise and/or interference suggest that an error is more likely to occur within the same location of a word across trials. It is also possible that the linguistic network is less able to correct the error. This does not imply, however, that the same incorrect phoneme is activated across trials (remember, error rate did not influence error variability). Rather, it is the same slot in the phonological frame that is constantly being filled in error. This may be explained by spread of activation, where the retrieval of a phoneme for a particular slot is repeatedly in error because noise in the system prevents activation from getting to where it needs to go in the appropriate amount of time. Impaired maintenance or phonological storage may be another example, where the system is unable to “hold on” to the initial activated phoneme in a particular slot, therefore, constantly filling the slot in error. For example, an individual with verbal short-term memory deficits may have difficulty correctly filling slots in the phonological frame as a result of impaired activation maintenance or decay rate (Martin & Gupta, 2004). One factor that may have influenced the consistency of error location in individuals with high error rates (i.e., large amounts of noise and interference in the phonological network) is the stimuli employed in this study. The literature suggests that phonological errors are more likely to occur on longer words (e.g., Ellis, Miller, & Sin, 1983; Goodglass, Kaplan, Weintraub, & Ackerman, 1976; Kohn, 1989; Nickels, 1995; Shallice, Rumati, & Zadini, 2000). In particular, a study by Nickels and Howard (2004) found that the number of phonemes in a word influences phonological errors in aphasia. Specifically, results of Nickels and Howard (2004) indicate that a phonological error is more likely to occur when a word has more phonemes. In the context of the IA model, the more phonemes requiring activation and placement into slots in the phonological frame may increase processing load, time course required for activation spread, and opportunities for interference (Dell, 1988; Levelt, 1999; Rolefs, 2002). Thus, stimuli made up of long words, or a large number of

phonemes, (such as that employed in this study) may facilitate error production, and perhaps error location, in individuals with more significant phonological retrieval deficits.

The DIVA model does not directly address error location; however, the results of this investigation indicate that severity of impairment influences the consistency of error location in individuals with AOS and aphasia. This finding can be explained by the feedforward hypothesis of AOS, where individuals with AOS rely on the feedback control subsystem because the feedforward control subsystem is impaired. Individuals who demonstrate more severe motor planning deficits (e.g., motorically battered speech output) likely have greater impairment to feedforward control, requiring even more assistance from feedback control. This increased dependence on the less skilled subsystem may lead to increased speech errors. According to the literature, the complexity of speech tasks influence breakdown in AOS (e.g., consonant clusters will result in more errors than singleton consonants; Darley, 1982). The stimuli employed in this investigation were created with the aim to facilitate errors in individuals with AOS and aphasia. Thus, individuals with a greater impairment, i.e. greater reliance on the feedback control subsystem, are more likely to be influenced or challenged by these stimuli. In other words, the complex stimuli included in this investigation may tax the feedback control subsystem more consistently in individuals who are more impaired, thus resulting in more consistent and predictable errors.

The results of error type indicate that individuals with AOS and aphasia demonstrate more variable errors compared to individuals with aphasia with PP. These findings can be explained by combining the phonologic errors and noise and sources of interference (spreading activation, previous and upcoming stimuli, extraneous cognition) proposed by the IA model and the distortion errors and impaired feedforward hypothesis of AOS supported by the DIVA model (reliance on an less skilled and more variable subsystem). The layering of these models support a range of error types (also supported by the Four-level Framework; van der Merwe, 1997, 2009), such as sequential and nonsequential phonemic paraphasias, additions, omissions, distortions, distorted substitutions, and distorted additions, and a variety of sources of interference/breakdown. The results of this study indicate that variability of error type is not influenced by the extent of damage to the linguistic and/or motor speech networks,

rather, it is the combined impairment to these two systems that contribute to the variability in which errors are produced in individuals with AOS and aphasia.

