Published in final edited form as:

Aphasiology. 2016 ; 30(1): 74–95. doi:10.1080/02687038.2015.1063581.

The effect of speaking rate on serial-order sound-level errors in normal healthy controls and persons with aphasia

Tepanta R. D. Fossett¹, Malcolm R. McNeil^{1,2}, Sheila R. Pratt^{1,2}, Connie A. Tompkins^{1,3}, and Linda I. Shuster⁴

¹Department of Communication Science & Disorders, University of Pittsburgh

²Geriatric Research Education and Clinical Center, VA Pittsburgh Healthcare System

³Center for the Neural Basis of Cognition, University of Pittsburgh and Carnegie Mellon University

⁴Department of Speech Pathology & Audiology, West Virginia University

Abstract

Background—Although many speech errors can be generated at either a linguistic or motoric level of production, phonetically well-formed sound-level serial-order errors are generally assumed to result from disruption of phonologic encoding (PE) processes. An influential model of PE (Dell, 1986; Dell, Burger & Svec, 1997) predicts that speaking rate should affect the relative proportion of these serial-order sound errors (anticipations, perseverations, exchanges). These predictions have been extended to, and have special relevance for persons with aphasia (PWA) because of the increased frequency with which speech errors occur and because their localization within the functional linguistic architecture may help in diagnosis and treatment. Supporting evidence regarding the effect of speaking rate on phonological encoding has been provided by

Tepanta R. D. Fossett was a Ph.D. student in the Department of Communication Science and Disorders at the University of Pittsburgh and affiliated with the Department of Audiology and Speech Pathology in the VA Pittsburgh Healthcare System during the course of this project. She is now an assistant professor in the Department of Speech & Hearing Sciences at Indiana University. Linda I. Shuster is now a professor in the Department of Communication Sciences & Disorders at Grand Valley State University.

Authors' Addresses:

Tepanta R. D. Fossett, Department of Speech-Language-Hearing Sciences, Loyola University Maryland, 4501 N. Charles St., Baltimore, MD 21210, 410-617-5291, trfossett@loyola.edu

Malcolm R. McNeil, Dept. of Communication Science & Disorders, University of Pittsburgh, 4033 Forbes Ave., Pittsburgh, PA 15260, (412) 383-6541, mcneil@pitt.edu

Sheila R. Pratt, Dept. of Communication Science & Disorders, University of Pittsburgh, 4033 Forbes Ave., Pittsburgh, PA 15260, (412) 383-6537, spratt@pitt.edu

Connie A. Tompkins, Dept. of Communication Science & Disorders, University of Pittsburgh, 4033 Forbes Ave., Pittsburgh, PA 15260, (412) 383-6536, tompkins@pitt.edu

Linda I. Shuster, Department of Speech Pathology and Audiology, Western Michigan University, 1903 W Michigan Ave, Kalamazoo MI 49008, 269-387-8054, linda.shuster@wmich.edu

The data presented in this manuscript represent data collected for the doctoral dissertation of the first author. Although this study was completed at the University of Pittsburgh, Malcolm R McNeil and Sheila R. Pratt provided resources and facilities through the Geriatric Research Education and Clinical Center in the VA Pittsburgh Healthcare System. Nonetheless, the contents of this manuscript do not represent the views of the Department of Veterans Affairs or the United States Government.

A portion of the data presented in this manuscript also were included in an abstract submitted to the Academy of Aphasia and previously published in *Brain & Language* (2007). The authors wish to acknowledge the multiple recruitment sites involved in facilitating data collection for disordered participants, with special acknowledgement to Drs. Nadine Martin and Myrna Schwartz at Temple University and Moss Rehabilitation Hospital, respectively, and Dr. Julie Wambaugh, at the VA Salt Lake City Healthcare System. Additionally, they wish to thank Dr. Julie A. Fiez in the Department of Psychology at the University of Pittsburgh for her contributions to the project.

studies using young normal language (NL) speakers and computer simulations. Limited data exist for older NL users and no group data exist for PWA.

Aims—This study tested the phonologic encoding properties of Dell's model of speech production (Dell, 1986; Dell, et al., 1997), which predicts that increasing speaking rate affects the relative proportion of serial-order sound errors (i.e., anticipations, perseverations, and exchanges).

Methods & Procedures—The effects of speech rate on the error ratios of anticipation/exchange (AE), anticipation/perseveration (AP) and vocal reaction time (VRT) were examined in 16 normal healthy controls (NHC) and 16 PWA without concomitant motor speech disorders. The participants were recorded performing a phonologically challenging (tongue twister) speech production task at their typical and two faster speaking rates.

Outcomes & Results—A significant effect of increased rate was obtained for the AP but not the AE ratio. Significant effects of group and rate were obtained for VRT.

Conclusion—Although the significant effect of rate for the AP ratio provided evidence that changes in speaking rate did affect PE, the results failed to support the model derived predictions regarding the direction of change for error type proportions. The current findings argued for an alternative concept of the role of activation and decay in influencing types of serial-order sound errors. Rather than a slow activation decay rate (Dell, 1986), the results of the current study were more compatible with an alternative explanation of rapid activation decay or slow build-up of residual activation.

Keywords

phonologic encoding; speech production; aphasia; serial-order errors; speech rate

Speech production is not an error free process for normal healthy control (NHC) speakers or for persons with aphasia (PWA). The phonologic encoding (PE) of speech production involves the selection, retrieval, and sequencing of sounds appropriate for the intended target production. Errors in PE, including phonological paraphasias (e.g., serial-order errors, including transposition (exchange), anticipation and perseveration errors (Schwartz, Saffran, Bloch, & Dell, 1994)), frequently occur in both healthy (Boomer & Laver, 1973; Cohen, 1973; Frisch & Wright, 2002; MacKay & James, 2004) and disordered speakers (Blumstein, 1973; Schwartz, et al., 1994; Wilshire & McCarthy, 1996). At least one dominant speech production model (Dell, 1986) has identified linguistic (e.g., phonemic similarity) and motor level (e.g., speaking rate) variables that might influence accurate sound production. Dell proposed a model that describes processing at the semantic, syntactic, morphologic, and phonologic levels, but is most completely specified at the PE level. This theoretical account specifies how serial order is maintained, the relationship between specific variables (e.g., speaking rate) identified as relevant for serial-order behavior and how whole segment serial-order (anticipations, perseverations, exchanges) errors occur. The model proposes the mechanism of bi-directional spreading activation between/among processing levels, and emphasizes the excitatory characteristics of processing within this framework. Spreading activation and decay rates reflect the rate of processing among levels and based on the model, the rate of processing in the lexical network is assumed to be constant. Bi-directional spreading activation allows for feedback among processing levels. Here the focus is limited

specifically to the level of phonologic encoding and associated phonemes. Unique to this model and specifically related to the temporal nature of spreading activation, are predictions regarding phonological encoding and the manipulation of processing time. Serial-order errors may result as a consequence of the manner in which activation spreads. Serial-order is maintained as a function of turning off the activation of previously produced target units, activating the immediate target unit, and priming the future activation of targets to be produced (Martin & Dell, 2004). For example, an anticipation error is likely when there is altered activation of the current target unit and priming of the activation of a future phoneme target. A perseverative error is likely with altered activation of the current target unit and failure to extinguish the activation of a previous target unit (Martin & Dell, 2004, p. 351). Altered activation strengths, along with the rate of activation affect relative activation levels of other target and competing units, and also influence the residual activation of recently produced targets. Weak connections enhance the effect of residual activation and lead to more perseverative errors. Reduced activation strength (such as that resulting from aphasia) creates an environment that is vulnerable to disruption (Martin & Dell, 2004).