Clinical Implications

As a result of these findings, it is recommended that the characteristic of error consistency not be considered instrumental in the differential diagnosis of AOS and aphasia from aphasia with PP. Moreover, light has been shed on a common misnomer that bears clarification. Upon conversations with clinicians and researchers in the field it has become apparent that the commonly used term “variability of error type” is misleading, as it does not refer to error type categories, which is often the assumption, but instead the reoccurrence of a specific error (e.g., devoiced /d/) across repeated trials. It is recommended that this terminology be changed to “variability of errors” to better describe the phenomena being measured.

Findings support the continued use of distortion errors in the differential diagnosis of these two clinical populations. The occurrence of distortion errors, however, is not non-existent in individuals with aphasia without AOS and is a primary characteristic of dysarthria. Thus, the observation of distortions alone does not warrant a diagnosis of AOS, but rather a combination or pattern of errors (e.g., distortions, slow rate, and abnormal prosody) observed across a variety of different speech tasks.

The differential diagnosis of AOS and aphasia from aphasia with PP continues to be a challenging task for clinicians. Although the results of this study identify characteristics that are both beneficial and unhelpful in differentiating these groups, further investigation is warranted. Specifically, studies improving upon our current diagnostic criteria and diagnostic process are of great value. The development of a reliable assessment tool that can be utilized across clinical settings is desperately needed. Further, examining the way in which diagnosis is currently performed by practicing clinicians may provide insight into the triumphs of differential diagnosis in the field. In addition to the similarities between these two populations, misconceptions about AOS and aphasia with PP, confusion surrounding primary characteristics, and the continued use of outdated diagnostic criteria, likely contribute to this challenge.

Study Limitations

Limitations of the current study are discussed below. If addressed, these limitations may strengthen future investigations of error consistency and/ or differential diagnosis in these populations.

Sample size is often an issue in investigations focused on rare or special populations. The current investigation consisted of 21 participants – ten individuals with AOS and concomitant aphasia and 11 individuals with aphasia with PP. When examining the influence of error rate (Research Question 2a) and severity of impairment (Research Question 2b) on error consistency, each diagnostic group was divided into even smaller error rate and severity groups. For example, when examining the influence of error rate on error consistency Group A was then divided into a low error rate group (n=2) and a high error rate group (n=8). The low error rate group consisted of less than half of the number of participants included in the high error rate group. A similar issue occurred when examining severity of impairment, where there was a disproportionate number of participants across severity of impairment groups (e.g., mild, moderate, severe). A larger sample size may have lead to more balanced error rate and severity impairment groups, revealed more robust outcomes, and permitted more advanced statistical procedures (e.g., multivariate linear or logistic regression; Hackshaw, 2008). Results from studies with small samples sizes do not always yield reliable or precise estimates reflective of a specified population, and therefore, should be interpreted with caution (Hackshaw, 2008).

In the current study, sequential substitution errors occurred infrequently compared to nonsequential substitution errors. For example, sequential errors made up 4.05% of errors for Group P, whereas nonsequential errors made up 32.45% of errors. The low occurrence of sequential substitution errors reported in this study might have been influenced by the stimuli employed in this investigation and/or the error coding procedure. First, the stimuli included in the present study consisted of multisyllabic words, emulating past investigations of error consistency. Much of the literature on sequential errors, however, describes errors occurring on two word utterances or short phrases, e.g., barn door → darn bore (Dell, 1986, 1988; Fromkin, 1971, 1973; Shattuck-Hufnagle, 1979). Thus, the stimuli employed in the current investigation may not facilitate the occurrence of sequential errors at the same frequency as two word utterances or short phrases. Second, the manner in which errors were coded may have influenced

the frequency in which sequential errors were reported. In the current study participant responses were coded for errors based on the target word. For example, if the target word was *merchant* and the participant response was *berchant* the error was coded as a nonsequential substitution error. According to Dell (1986), upcoming or preceding words may influence linguistic errors; therefore, it is possible preceding stimuli influenced the erred response, *berchant*. If the stimulus *blessing* preceded the stimulus *merchant*, the /b/ in *berchant* may actually reflect a perseverative error, i.e., a sequential substitution (the author acknowledges that there are additional explanations for this error). Thus, the error-coding scheme employed in the current study may have influenced the categorization of substitution errors.

Previous studies examining error consistency have been inconsistent or unclear in how a specific error, intrusive schwa, has been treated. An intrusive schwa is an /a/ inserted between syllables or consonant clusters, believed to result from impaired timing of the articulators during sound production (McNeil et al., 2004). Specifically, past investigations have either excluded this error from the performed analysis (Haley et al., 2013) or did not address the presence of schwas in the publication (McNeil et al., 1995; Miller 1992). Furthermore, the few studies that have examined error type categories in AOS, and have employed narrow phonetic transcription, have been inconsistent or unclear in how intrusive schwas have been coded. For example Odell and colleagues (1990, 1991) coded schwas as distortion errors, whereas Mauszycki et al. (2010a, 2010b, 2012) and Square et al. (1982) did not address the presence of these errors in their data. In the current study schwas were included in the analysis. The decision to include schwas in this analysis was supported by the belief that these errors are clinically perceptible speech errors characteristic of AOS (Duffy, 2013). In regard to the error type analysis, Research Question 3, a specified error-coding category was not assigned to schwas; rather the error code was determined by the transcriber's perception of the error. Therefore, if a schwa was perceived as a distortion, distorted addition, or addition error it was coded as such. It is possible that the inclusion of schwas in the error consistency and error type analyses influenced findings. Future investigations should take this into consideration and strive to develop consistency in which these errors are analyzed.

Lastly, although not the purpose of this study, these findings do not address the nature of error consistency in AOS alone. Further examination of the nature of error consistency in relatively pure AOS is

valuable to our understanding of the underlying impaired mechanism and would inform models of speech production.

Future Directions

In addition to the post-hoc analyses reported in the “Results” section, there are many other ways in which to examine this data. A few of areas of interest are explained below.

First, the author plans to expand on the work of Odell, 1990 and perform a more detailed investigation of error type, extending beyond error categories to examine error type patterns, such as prominent types of distortion and substitution errors (e.g., voicing errors), as well as within word variables that influence breakdown, such as word and syllable location (e.g., onset vs. coda, singletons vs. clusters). This information may aid in the diagnosis process, inform models of disordered speech, and direct treatment.

Another area of interest is adaption across trials, as well as over time. Specifically, the author is interested in assessing the presence of an adaptation effect, not only for the current data (i.e., repeated trials within one session), but also across sampling occasions (expanding on the work of Mauszycki, 2010a, 2010b, 2012). There is theoretical support that repeated exposure could lead to motor learning in individuals with AOS, without external feedback (Guenther, 2006; Schmidt & Lee, 2005; Tourville & Guenther, 2011). Examining adaptation could provide valuable information about error awareness and learning without feedback, as well as differences in learning in these two populations.

Lastly, as mentioned throughout the document, individual variability was observed in each group, especially in Group P. The author would like to further examine individual variables that may influence participant performance on measures of error consistency, such as verbal working memory (Martin, Ayala, & Saffran, 2002; Martin & Gupta, 2004; Martin & Saffran, 1997) and error awareness (Bailey et al., in press; Goodglass, 1992; Sullivan, Fisher, & Marshall, 1986).

CHAPTER 6

Conclusion

The primary aim of the present study was to determine whether error consistency helps distinguish between two clinical populations. The finding that groups performed similarly in the consistency of error location is in line with the results of Haley et al. (2013) and Miller (1992). Although variability of error type differentiated groups in the present study, this was only observed in the blocked condition and there was overlap in participant performance across groups. In addition, these findings demonstrate the opposite effect of that reported by McNeil et al (1995), when comparing the performance of individuals with pure AOS and CA. Overall, these findings provide sufficient evidence that error consistency is not a valid measure in which to differentiate individuals with AOS and aphasia from aphasia with PP. The external validity of the present study is strengthened by the use of a participant sample that demonstrates a range of impairment and representative of the clinical populations served by SLPs. These findings, however, do not address error consistency in rare diagnostic entities, such as AOS without concomitant aphasia or the usefulness of error consistency in the differential diagnosis of dysarthria from AOS and/or aphasia. Lastly, results of the error type analysis support the continued classification of distortion errors as a primary characteristic of AOS.