Schwartz et al. (1994) and Dell et al. (1997) proposed that speaking rate and aphasia influence the occurrence of serial-order errors and that these variables are associated with reduced connection strength, as compared to an intact, future-oriented system. According to the Dell (1986) model, the interaction of speaking rate and spreading activation can affect PE such that predicted ratio patterns of anticipations, exchanges and perseverations may occur. Dell predicted that anticipation errors would be more likely at slower speaking rates because the increased time for encoding allows the sound selected in error to be activated, experience activation decay, and then rebound to a high enough level to be re-selected in its appropriate location. At fast speaking rates an exchange error is likely because activation from an anticipated sound doesn't have time to decay and rebound and thus cannot be chosen again for its intended location. Consequently, Dell predicted that the ratio of sound-level exchange to anticipation errors should decrease at slower speaking rates. Perseveration errors occur more frequently at faster speaking rates because of the decreased amount of time between previously encoded units and current targets. Though there were methodological differences in determining error ratios, the speaking rates themselves, and whether speaking rate manipulation was within or between subjects, results of examined error patterns in young NHCs, from both Dell (1986) and Dell et al., (1997) provided some support for these predictions. These predictions contrast the perspective of McNeil and Kent (1990) who asserted that manipulating speaking rate should selectively affect speech motor processes. Alternatively, Levelt (1989) suggested that speaking rate potentially affects all aspects of PE, physical production of the message (including pause insertion), and manipulation of the actual duration of produced units. Levelt's model does not make predictions regarding speaking rate, aphasia and the occurrence of serial-order errors. It should be noted that the studies of Dell (1986) and colleagues (1997) do not address motor planning/programming processes, nor provide measures to indicate the time course of such processing (i.e. vocal reaction time), relative to articulation. Thus, changes in performance as a result of the speaking rate manipulation may have resulted from changes in the available time for PE, motor response planning, or both.

Vocal reaction time (VRT) has been used to capture the time course of stages of information processing (Schmidt & Lee, 2014; Fozo & Watson, 1998). In speech production, this includes cognitive-linguistic processing (Kello, 2003) and response preparation (Klapp, 2003; Maas & Mailend, 2012) for motor level processes. Previous investigations have yielded conflicting results regarding the effects of pre-speech initiation response time on production accuracy (Deal & Darley, 1972; Mailend & Maas, 2013; Towne & Crary, 1972) in patients with impaired PE and/or motor-speech. Towne and Crary (1972) examined verbal reaction time in three patients with post-stroke, left-hemisphere lesions (one anterior, two posterior) in a single word reading task with both a delay and no delay condition. Of the two participants who produced both correct and incorrect responses, production accuracy increased in the delay condition for the participant with the anterior lesion. Verbal reaction times for the posterior participant with both correct and incorrect responses did not differ from the control group reaction times in either delay condition. Verbal reaction times prior to the initiation of speech for the patient with the anterior lesion were longer than that of the control group. Faster verbal reaction times were obtained in the delay condition for all participants. Deal and Darley (1972) reported no effect of delay on phonemic accuracy in 12 participants identified as speakers with apraxia of speech. In a delayed picture-word interference task including healthy controls and participants with aphasia and apraxia of speech and aphasia only, Mailend and Maas (2013) reported no significant VRT differences between the participants with aphasia without motor speech disorder and their healthy control participants. However, other studies examining speech production involving controls (Dell, et al., 1997), and/or neurologically impaired participants (Schwartz, et al., 1994) have not controlled for or otherwise evaluated the effect of response time on speech production performance, including phonological encoding.

Dell's (1986) model has been theoretically and experimentally extended to aspects of semantic and phonological performance in PWA (Dell et al., 1997; Martin, Dell, Saffran, & Schwartz, 1994; Schwartz et al., 1994). Dell et al. (1997) proposed that aphasia should lower the ratio of anticipation and perseveration errors (AP proportion), (calculated as the number of anticipations/number of anticipations + perseverations). Martin and Dell (2004) and Dell et al. (1997) have suggested that the brain damage in aphasia reduces connection weights and that both faster speaking rates and brain damage in aphasia result in less activated targets. Thus, it is reasonable to hypothesize different serial-order error ratios between NHCs and PWA as well as differences depending on speaking rate. It is expected that with increasing speaking rates and aphasia, PWA should produce significantly more perseveration and exchange errors than NHCs and that all participants should produce more of these error types at faster speaking rates. It is expected that faster than normal speaking rates will differentially affect PWA because of the presence of brain injury and the demands of increased speaking rate.

There is evidence that phonological errors associated with aphasia (regardless of aphasia type) are similar to those of normal slips of the tongue (Buckingham, 1992; Schwartz et al., 1994) and include serial-order errors as well as sound substitutions (Blumstein, 1973). However, interpretation of the level of processing disruption associated with the sound-level errors in PWA has been questioned due to several factors, including the common co-occurrence of dysarthria or apraxia of speech with aphasia and the precision of perceptual

analyses (McNeil & Kent, 1990; Odell, McNeil, Rosenbek, & Hunter, 1990). The perceptual analyses in most studies have used broad phonetic transcription to describe sound-level production errors, though there is evidence to suggest that this level of precision may obscure the accurate interpretation of such errors (Odell et al., 1990). McNeil, Robin, and Schmidt (2009) argued for the use of narrow phonetic transcription for speech production errors when perceptual analysis is used so that detailed characteristics of the speech error might be captured rather than categorizing only the phonological category in which it is perceived. Without broad phonetic transcription it is not possible to determine if sound production errors are errors that can be attributed solely to the level of PE (McNeil et al., 2009; Shuster & Wambaugh, 2000). However, some researchers espouse an interaction between phonologic and phonetic processing and have questioned whether different types of sound errors can be attributed to a specific stage of phonologic – phonetic processing (Poupier, Marin, & Waiti, 2014; Laganaro, 2012). An argument against this obligatory interaction is that these error types do show double dissociations. That is, persons with PE errors but without evidence of motor impairments (dysarthria or apraxia of speech) are frequently encountered. Likewise, persons without PE errors but with motor speech disorders also do occur (McNeil, et al., 2009).

Phonological-level errors should include serial-order errors, and potentially phoneme substitutions¹ (errors of selection) that are without distortions (McNeil, et al, 2009; Van der Merwe, 2009). Speech production errors that result from disruption to motor planning and motor programming processes (i.e., most dysarthria types and AOS) should result in temporally or spatially disrupted speech (including delayed speech onset), and as supported by perceptual (Haley, Bays, & Ohde, 2001), acoustic, and physiologic investigations (McNeil & Kent, 1990), result in sound-level errors that are perceived or measured as distortions. A variety of speech errors are associated with the impairments of aphasia, apraxia, and the dysarthrias. McNeil and Kent (1990) and McNeil, et al. (1997) state that speech errors have traditionally been used for classification within these neurological impairments. McNeil et al. proposed that some speech errors can be confidently attributed to either the linguistic or motor levels, however, many error types can be generated at either level. For example a speech error characterized as a sound substitution might result from an incorrectly selected phoneme, a motor planning error where degrees of co-articulation are assigned, or from a timing error during articulatory execution. From the perspective of McNeil and Kent (1990), only errors that are the result of an incorrectly selected phoneme, or an error in slot construction or assignment may be assigned to a linguistic level. These researchers suggest that sound sequencing errors can be attributed to only the linguistic level and distortion errors only to the motor level. Currently accepted criteria for differential diagnosis of apraxia of speech from phonologic paraphasias (Wambaugh, Duffy, McNeil, Robin, & Rogers, 2006) include reference to the type of produced sound error (distorted vs. phonologic paraphasia). Thus, this perspective suggests little confidence can be placed in

¹Distortions can also be perceived of as sound-level substitutions and therefore substitutions cannot unambiguously be attributed to the PE level of production (McNeil et al., 2009). These authors proposed that distorted sound substitutions or distorted sound sequencing errors occur when both phonological and motor level processing are disturbed. The differential diagnostic value of undistorted sound sequencing errors is that they, as compared with other error types, can more confidently be attributed to disruption of the PE (linguistic-symbolic) level of production (McNeil et al., 2009). The models of Dell (1986, 1988) and Dell and colleagues (Dell et al., 1997) are consistent with this level of error assignment.

level assignment of any errors other than non-distorted serial-order and distortion errors until more definitive knowledge about the linguistic-motoric interaction is known and can be incorporated into the descriptions and explanations of various disorders.