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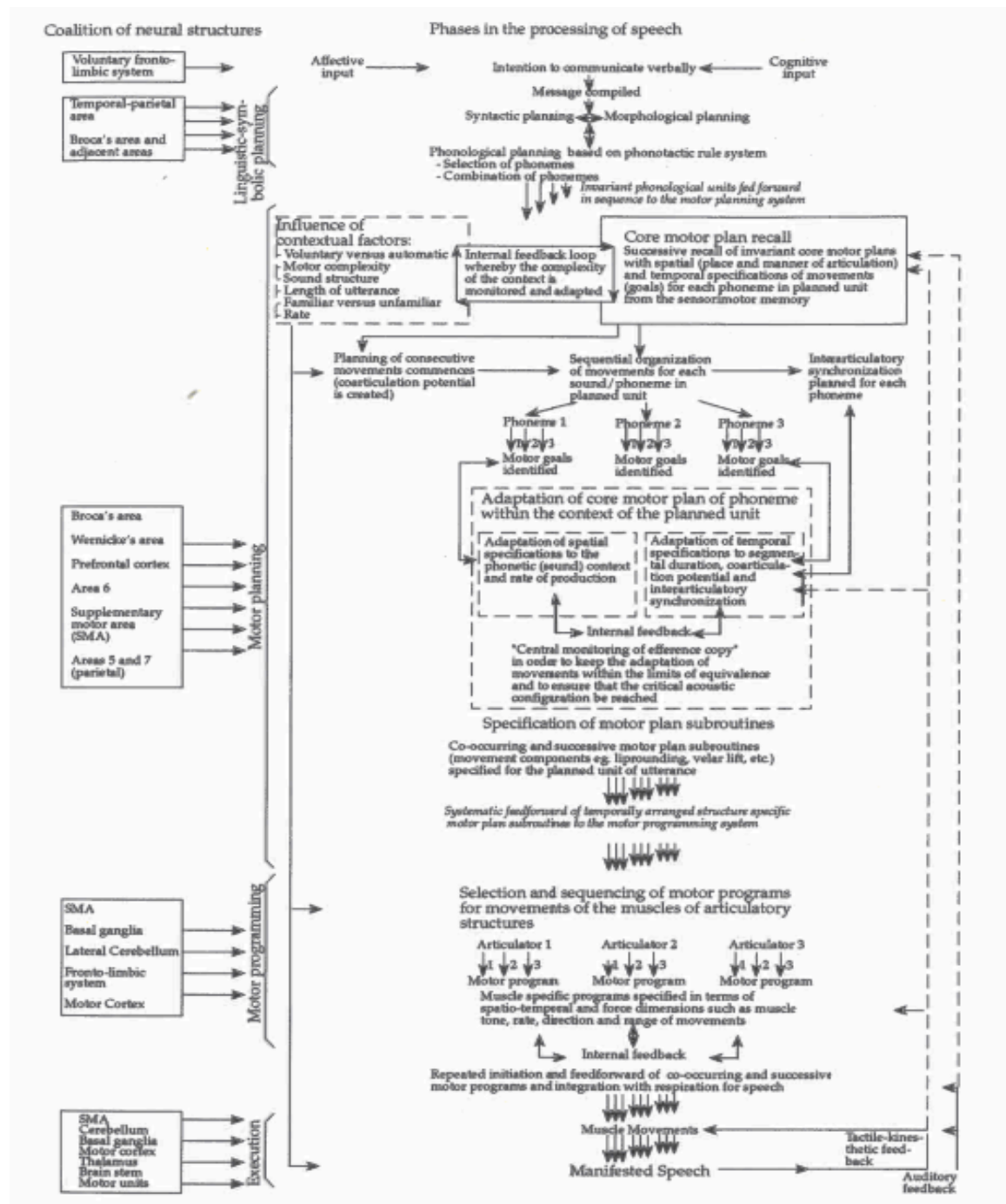
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Appendices

Appendix I: Four-Level Framework



A Theoretical Framework for the Characterization of Pathological Speech Sensorimotor Control (taken from van der Merwe, 1997).

Appendix II: Stimuli lists

Number	List A Blocked Condition	List B Random Condition
1	Blessing	Aspect
2	Exchange	Creature
3	Freedom	Discharge
4	Merchant	Garment
5	Platform	Silence
6	Announcement	Compliment
7	Consequence	Destruction
8	Detective	Employment
9	Procession	Photograph
10	Sympathy	Preference
11	Civilization	Administration
12	Continuation	Consideration
13	Inefficiency	Electricity
14	Investigation	Individual
15	Undergraduate	Justification

Appendix III: Consensus Rules for Phonetic Transcription

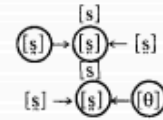
Table from Shirberg et al., 1984.

Rule	Descriptive Label	Definition/Content	Example
<i>Rules That Never Involve Circling</i>			
1.	"Sure, I can hear it"	If one transcriber immediately "hears" and accepts the other transcriber's transcription as more accurate, select that transcription as the CT.	
2.	"Let's keep it simple"	If either transcription contains a List A diacritic (i.e., a nonerror diacritic), select as the CT the transcription that does not contain the nonerror diacritic.	$\begin{array}{c} [s] \\ [s̺] \rightarrow [s] \leftarrow [s] \\ [k] \\ [k̺] \rightarrow [k] \leftarrow [k̺] \end{array}$
3.	"We both hear the diacritic"	If List A diacritics were used in both transcriptions the CT should contain only those included in both transcriptions.	$\begin{array}{c} [z] \\ [z̺] \rightarrow [z] \leftarrow [z̺] \end{array}$
4.	"Let's be conservative—nasals"	If optional nasal diacritics are used in both transcriptions, select as the CT the more conservative diacritic. The conservative hierarchy from most to least conservative is: [̃] (e.g., [ñ]); [̄] ([̄] is used for <i>weak release</i>); [̆].	$\begin{array}{c} [m] \\ [m̃] \rightarrow [m̄] \leftarrow [m̆] \end{array}$
5.	"Let's be conservative—stops"	If a final stop is perceived as unreleased in one transcription (i.e., is barely evident) and perceived as released in the other (with or without a List A diacritic), select as the CT the unreleased stop.	$\begin{array}{c} [d] \\ [d̚] \rightarrow [d̚] \leftarrow [d̚] \\ [d] \\ [d̚] \rightarrow [d̚] \leftarrow [d̚] \end{array}$
6.	"Let's use the more probable diacritic"	If both transcriptions include List B diacritics, select as the CT the distortion highest on the distortion hierarchy. The distortion hierarchy is based on the frequency of error type, with the most frequently occurring error type highest on the hierarchy: /s/: [s̺], [s̺̄], [s̺̆]; /r/: [r̺], [r̺̄]; /l/: [l̺], [l̺̄].	$\begin{array}{c} [s] \\ [s̺] \rightarrow [s] \leftarrow [s] \\ [r] \\ [r̺] \rightarrow [r] \leftarrow [r] \\ [l] \\ [l̺] \rightarrow [l̺] \leftarrow [l̺] \end{array}$
<i>Rules That Always Involve Circling</i>			
7.	"Corrects outrank distortions"	If one transcription is a correct segment and the other a distortion (i.e., contains a List B diacritic), select as the CT the correct segment and circle it.	$[z] \rightarrow \textcircled{[z]} \leftarrow [z̺]$
8.	"Corrects outrank deletions"	If one transcription is a correct segment and the other is a deletion, select as the CT the correct segment and circle it.	$[l] \rightarrow \textcircled{[l]} \leftarrow [∅]$
9.	"Distortions outrank substitutions"	If one transcription is a phonemic distortion (i.e., contains a List B diacritic) and the other is a phonemic substitution, select as the CT the distortion and circle it.	$[ʒ] \rightarrow \textcircled{[ʒ̺]} \leftarrow [ʒ̄]$
10.	"Distortions outrank deletions"	If one transcription is a distortion and the other a deletion, select as the CT the distortion and circle it.	$[l̺] \rightarrow \textcircled{[l̺]} \leftarrow [∅]$
11.	"Substitutions outrank deletions"	If one transcription is a substitution and the other is a deletion, select as the CT the substitution and circle it.	$[w] \rightarrow \textcircled{[w̺]} \leftarrow [∅]$

12. "Let's use the closest substitution"
 If both transcriptions are substitutions, select as the CT the closest substitution to the IS (based on place/manner features) and circle it.