Dell (1986) and others (Acheson & MacDonald, 2009) have identified phonologic similarity as one variable that might affect accurate sound production, as phonologically similar items, have fewer distinguishing features (Roelofs, 1999) and a higher percentage of errors for response accuracy when place and manner are shared (Rogers & Storkel, 1998). Roelofs (1997) posited that featural similarity effects occur because features are part of the phonetic/mental syllabary (a store that is important for translation of phonologic to phonetic information) and target phonemes and the substituting erred phoneme typically share features. Thus, there is both experimental and theoretical support that manipulation of phonologic similarity may influence PE. Indeed, PE errors occur spontaneously in conversational speech but erred productions may be enhanced in experimental tasks by manipulating phoneme similarity (Wilshire, 1999). PE error rates ranging from 4% - 13% have been obtained in tasks manipulating phoneme similarity and repetition pattern in NHC middle-aged adult speakers, with higher percentages in tasks that also require multiple stimulus repetitions (Wilshire, 1999). Wilshire (1999), reported a high proportion of anticipation errors (about 78%), but few perseveration errors (about 22%) in these NHC speakers. Because many of the results paralleled those found with other elicitation methods (Baars, Motley, & MacKay, 1975; Dell, 1986), Wilshire argued for the validity of using the tongue twister paradigm for investigations of PE.

The current study examined the effects of speech rate on the ratio of phonological sequencing errors - *anticipation/exchange* (AE) and *anticipation/perseveration* (AP) errors, in NHCs and in PWA who were without concomitant motor speech disorders. It is assumed that errors produced without distortion represent errors of PE. Each error ratio was calculated with the anticipation error count in the numerator and the exchange and perseveration error counts in the denominator for the AE (anticipations/exchanges) and AP (anticipations/perseverations) error ratios, respectively. Significant differences and interactions for speaking rate (normal, fast, faster) and participant group (NHC, PWA) were examined. It was predicted that there would be a significant decrease in the ratio of AP (anticipation/perseveration) errors with increased speaking rates for both participant groups. A significant interaction was predicted between speaking rate and group, with PWA producing significantly more perseveration errors than NHC at the faster than typical speaking rates, due to the rate manipulation as well as brain-damaged. Similarly, it was predicted that AE (anticipation/exchange) errors would decrease with increasing speaking rate for both groups and there would be a significant interaction between speaking rate and group with a significantly lower AE ratio at the faster rate for the PWA than NHC. Finally, it was predicted that there would be no difference in vocal reaction time across speaking rate for either group, as well as no significant interactions. The predictions of the model have special relevance for persons with aphasia (PWA) because of the increased frequency with which speech errors occur in this population and because understanding the mechanism may influence diagnosis and intervention.

Methods

Participants

Demographic Information—Thirty-two adult (16 NHC and 16 PWA) native speakers of English volunteered as participants. The NHC and PWA ranged in age from 41–75, ($M = 60$; $SD = 10.2$) and 36–77 ($M = 56$, $SD = 11.08$), respectively. The mean years of education were 16.38 and 15.19 for the NHCs and the PWA, respectively. Table 1 presents demographic information for all participants and the lesion data for the PWA when it was available from medical records.

Screening Measures—The NHCs were without a history or evidence of speech, language, cognitive, or neurologic deficits as indicated by self-report and measured by a large battery of standardized speech, language and cognitive tests administered by a licensed and professionally certified speech-language pathologist as reported below. The PWA met the criteria for the definition of aphasia (inefficiency with verbal symbolic processing) as proposed by McNeil and Pratt (2001) and were selected based on their performance on the Revised Token Test (RTT; McNeil & Prescott, 1978) and the shortened version of the Porch Index of Communicative Abilities (SPICA; DiSimoni, Keith, & Darley, 1980), a multi-modality measure of language processing. This measure included the following verbal subtests: naming, imitative naming, object functions, and sentence completion. Additionally, prior to the experimental task, all participants were administered a sentence repetition task consisting of eight non-phonologically challenging sentences. Participants were required to repeat at least six of the eight sentences without fillers (e.g., ‘uh,’ or ‘something’) and with the same number of syllables as the target sentence to be included in the study.

All participants passed a pure tone audiometric screening at 30 dB HL in at least one ear and demonstrated word recognition scores above 67% on the Picture Identification Task (PIT; Wilson & Antablin, 1980). They performed within the range of NHCs on nonverbal reasoning skills (above the 5th percentile) as measured by the Coloured Progressive Matrices (RCPM; Raven, 1963; 1988) and verbal intermediate-term memory as measured by the Story-Retelling (immediate/delayed) subtest from the Arizona Battery for Communication Disorders in Dementia (ABCD; Bayles & Tomoeda, 1993). The NHCs performed above the cutoff score of the 1st percentile for auditory language processing as measured by the 55-item version (Arvedson, McNeil & West, 1986) of the Revised Token Test (RTT) (McNeil & Prescott, 1978) and ranged from the 2nd to 100th percentile for NHC participants ($M = 48.44$, $SD = 28.89$). The PWA ranged from the 2nd to 91st percentile ($M = 74.50$, $SD = 25.75$) for left-hemisphere brain-damaged PWA. SPICA percentiles (estimated using the full 180-item PICA (Porch, 1981) normative data for PWA) ranged from 57–96 ($M = 80.81$, $SD = 11.11$) for the PWA. The PWA were administered some subtests from the short version of the Boston Diagnostic Aphasia Examination (BDAE; Goodglass, Kaplan, & Barresi, 2001) summarized in Table 2. The shortened version of the Boston Naming Test (BNT; Kaplan, Goodglass & Weintraub, 2001), was administered and yielded a mean percentage score of 81.5 ($SD = 16.26$) with a range of 40–100. Based on these measures, the language production abilities of the PWA ranged from mild to moderate severity. Screening and descriptive data for all participants are summarized in Tables 1 and 2.

Subtests from the Apraxia Battery for Adults-2 (ABA) (Dabul, 2000) and the Dysarthria Examination Battery (DEB; Drummond, 1993) were administered to all participants to elicit behaviors from which the presence of apraxia of speech and dysarthria were judged. Twelve participants with dysarthria or AOS were excluded. The presence of dysarthria was independently perceptually judged by the first and second authors, both licensed and certified speech-language pathologists experienced in the diagnosis of motor speech disorders. These judgments considered speech and voice characteristics that contribute to the diagnosis of a motor speech disorder (Darley, Aronson & Brown, 1969a; 1969b). Criteria for determining the presence/absence of AOS were also based on characteristics defined by McNeil et al. (2009), and included the following: prolonged vowel and consonant durations in multisyllabic words, increased inter-segment durations, and the presence of sound-level distortions, (including distorted serial-order phoneme errors) and distorted sound substitutions. Measures of phonologic processing and working memory span are summarized in Supplemental Content Table 2. As expected, the PWA performed significantly more poorly than the NHC on all of these measures as tested with ANOVA and nonparametric procedures.

Stimuli

The stimuli consisted of 36 phonologically challenging “tongue twister” sentences (12 experimental and 24 fillers) previously used by Fossett et al. (2000) and adapted from previously published stimuli and children's literature (Supplemental Content).

Experimental and filler stimuli were seven or eight syllables in length. Sentence production difficulty for experimental stimuli was increased beyond that of the filler sentences by the presence of similar and repeating phonemes. Filler stimuli were included to discourage early responses (i.e., responding before the signal to produce the experimental sentence). The stimuli were produced by a male speaker seated in a sound attenuated booth and recorded at a rate of approximately 3 syllables per second, with a head mounted condenser microphone (Crown CM 312) and digitized on a Macintosh desktop computer at a rate of 22 kHz. Using sound editing software (Superscope), the productions were equalized across sentences for intensity and duration (range 3-4s). After editing, an independent listener transcribed the sentences orthographically and if any word within a sentence was not identified correctly the sentences was re-edited until another independent listener was able to identify all words within all sentences correctly. The stimuli were presented electronically from a Dell laptop computer, using the E-Prime 2.0 software (Psychology Software Tools, Pittsburgh, PA).