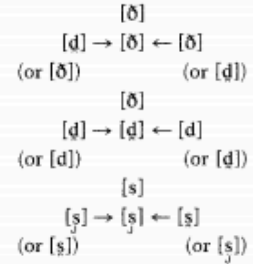


13. "Once circled, always circled"
 If either (or both) of the transcribers has circled his/her transcription before or after replay, the CT also must be circled, even if transcribers agree on the segment. If the segments are different, apply the appropriate consensus rule and circle the CT.
 (Consensus Rule 9 also applies)

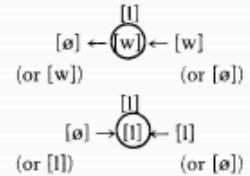


Rules That Sometimes Involve Circling

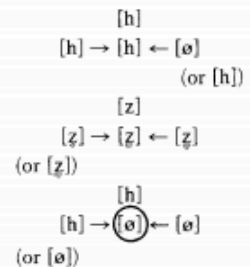
14. "Sure, we both can hear it"
 If (a) both transcribers can hear both transcriptions, (b) the transcriptions are functionally equivalent, and (c) neither transcription is a deletion, select as the CT the transcription that is closest (place/manner features) to the IS.



Deletion Exception: If (a) both transcribers can "hear" both transcriptions, and (b) only one of the transcriptions is a deletion, select as the CT the nondeletion transcription and circle it.

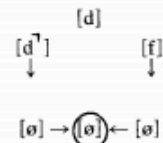


15. "I hear yours, but you don't hear mine."
 If (a) only one of the transcribers can "hear" both his/her own transcription and the other transcriber's, and (b) the transcription heard by both is not a deletion, select as the CT the transcription heard by both transcribers.



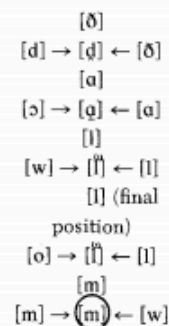
Deletion Exception: If the transcription both transcribers can hear is a deletion, select it as the CT and circle it.

16. "We both hear something new"
 If (a) on replay both transcribers hear the same transcription, one that is different from either of their original, and (b) neither original transcription was a deletion, select it as the CT.



Deletion Exception: If the new transcription is a deletion, circle it.

17. "Let's find a good compromise"
 If one transcription is the correct IS and the other is a substitution, select as a CT a transcription that is somewhere midway between the two transcriptions.



If a suitable compromise is not possible, select as the CT the correct transcription and circle it.

Appendix IV: Post Hoc Analyses

Post-Hoc 1: Rationale, Methods, and Preliminary Results

The first post-hoc analysis attempts to isolate motor planning/programming errors from linguistic processing errors by calculating error consistency for motor and linguistic data separately. Difficulty arises in the ability to reliably separate motor from linguistic errors. In particular, it is difficult to know for sure the mechanism behind all nonsequential substitution errors additions, omissions, and even distorted substitution errors. There is theoretical support and participant data available to support that nonsequential substitution, addition, and omission errors result from impaired linguistic processing (Blumstein, 1973; Dell, 1986, 1988; Dell et al., 1997; Shattuck-Hufnagel, 1979), however, there is also participant data to support that these errors may also result from impaired motor planning/programming (Itoh et al., 1979; Odell et al., 1990, 1991). According to the DIVA model, impairment to the speech sound map would likely account for perceived substitution errors in AOS (Bohland et al, 2009; Mailend & Maas, 2013). In regard to distorted substitution errors, van der Merwe (1997, 2009) suggests these errors demonstrate breakdown at the linguistic level (e.g., substitution errors) followed by disruption in motor planning/programming (e.g., distortion errors). Distorted substitution errors may also reflect a sound distortion error that crosses phonemic boundaries. It is difficult to reliably categorize these error types as linguistic or motor inaccuracies. Errors that are most reliably attributed to a breakdown in motor planning/programming are distortion errors (McNeil et al., 2004). There is theoretical support and participant data available to support this claim (Jacks, 2008; Maas, et al., 2012; Odell et al., 1990, 1991; Square et al., 1982; Tourville & Guenther, 2011; van der Merwe, 1997, 2009). Errors that are most reliably attributed to a breakdown in linguistic processing – at the phonological level – include sequential substitution errors, such as anticipations, perseverations, and transposition errors. There is theoretical support, participant data, and normal control data to support this claim (Dell, 1986; Dell et al., 1997; Fromkin, 1971; Garrett, 1980, 1984; Monoi, Fukusako, Itoh, & Sasanuma, 1983). Therefore, for this analysis only sound distortion errors and sequential phonemic errors were analyzed.

A trained research assistant – an undergraduate working in the aphasia lab – assisted in this task. To calculate error consistency for motor and linguistic errors separately, the research assistant

revisited the raw data stored in an excel spreadsheet. First, the research assistant combed through the data selecting sound distortion errors only, for both Group A and Group P. Second, the research assistant combed through the data selected sequential phonemic errors only, for both Group A and Group P. Consistency of error location and variability of error type was calculated for distortions and sequential phonemic errors separately – using the same equations employed on the larger data set (same as McNeil et al., 1995).

Independent-samples t-tests were performed to examine between group differences in performance on the consistency of error location and variability of error type for sound distortion errors and sequential phonemic errors, separately. For sound distortion errors, there were no significant differences between Group A (M= 49.09%; SD= 17.52) and Group P (M= 44.45%; SD= 24.89) on consistency of error location; $t(17.950)=0.498$, $p= 0.630$. There were no significant differences between Group A (M= 18.43%; SD= 23.75) and Group P (M= 10.70; SD= 12.40%) on variability of error type; $t(13.852)=0.901$, $p= 0.383$. For sequential phonemic errors, there were no significant differences between Group A (M= 55.66%; SD= 35.55) and Group P (M= 39.40%; SD= 47.30) on the consistency of error location; $t(18.396)=0.8.96$, $p= 0.382$. Independent-samples t-test for the variability of error type could not be computed for either group because neither group demonstrated variability (M= 0.00%). These results likely reflect the small sample from which sequential errors were examined. Since no between group differences were observed, groups were collapsed and independent-samples t-tests were performed to examine differences between error types (i.e., sound distortions and sequential phonemic errors), across groups, on the consistency of error location and variability of error type for individuals with aphasia with and without AOS. There was no significant difference between sound distortion errors (M= 46.66%; SD= 21.29) and sequential phonemic errors (M= 47.14%; SD= 41.91) on the consistency of error location; $t(29.680)=-0.047$, $p= .0.963$, and a significant difference between sound distortion errors (M= 14.77%; SD= 19.13) and sequential phonemic errors (M= 0.00%; SD= 0.00) on variability of error type; $t(18.00)=3.365$, $p= 0.003$.

Preliminary results suggest that distortion errors demonstrate greater variability of error type compared to sequential substitutions in participants with AOS and aphasia and aphasia with PP.

Distortion and sequential substitution errors did not differ in regard to error location. These results should be interpreted with extreme caution as they are based on a small sample of specific error types pulled from a larger sample of sound errors.

There are issues with this analysis that require attention prior to moving forward. First, the author is concerned that the data included in this analysis make up only a small portion of the errors produced by the participants in this study. Furthermore, the error types included in this analysis (sound distortions and sequential substitution errors) are significantly disproportionate in number. Specifically, when totaled across groups sound distortion errors occurred 494 times (this number does not include distorted substitutions or distorted additions), whereas sequential substitution errors only occurred 158 times. The number of errors actually included in the analysis to assess error location and error type is even lower (remember, to assess consistency of error location errors need to occur at least two times in the same segment and to assess variability of error type errors needed to occur at least two times in the same location). This difference in error frequency likely impacts results. In particular, the lack of variability across groups – for variability of error type – may reflect the small number of sequential errors included in the error type analysis, not the nature of error consistency in linguistic processing disorders.