Experimental Procedures

Each participant was evaluated individually in a quiet room (laboratory, hospital or nursing care facility, or participant's home) after providing informed consent in compliance with primary and site-specific Institutional Review Board requirements. All of the screening and experimental procedures were completed within two weeks and within 3 sessions for all but two participants with PWA (4 and 5 sessions). Most PWA and NHCs completed the protocol in about 3½ hrs.

Participants repeated each experimental sentence one time in each of three speaking rate conditions: typical, fast and faster. Rate conditions were defined relative to each participant's own speaking rate. All experimental and filler sentences were presented in each speaking rate condition. Both the sentences and task instructions were presented from the computer with signals routed through a portable audiometer (Maico MA 25) to insert earphones (EarTone 3A). The presentation level was 55 dB HL unless a participant requested a higher level. A 100 ms, 1000 Hz tone followed all stimuli that were to be produced by the participants (experimental sentences only) and the participants were asked to begin their responses as quickly as possible after hearing the tone. A 6-inch horizontal line intersected by three vertical lines was displayed on the computer screen and the examiner pointed to a vertical line to indicate the target speaking-rate for each condition. The examiner also controlled the inter-stimulus presentation rate to allow for anticipated variability in production time among participants. One channel of the portable audiometer was monitored to ensure that the stimuli were presented as intended and that participants were responding to target stimuli and not fillers. Prior to starting the experiment, participants were given 32 practice sentences.

The sentences were presented randomly within rate condition, and condition order was counterbalanced across participants. A 2-min break was provided after every 36 trials and before instructions to produce the stimuli at the next rate (See online Supplemental Content for instructions). Rate compliance was encouraged as necessary, with both spoken (e.g., “go a little faster”) and non-spoken cues (i.e., pointing to the appropriate mark on the visual scale).

Participants' productions and all of the experimental and filler stimuli were recorded with a head mounted condenser microphone (Crown CM 312) positioned 1 in from the corner of the mouth and connected to a Tascam Porta 02MKII audio recorder. Participants also wore an omnidirectional lavalier microphone (Audio-Technic ATR335) that was connected to the computer via a response box that activated a trigger when a production was initiated.

Response Recording and Analysis

Productions were digitized with the Computerized Speech Laboratory (CSL; Kay Elemetrics) at a sampling rate of 25 kHz. Recordings included the tone cue through the end of the participant's production (plus 1 second post-termination of production when possible) with an allowable maximum sample length of 12 sec. Stimuli were digitized to facilitate analysis should any portion of the participant's production be unintelligible.

Data Organization—Because the questions addressed in this study were based on assumptions about the time-course, mechanisms of spreading activation and the phonological error proportions related to changes in speaking rate, the responses collected from the fast and faster conditions were re-categorized to reflect actual produced speaking rate for each response relative to the participant's typical speaking rate productions. All tokens were categorized as “typical” that were produced up to the $M + 1 SD$ of the “typical” speaking rate, regardless of the conditions at which the token was originally elicited. That is, productions that were elicited at the “faster” condition but that fell within the $M + 1SD$ of

the individual participant's typical rate productions were re-categorized and analyzed as “typical” rate productions. Responses produced faster than the $M + 1SD$ of the “typical” rate and up to the $M + 1SD$ of those originally categorized “fast” were categorized as “fast”. Any responses that exceeded the $M + 1SD$ of the original fast rate were categorized or re-categorized as “faster”.

Transcription and Transcription Reliability—All utterances were transcribed by the first author with broad phonetic transcription unless perceived to be distorted, in which case they were narrowly transcribed using the phonetic transcription conventions of Shriberg and Kent (1995). Inter- and intra-rater reliabilities were obtained on 16% of the target stimuli (2 tokens from each rate condition) for each participant (transcripts were based on fewer tokens for some participants due to insufficient erred productions). Transcription procedures are detailed in the Supplemental Content. Inter-rater reliability results for broad phonetic transcription yielded point-to-point agreement of 94% and 88% for the NHC and PWA groups, respectively. Agreement regarding the presence of a distortion and thus a need for narrow phonetic transcription was 55%. Identification of the same distorted phoneme was high for both the NHC (100%) and PWA (95%) groups. Similar inter-rater agreements were achieved for identification of the specific distortion (i.e., same diacritic symbol) with 100% and 91% for NHC and PWA participants, respectively.

Error Coding and Error Coding Reliability—Subsequent to transcription, error type was coded. Categories included non-distorted and distorted serial-order errors (i.e., anticipation, perseveration, and exchange errors). Other sound error types were coded for descriptive purposes (See Supplemental Content for definitions and scoring rules for all error types). The data contributing to the serial-order error ratios in this study are limited to those determined to be produced without distortion (but see Buchwald & Miozzo, 2011 for a contrasting perspective). Inter-rater reliability was obtained on 25% of erred stimuli at each speaking rate for four participants from each group. The percentage of agreement at each speaking rate for NHC participants regardless of error type was 100% and for PWA it ranged from 53% - 79% with an average of 70%. Inter-rater agreement was 53% for serial-order errors for PWA at the faster speaking rate and 79% and 75% for typical and fast rates respectively.

Phonological Error Opportunities—To determine the proportion of phonological error types produced, it was necessary to first determine the number of opportunities for each error type to occur within a target sentence. Thus, each sound from each phonetically transcribed target sentence was judged as to whether or not that sound could replace or exchange positions with any other sound in the sentence and not violate the phonotactic rules of American English (e.g., a schwa could not replace a vowel in a stressed syllable, because the schwa sound only occurs in unstressed syllables). The error opportunities (potential errors) in each of the 12 target stimuli for each phonological error type and the rules used to determine phonological error opportunities are detailed in Table 3 of the Online Supplemental Content.

Acoustic Measurement—For a response to be eligible for acoustic analysis it had to contain the same number of syllables as the target, be without false starts, speech interruptions (i.e., “um”), non-speech interruptions (i.e., coughs), or place holder words (i.e., “something” or “ahhh”). Productions containing non-words, phonological errors and distortions were included as long as they met other inclusion criteria. Vocal reaction time (VRT) and speaking rate based on total duration were measured from waveform and wide-band spectrogram (286 Hz bandwidth) displays using the CSL acoustic analysis software. Information from both the waveform and the spectrogram was used to guide measurement decisions. The VRT was defined as the time from the offset of the tone to the initiation of sound production. VRT measurements included erred productions as they were intended to reflect the time course within which compromised processing occurred. Total duration was defined as the time from the initiation to termination of production of the experimental sentence and speaking rate was based on syllables per s. Inter- and intra-judge reliability from two judges was obtained on 17% and 44% of the target stimuli for NHC and PWA respectively, using Pearson Product Moment or Spearman Rho correlation coefficients. For NHC, inter- and intra-judge reliability for VRT ranged from .97 – 1.0 ($M = .99$; $M = .99$) respectively. For PWA, both inter- and intra-judge reliability for VRT was 1.0. Speaking rate decisions were based on the total duration of the produced sentence. Inter-judge reliability for speaking rate for NHC was 1.0 and intra-judge reliability ranged from .98 – 1.0 ($M = .99$). Inter-judge speaking rate reliability ranged from .98 – 1.0 ($M = .99$) and intra-judge reliability was 1.0 for PWA.

Results

Analyses

Missing Data—Sixty-nine percent (399/576) of the productions from NHCs and 27% (157/576) of the productions from PWA were eligible for acoustic and perceptual analyses. The total number of re-categorized sentences for the typical, fast, and faster speaking rates was 139, 185, and 75 for the NHC participants and 66, 62, and 29 for the PWA respectively. See the Supplemental Content (Table 4) for eligibility criteria and total number of re-categorized sentences for individual NHC and PWA at each speaking rate.

Data Description

The mean percentage of errors (number/opportunity) either remained the same or increased with increasing speaking rate for all error types for NHC, except for a decrease in the mean percentage of perseveration errors from the ‘fast’ to ‘faster’ speaking rate conditions. For PWA, the mean percentages of errors varied with increases in anticipation errors, decreases in perseveration errors with increasing speaking rate, and decreased exchange errors from the typical to fast speaking rates, but an increase from the fast to faster speaking rates. Figures 1 and 2 present the pattern of performance on each of the error types (number/opportunity). Although the experimental questions in this study are specific to the serial-order errors produced without distortion, the error summary for all categories, at each speaking rate, along with the raw number of each serial order error type produced for individuals in both groups are presented in the online Supplemental Content (Tables 5 – 10).

Statistical Analyses

Arcsine transformations were applied to normalize error-data distributions for sound error types. An alpha level of .05 was used for all comparisons. ANOVA results for the AP ratio revealed a significant main effect for rate, $F(2,46) = 4.773$, $p = .01$, $\eta^2 = .17$, and a non-significant main effect for group, $F(1,23) = .512$, $p = .48$, $\eta^2 = .02$, (power = .11), and no significant interaction, $F(2,46) = 1.477$, $p = .24$, $\eta^2 = .06$, (power = .30). Post hoc analyses with a Bonferroni adjustment for multiple comparisons revealed a significant ($p = .02$) increase in the AP ratio between the typical and faster rate. There were no significant differences between typical and fast or fast and faster rates, for either group. The NHC average transformed AP error ratios ranged from .674 – 1.373 ($M = .910$, $SD = .173$) for the typical speaking rate, and .739 – 1.585 ($M = 1.013$, $SD = .210$), and .837 – 2.288 ($M = 1.355$, $SD = .437$) for the fast and faster speaking rates respectively. The PWA average transformed AP error ratios ranged from .612 – 2.083 ($M = .995$, $SD = .392$), .833 – 2.083 ($M = 1.251$, $SD = .373$), and .625 – 2.430 ($M = 1.222$, $SD = .604$), for the typical, fast, and faster speaking rates, respectively. Figure 3 illustrates the performance of both groups on the transformed AP error ratio. The general pattern of performance for each group is consistent with that for the AE ratio (Figure 4).

Results from a two-way repeated measures ANOVA for the AE error ratio revealed no significant main effect for rate, $F(2,46) = 1.727$, $p = .19$, $\eta^2 = .07$, ; or group, $F(1,23) = .907$, $p = .35$, $\eta^2 = .04$ (power = .10). The NHC average transformed AE error ratios ranged from .643 – 1.322 ($M = .910$, $SD = .183$) for the typical speaking rate, and .692 – 1.414 ($M = 1.043$, $SD = .198$), and .692 – 1.860 ($M = 1.127$, $SD = .379$) for the fast and faster speaking rates respectively. The PWA average transformed AE error ratios ranged from .285 – 1.679 ($M = .995$, $SD = .403$), .794 – 1.890 ($M = 1.203$, $SD = .345$), and .618 – 2.553 ($M = 1.116$, $SD = .592$), for the typical, fast, and faster speaking rates, respectively. Figure 4 illustrates performance across speaking rates with the arcsine-transformed data. The observed power for the AE error ratio calculation for the main effect of rate was .34 with a η^2 of .07. Given these power and effect size calculations, a post-hoc power analysis estimated that 56 participants would be necessary in each group to attain a power of .80.

To provide evidence that the participants met the experimental task demand to initiate speech immediately after the signal cue in order to limit PE preparation/processing time, a secondary analysis explored VRT data between groups and across rates. Normality assumptions were met and a two-way repeated measures ANOVA revealed a significant main effect of rate, $F(2,44) = 13.81$, $p = .00$, $\eta^2 = .39$, and group, $F(1,22) = 10.16$, $p = .00$, $\eta^2 = .32$, and no significant interaction, $F(2,44) = .99$, $p = .38$, $\eta^2 = .04$ (power = .21). Overall, longer VRTs were produced at the typical speaking rate ($M = .604$ ms) compared with the fast (.504 ms); which were significantly longer compared to the faster (.447 ms) rates. The significant group difference resulted from an overall longer VRT for the PWA ($M = .606$ ms) than the NHC participants ($M = .432$ ms) at each rate.

Discussion

Serial-order speech error predictions for both the NHC and PWA were based on the assumptions and predictions of the interactive spreading activation models of Dell (1986)

and Dell et al. (1997). The primary assertion is that speaking rate manipulation can affect PE as evidenced by a change in the ratio of phonologic errors in those studies. Current study results also provided evidence of change in the ratio of serial-order errors with changes in speaking rate. Results revealed a significant increase in the AP ratio (defined as anticipations/perseverations) from the typical to faster speaking rate condition, though this finding was in the opposite direction of that predicted by Dell et al., (1997). The results revealed an increase in the number of anticipatory relative to perseveration errors with increased speaking rate. These results replicated the higher anticipation to perseveration ratio for NHCs (Garnham, Shillcock, Brown, Mill, & Cutler, 1981) and PWA (Blumstein, 1973; Wilshire & McCarthy, 1996). In contrast, though speaking rate effects were not investigated, Schwartz et al., (1994) and Kohn and Smith (1990), each reported more perseverations than anticipations in a single person with aphasia. Contrary to our prediction, no significant group differences were obtained in the current study. This finding is in concert with those of Wilshire and McCarthy (1996) in which a single person with aphasia and impaired PE produced a ratio of anticipations to perseverations within the range of the NHCs. Both theory (Buckingham, 1980; Dell, Schwartz, Martin, Saffran, & Gagnon, 1997) and experimental evidence across language domains (Dell, Schwartz, et al., 1997; Martin, Dell, Saffran, & Schwartz, 1994; Wilshire & McCarthy) suggest that this finding is not untenable, as it has been proposed that aphasia and normal language processing exist on a continuum (aka, the continuity hypothesis) (but see Rumel, Caramazza, Capasso, & Miceli, 2005 for an alternative perspective). Both participant groups in this study produced errors of PE, and the pattern of error ratios remained the same between groups. The lack of differences in the error ratio patterns suggests that the model's postulated assumptions about the effect of brain damage in aphasia on the mechanism of spreading activation, is incorrect or that the effect of speaking rate on the behavior of spreading activation was insufficient to reveal the effects. Additionally, other variables that may have contributed to the lack of group differences include aphasia severity level and the restricted number of analyzable errors resulting from response exclusion criteria.

Dell et al. (1997) and Martin and Dell (2004) hypothesized that anticipation errors result from the co-occurrence of disrupted activation to the current target and to an upcoming unit in the plan. Anticipation errors occur, when an upcoming phoneme is more highly activated than the current target. One hypothesis regarding the increase in anticipation rather than perseveration errors with increased speaking rate in this study is that the construction of the phonologically challenging sentences promoted an imbalance between the turn-on and prime functions described in Dell et al. (1997). That is, elements in the 'future' receive activation from the plan, but are also primed with anticipatory activation. The phoneme similarity and manipulation of repeated phonemes that make the current study stimuli phonologically challenging may further prime future elements, and contribute to higher activation levels than that of the intended target. However, Dell et al. (1997) obtained different results using phonologically challenging phrases, but with young healthy controls. It also is possible that the increase in speaking rate was not sufficient in magnitude to highlight the effects of a slow decay rate. According to the model, slow decay would promote exchanges and perseveration errors over anticipation errors as the activation of the selected unit remains high enough to be reselected. If achieved speaking rates were insufficient in the current

study, the activation level of the selected unit may have had time to decay but not sufficiently rebound, reducing chances that the unit would be re-selected. This would result in the production of more anticipation errors.