Post-Hoc 2: Rationale, Methods, Preliminary Results

Two alternative calculations were employed to examine the consistency of error location and variability of error type in individuals with AOS and aphasia and individuals with aphasia with PP. Consistency of error location was calculated as the number of matches (errors that occurred in the same location) over the number of possible matches (See Figure 25). For example, across five productions there is opportunity for four matches (e.g., 4/4). If two errors occur in the same location that is 1 match over 4 possible matches (e.g., 1/4). If only one error occurred then there were no matches (e.g., 0/4). This calculation is different from the calculation initially employed in this study (and used by McNeil et al., 1995).

Announcement	ə	'n	aʊ	n	s	m	ə	n	t
Trial 1	æ	'n	aʊ	n	s	m	ə	n	t
Trial 2	æ	'n	aʊ	n	s	m	ə	n	t
Trial 3	ə	'n	aʊ	n	s	m	ə	n	t
Trial 4	æ	'n	aʊ	n	s	m	ə	n	t
Trial 5	æ	'n	aʊ	n	s	m	ə	n	t
Error Location: Number of Matches/ Possible Matches	3/4								

Figure 25. Error Location: Total Matches

To examine variability of error type, change scores were calculated to capture the extent in which new error types occurred across trials. Specifically, this analysis compares the first production to subsequent productions (e.g., 2nd – 5th productions) with the goal of capturing the extent of change, in regard to error type categories, from the first production onward (See Table 7). If the same error is produced across productions then no points are given, but if a new error type arises one point is given per the occurrence of each new error type that surfaces on subsequent trials. Although this analysis is not as sensitive as the previous measure of error type, it is more clinically applicable because it is not requiring clinicians to distinguish between types of distortions being produced (SLPs are not traditionally trained in narrow phonetic transcription).

Table 6. Change score for error type across productions

	Distortion	Distorted Substitution	Distorted Addition	Sequential Substitution	Non sequential Substitution	Addition	Omission
Trial 1	X	X					
Trials 2 – 5	0	0	1		3	0	0
Change Score				4			

The author performed these alternative calculations. Independent-samples t-tests were conducted to examine between group differences in performance on consistency of error location and variability of error type. There were no significant differences between Group A (M= 0.30; SD= 0.15) and

Group P (M= 0.37; SD= 0.19) on consistency of error location; $t(18.580)=-0.942$, $p= 0.358$. There were no significant differences between Group A (M= 1.88; SD= 0.64) and Group P (M= 1.67; SD= 0.66) on variability of error type; $t(18.919)=0.749$, $p= 0.463$. Preliminary results for consistency of error location are in agreement with previous findings and indicate that individuals with AOS and aphasia perform similarly to individuals with aphasia with PP. The results of error type indicate that change scores do not differentiate between groups.

Prior to moving forward with this analysis, there are issues with computing change scores for the variability of error type that require attention. One issue that became apparent during data analysis is that the change score does not reflect the consistency of an error type, only the variability. For example, if a participant produces the same error across all five trials, but also produces a number of new error types, the change score reflects the occurrence of new error types, but not the consistent production of the error produced across all five trials. Another issue is that the change score does not reflect the discontinuation of an error. For example, if an individual produces a distortion error on the first trial but does not produce this error on subsequent trials this change is not reflected in the change score. Similarly, if an individual produces all seven error types during the first production (e.g., distortion, distorted substitution, distorted addition, sequential substitution, nonsequential substitution, addition, and omission errors), but discontinues some of these error types on subsequent trials this change is not reflected by the change score. This individual is given a change score of zero. On the other hand, if an individual does not produce an error on the first production, but produces a distortion and a nonsequential error on all subsequent trials the change score only reflects the difference or change from the first production and does not reflect the consistency in which errors were produced on later trials. This individual is given a change score of two.