The rate of activation decay during processing has been identified as a variable that may contribute to serial-order errors. For example, Stemmer (1989) proposed that children have a slower rate of activation decay than adults, based on the higher rates of perseveration errors. This suggestion counters Dell and colleagues (1997), who hypothesized that the AP findings in children are due to decreased connection strength. An alternative to the proposal of slow decay, however, is the possibility of fast decay. If the rate of activation decay is generally rapid, the tendency for increased perseveration or exchange errors might not be observable unless speaking rates were exceedingly rapid. The alternative possibility of fast decay has been suggested by others relative to naming errors in PWA (Martin et al., 1994) and is acknowledged by Dell and colleagues (1997) relative to the potential effects of aphasia on AP. Thus, although Dell (1986) proposed that slow rates of activation decay would allow for higher numbers of perseveration errors, fast activation decay and/or slow re-bounce of activation following activation decay are plausible alternatives that would accommodate the current study results. However, the alternative explanation of fast decay, as presented here, is not specific to the effects of aphasia, as in the current study no group differences were obtained relative to the serial-order and speaking rate questions. It is unclear whether severity of aphasia contributed to the findings in this study. In a study examining perseverative behavior in both healthy and impaired persons, Helm-Estabrook, Bayles, and Bryant (1994) concluded that perseverative behavior was related to impairment severity. Based on assessment measures, PWA in this study presented with a mild-moderately impaired profile. It is an empirical question as to whether a more impaired group of PWA might have resulted in different findings.

Using a spreading activation model similar to that of Dell (1986), Martin et al. (1994) suggested that decreased connection strength explained production differences between a single person with aphasia and simulations of the production performance of NHC participants. They further indicated that relative to aphasia, brain damage usually is associated with weak connections. Martin and Dell (2004) suggested that reduced connection strengths result in lower activation levels of target items and thus residual activation of previously selected items may more easily achieve high enough levels to allow for re-selection. However, decreased connection strength would not explain the current findings as no group differences were obtained on the serial-order speaking rate questions and there is no reason to posit low connection strength in the NHCs.

Other methodologic factors also may account for the discrepancies between predicted and achieved results. Though error coding reliability for serial-order errors was 100% regardless of speaking rate for NHCs and was acceptable for the typical and fast speaking rates for PWA, it was lower at the faster speaking rate for PWA. This may reflect the challenge of interpreting error type in the presence of other non-serial order errors (i.e., sound deletion), which may frequently occur at increased speaking rates in PWA. Low error coding reliability at the faster speaking rate for PWA may have obscured findings of a change in the error ratios at this speaking rate. Furthermore, inclusion/exclusion criteria utterances limited

the number of analyzable utterances, thereby affecting the overall error rate. As asserted by Schwartz, et al. (1994), perseveration errors are associated with higher overall error rates. Thus the lower overall error rates in this study may have contributed to the increased AP ratio at faster speaking rates. For the current study error rates, defined as the produced number of non-distorted serial-order errors/serial-order error opportunities, ranged from .2 – 1.2% for NHCs and from .1 – 1.3% for PWA, with the lowest error rate at the faster speaking rate. Additionally, the fewest number of analyzable tokens occurred at the fast and faster speaking rates for the PWA. For two reported experiments, Wilshire (1999) reported an overall error percentage of 4.5% and 7.8% of words with sound errors. In Wilshire (1999), potentially relevant methodological issues included broad rather than narrow phonetic transcription, allowing for the inclusion of distorted serial-order errors, and no obvious exclusion of sound substitution errors. Serial-order errors perceived to be distorted were excluded from analyses in the current study and may have contributed to the low overall error rate. It is not clear how the overall error percentages from the current study and Wilshire (1999) would compare had the same inclusion/exclusion criteria been applied. Pouplier and Goldstein (2010) have provided findings they interpret as challenging the utility of perceptual identification and error categorization. Furthermore, most previous AP error ratio findings were obtained from young to middle aged NHC speakers in varied production tasks. Few studies have examined the AP error ratio in the elderly (but see Vousden & Maylor; 2006) or in groups of PWA. Notably, Vousden and Maylor did not obtain an AP effect for their older participants because they had difficulty manipulating their speaking rate.

Analysis of the AE serial-order error ratio revealed no significant main effect or interactions. As the outcome of this analysis was dependent on the behavior of two types of errors, an examination of the raw data for both groups is essential in order to interpret the results. The data revealed that few exchange errors were produced in total across all speaking rate conditions. Across all three speaking rates, there were 7 exchange errors for PWA and 23 for the NHC group. Exchanges represented 9%, 6%, and 6% of the serial-order errors at the typical, fast and faster speaking rates for PWA and 16%, 9%, and 15% of the data for NHCs at those rates, respectively. Thus, the frequency of occurrence of exchange errors was low even under presumed error-producing conditions (phonologically challenging phrases, increased speaking rate and aphasia). Previous studies have similarly reported the infrequency of occurrence of exchange errors (i.e., Dell, 1986; Vousden, Brown, & Harley, 2000). Results from the current study, however, do not provide support or counter evidence for the AE ratio prediction because of the statistical low power.

The effect of speaking rate on the presence of serial-order exchange errors has not been adequately tested. From a theoretical perspective, based on the sheer paucity of relevant errors, this lack of evidence may raise questions about the influence of temporal processing and the behavior of spreading activation relative to the frequency and conditions under which exchange errors occur. According to Dell (1986), exchange errors are not independent of the occurrence of anticipation or perseveration errors. He asserted that the production of an exchange error is related to the integrity of all processing functions and is affected by the degree of anticipatory or perseveratory bias during processing. Although the lack of statistical power in this study limits interpretation, conceptually, reliably identified non-

distorted exchange errors continue to be differentially diagnostic when they occur as they are unambiguously attributable to the level of PE, though their rare occurrence does diminish their clinical value. We are unaware of any speech production model that posits appropriate retrieval of a phoneme sized unit, which then activates and sequentially mis-orders an erroneous packet of gestures (or more than one in the case of an exchange error), which faithfully represent another phoneme sized unit. Given the limited frequency of occurrence of serial-order sound exchange errors in two human experimental studies, it may be that they are less interesting than other serial-order error types, but still theoretically important because when they do occur more than in NHCs, they are markers of the compromised processing.

This study also examined the effect of speaking rate on VRT. The primary purpose of this measurement was to provide evidence that participants had responded immediately after the tone cue and no significant effects of rate or group were predicted. The results, however, revealed significant effects of speaking rate and group, with means for the PWA group longer than those of NHC participants at all speaking rates. Although other studies have investigated the effects of speaking rate on PE (Dell, 1986; Dell et al., 1997; Vousden & Maylor, 2006; Wilshire & McCarthy, 1996), none have provided evidence that participants responded immediately after the cue to speak. Failure to initiate speech immediately after stimulus presentation could allow additional processing time for PE and might not validly reflect only the effect of the speaking rate manipulation. The decrease in VRT with the demand of increasing speaking rate is thought to reflect a decrease in preparation time with a concomitant reduced response time as a result of the time pressure to respond. This finding continues to allow for the possibility that speaking rate manipulation affects both PE and pre-articulatory processing.

As PWA were screened for motor-level deficits, the finding of a group main effect for VRT was not predicted. Data from Towne and Crary (1988) suggested that participants with more posterior lesions produced verbal reaction times similar to NHC participants. Insufficient lesion data (9/16 PWA) was obtained in this study to interpret the contribution of lesion site to these findings. Longer reaction times in PWA relative to NHCs have been reported on language tasks (Bose, van Lieshout, & Square, 2007; Dunn, Russell, & Drummond, 1989), regardless of modality. Additionally, McNeil and Kent (1990) revealed that participants described as having conduction aphasia with no clinically identifiable concomitant apraxia of speech or dysarthria, did demonstrate some deficits in motor control. The most parsimonious explanation for this finding is that they had concomitant, but subclinical, motor speech involvement. Thus, despite the fact that the PWA in this study did not demonstrate motor speech deficits on the screening measures, unambiguous assignment of the increased level of response planning to either PE or motor speech processing is not addressed by these data.

Summary

Although the results for one of two serial order ratios were significantly affected by speech rate, the finding was in the opposite direction (a rise in AP ratio) of that predicted by the models of Dell (1986) and colleagues (1997). Additionally, no significant main effect of

group and no significant interactions were obtained. The data in this study do not provide conclusive evidence to differentiate the effects of speaking rate manipulation on phonologic encoding to phonetic encoding processes.

The results of the current study may have implications for the speaking rate assumptions of Dell (1986) and Dell et al. (1997) as the AP error ratio results challenged the assumption regarding the effect of speaking rate on the proportions of some serial-order errors. The effect of speaking rate on the AE error ratios neither challenged nor provided support for the assumptions of the models, as non-significant results were obtained. The current findings argue for an alternative concept of the role of activation and decay in influencing types of serial-order sound errors. Rather than a slow activation decay rate (Dell, 1986), the results of the current study are more compatible with an alternative explanation of rapid activation decay or slow build-up of residual activation. The finding of a speaking rate effect as reflected in a change in the proportion of one serial-order phoneme error ratio provided evidence that speaking rate change or the available encoding time can affect the proportion of serial-order phoneme errors produced. Whereas the speaking rate manipulation may have affected more than serial-order phoneme errors, the error patterns represented are unlikely to represent errors generated at other than the level of PE. Some caution is suggested for the generalization of these results to non-experimental conditions as the data were obtained with experimentally manipulated stimuli designed to elicit speech production errors through the manipulation of repeated and similar phonemes, a condition not typically encountered in everyday speech production.

The results of this study, with a main finding different than that predicted, generate further questions regarding the effect of speaking rate on phonological serial-order error ratios. To disambiguate the effects of speaking rate manipulation of PE vs. phonetic encoding processes, a future study should replicate the current study and further investigate speaking rate effects on PE in participants with a motor-speech deficit but no cognitive-linguistic deficits. Given the low reliability for some measures reported in this study, continued exploration of the utility of narrow phonetic transcription in distinguishing among errors is warranted. Future investigations should also maximize error opportunity (i.e., controlling for error rate) given the large amount of data which needed to be excluded in this study. Furthermore, it is suggested that as strategies for rate manipulation may differ among individuals (Tsao, Weismer, & Iqbal, 2006), additional measures of rate change such as articulation rate and pause time should be addressed. These additional measures may provide additional information regarding response time planning not captured by the methods used in this study.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by a National Institutes of Health (NIH) F31 Pre-doctoral Fellowship award under Grant (1 F31 DC005940-01) and by the Provost's Development and Faculty of Arts and Sciences (FAS) African American Summer Graduate Research Awards from the University of Pittsburgh.

References

- Acheson DJ, MacDonald MC. Twisting tongues and memories: Explorations of the relationship between language production and verbal working memory. *Journal of Memory and Language*. 2009; 60:329–350. [PubMed: 21165150]
- Arvedson JC, McNeil MR, West TL. Prediction of Revised Token Test overall, subtest, and linguistic unit scores by two shortened versions. *Clinical Aphasiology*. 1986; 16:57–63.
- Baars BJ, Motley MT, MacKay DG. Output editing for lexical status in artificially elicited slips of the tongue. *Journal of Verbal Learning and Verbal Behavior*. 1975; 14:382–391.
- Bayles, KA.; Tomoeda, MS. *Arizona Battery for Communication Disorders of Dementia*. Canyonlands Publishing; Tucson, AZ: 1993.
- Blumstein, SE. *A phonological investigation of aphasic speech*. Mouton; Paris: 1973.
- Boomer, DS.; Laver, JDM. Slips of the tongue.. In: Fromkin, V., editor. *Speech errors as linguistic evidence*. Mouton & Co. N.V., Publishers; Paris: 1973. p. 120-131.(Reprinted from the *British Journal of Disorders of Communication* 3(1), 1968)
- Bose A, van Lieshout P, Square PA. Word frequency and bigram frequency effects on linguistic processing and speech motor performance in individuals with aphasia and normal speakers. *Journal of Neurolinguistics*. 2007; 20:65–88.
- Buchwald A, Miozzo M. Phonological and motor errors in individuals with acquired sound production impairment. *Journal of Speech, Language, and Hearing Research*. 2012; 55:S1573–1586.
- Buckingham, HW. Phonological production deficits in conduction aphasia.. In: Kohn, SE., editor. *Conduction aphasia*. Lawrence Erlbaum Associates; Hillsdale, NJ: 1992. p. 77-116.
- Buckingham HW. On correlating aphasic errors with slips-of-the-tongue. *Applied Psycholinguistics*. 1980; 1:199–120.
- Cohen, A. Errors of speech and their implication for understanding the strategy of language users.. In: Fromkin, V., editor. *Speech errors as linguistic evidence*. Mouton & Co. N.V., Publishers; Paris: 1973. p. 88-92.(Reprinted from *Zeitschrift fur Phonetik* 21 (1/2): 177-81)
- CSL Computerized Speech Lab Model 4300B Software Version 5.X. Kay Elemetrics Corp.; Lincoln Park, NJ: 1994.
- Dabul, B. *Apraxia Battery for Adults - 2*. C.C. Publications, Inc; Tigard, Oregon: 2000.
- Darley FL, Aronson AE, Brown JR. Clusters of deviant speech dimensions in the dysarthrias. *Journal of Speech and Hearing Research*. 1969a; 12:462–496. [PubMed: 5811846]
- Darley FL, Aronson AE, Brown JR. Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research*. 1969b; 12:246–269. [PubMed: 5808852]
- Deal JL, Darley FL. The influence of linguistic and situational variables on phonemic accuracy in apraxia of speech. *Journal of Speech-Language-Hearing Research*. 1972; 15:639–653.
- Dell GS. A spreading-activation theory of retrieval in sentence production. *Psychological Review*. 1986; 93:283–321. [PubMed: 3749399]
- Dell GS, Burger LK, Svec WR. Language production and serial order: A functional analysis and a model. *Psychological Review*. 1997; 104:123–147. [PubMed: 9009882]
- Dell GS, Schwartz MF, Martin N, Saffran EM, Gagnon DA. Lexical access in aphasic and nonaphasic speakers. *Psychological Review*. 1997; 104:801–838. [PubMed: 9337631]
- DiSimoni FG, Keith RL, Darley FL. Prediction of PICA overall score by short versions of the test. *Journal of Speech and Hearing Research*. 1980; 23:511–516. [PubMed: 7421154]
- Drummond, SS. *Dysarthria Examination Battery*. Communication Skill Builders; Tucson, AZ: 1993.
- Dunn ND, Russell SS, Drummond SS. Effect of stimulus context and response coding variables on word retrieval performances in dysphasia. *Journal of Communication Disorders*. 1989; 22:209–223. [PubMed: 2738193]
- Fossett, TRD.; McNeil, MR.; Pratt, SR. *The effects of speaking rate on phonological encoding in normal adults*. University of Pittsburgh; PA: 2000. Manuscript in preparation
- Frisch SA, Wright R. The phonetics of phonological speech errors: An acoustic analysis of slips of the tongue. *Journal of Phonetics*. 2002; 30:169–162.

- Fozo MS, Watson BC. Task complexity effect on vocal reaction time in aged speakers. *Journal of Voice*. 1998; 12(4):404–414. [PubMed: 9988027]
- Garnham A, Shillcock RC, Brown GDA, Mill AID, Cutler A. Slips of the tongue in the London-Lund corpus of spontaneous conversation. *Linguistics*. 1980; 19:805–817.
- Helms-Estabrooks N, Ramage A, Bayles KA, Cruz R. Perseverative behavior in fluent and non-fluent aphasic adults. *Aphasiology*. 1998; 12:689–698.
- Goodglass, H.; Kaplan, E.; Barresi, B. The assessment of aphasia and related disorders. 3rd edition. Lippincott Williams & Wilkins; Philadelphia: 2001.
- Haley KL, Bays GL, Ohde RN. Phonetic properties of aphasic-apraxic speech: A modified narrow transcription analysis. *Aphasiology*. 2001; 15:1125–1142.
- Kello CT. Patterns of timing in the acquisition, perception, and production of speech. *Journal of Phonetics*. 2003; 31:619–626.
- Klapp ST. Reaction time analysis of two types of motor preparation for speech articulation: Action as a sequence of chunks. *Journal of Motor Behavior*. 2003; 35:135–150. [PubMed: 12711585]
- Kohn SE, Smith KL. Between-word speech errors in conduction aphasia. *Cognitive Neuropsychology*. 1990; 7:133–156.
- Laganaro M. Patterns of impairments in AOS and mechanisms of interaction between phonological and phonetic encoding. *Journal of Speech, Language, and Hearing Research*. 2012; 55:S1535–S1543.
- Levelt, WJM. *Speaking: From intention to articulation*. The MIT Press; Cambridge, MA: 1989.
- Maas E, Mailend M-L. Speech planning happens before speech execution: Online reaction time methods in the study of apraxia of speech. *Journal of Speech-Language-Hearing Research*. 2012; 55:S1523–S1534.
- MacKay DG, James LE. Sequencing, speech production, and selective effects of aging on phonological and morphological speech errors. *Psychology & Aging*. 2004; 19:93–107. [PubMed: 15065934]
- Mailend M-L, Maas E. Speech motor programming in apraxia of speech: Evidence from a delayed picture-interference task. *American Journal of Speech Language Pathology*. 2013; 22:S380–396. [PubMed: 23695911]
- Martin N, Dell GS. Perseverations and anticipations in aphasia: Primed intrusions from the past and future. *Seminars in Speech and Language*. 2004; 25:349–362. [PubMed: 15599824]
- Martin N, Dell GS, Saffran EM, Schwartz MF. Origins of paraphasias in deep dysphasia: Testing the consequences of a decay impairment to an interactive spreading activation model of lexical retrieval. *Brain and Language*. 1994; 47:609–660. [PubMed: 7859057]
- McNeil, MR.; Kent, RD. Motoric characteristics of adult aphasic and apraxic speakers.. In: Hammond, GR., editor. *Advances in psychology: Cerebral control of speech and limb movements*. Elsevier/ North Holland; New York: 1990. p. 349-386.
- McNeil MR, Pratt SR. A Standard Definition of Aphasia: Toward a general theory of aphasia. *Aphasiology*. 2001; 15:901–911.
- McNeil, MR.; Prescott, TE. *Revised Token Test*. Pro-Ed; Austin, TX: 1978.
- McNeil, MR.; Robin, DA.; Schmidt, RA. Apraxia of speech: Definition, differentiation and treatment.. In: McNeil, MR., editor. *Clinical management of sensorimotor speech disorders*. Thieme Medical Publishers; New York: 2009. p. 311-344.
- Odell K, McNeil MR, Rosenbek JC, Hunter L. Perceptual characteristics of consonant production by apraxic speakers. *Journal of Speech and Hearing Disorders*. 1990; 55:345–359. [PubMed: 2329797]
- Porch, BE. *Porch Index of Communicative Ability*. Consulting Psychologists Press Psychological Software Tools, Inc.; Palo Alto, CA: 1981. 2003
- Pouplier M, Goldstein L. Asymmetries in the perception of speech production errors. *Journal of Phonetics*. 2005; 33:47–75.
- Pouplier M, Marin S, Waiti S. Voice onset time in consonant cluster errors: Can phonetic accommodation differentiate cognitive from motor errors? *Journal of Speech, Language, and Hearing Research*. 2014; 57:1577–1588.

- Raven, JC. The Coloured Progressive Matrices. The Psychological Corporation; New York: 1965.
- Roelofs A. Phonological segments and features as planning units in speech production. *Language and Cognitive Processes*. 1999; 14:173–200.
- Roelofs A. The WEAVER model of word-form encoding in speech production. *Cognition*. 1997; 64:249–284. [PubMed: 9426503]
- Rogers MA, Storkel HL. Reprogramming phonologically similar utterances: The role of phonetic features in pre-motor encoding. *Journal of Speech Language, and Hearing Research*. 1998; 41:258–274.
- Ruml W, Caramazza A, Capasso R, Miceli G. Interactivity and continuity in normal and aphasic language production. *Cognitive Neuropsychology*. 2005; 22:131–168. [PubMed: 21038244]
- Schmidt, RA.; Lee, TD. From principles to application. 5th edition. Human Kinetics; Champaign, IL: 2014. Motor learning and performance..
- Schneider, W.; Eschman, A.; Zuccolotto, A. E-Prime's user guide. Psychology Software Tools, Inc; Pittsburgh: 2002.
- Schwartz MF, Saffran EM, Bloch DE, Dell GS. Disordered speech production in aphasic and normal speakers. *Brain and Language*. 1994; 47:52–88. [PubMed: 7922477]
- Shriberg, LD.; Kent, RD. Clinical Phonetics. 2nd edition. Allyn & Bacon; Boston: 1995.
- Shuster LI, Wambaugh JL. Perceptual and acoustic analyses of speech sound errors in apraxia of speech accompanied by aphasia. *Aphasiology*. 2000; 14:635–651.
- Stemberger JP. Speech errors in early child language production. *Journal of Memory and Language*. 1989; 28:164–188.
- Towne RL, Crary MA. Verbal reaction time patterns in aphasic adults: Consideration for apraxia of speech. *Brain & Language*. 1988; 35:138–153. [PubMed: 2460184]
- Tsao Y-C, Weismer G, Iqbal K. The effect of intertalker speech rate variation on acoustic vowel space. *Journal of the Acoustical Society of America*. 2006; 119(2):1074–1082. [PubMed: 16521769]
- Van der Merwe, A. A theoretical framework for the characterization of pathological speech sensorimotor control.. In: McNeil, MR., editor. Clinical management of sensorimotor speech disorders. Thieme Medical Publishers; New York: 2009. p. 1-25.
- Vousden JI, Brown GDA, Harley TA. Serial control of phonology in speech production: A hierarchical model. *Cognitive Psychology*. 2000; 41:101–175. [PubMed: 10968924]
- Vousden J,I, Maylor EA. Speech errors across the lifespan. *Language Processes*. 2006; 21:48–77.
- Wambaugh JL, Duffy JR, McNeil MR, Robin DA, Rogers MA. Treatment guidelines for acquired apraxia of speech: A synthesis and evaluation of the evidence. *Journal of Medical Speech-Language Pathology*. 2006; 14:xv–xxxiii.
- Wilshire CE. The “tongue twister” paradigm as a technique for studying phonological encoding. *Language and Speech*. 1999; 42:57–82.
- Wilshire CE, McCarthy RA. Experimental investigations of an impairment in phonological encoding. *Cognitive Neuropsychology*. 1996; 13:1059–1098.
- Wilson RH, Antablin JK. A picture identification task as an estimate of the word-recognition performance of nonverbal adults. *Journal of Speech and Hearing Disorders*. 1980; 45:223–238. [PubMed: 7442156]

Figure 1.
Percentage of each error type relative to the opportunities for the error to occur for normal healthy controls (NHC)

Figure 2.

Percentage of each error type relative to the opportunities for the error to occur for persons with aphasia (PWA)

NHC and PWA Anticipation to Perservation (AP) Ratio

Figure 3.

Arcsine transformation values for the anticipation to perseveration error ratio for normal healthy controls (NHC) and persons with aphasia (PWA) across the three speaking rates

NHC and PWA Anticipation to Exchange (AE) Ratio

Figure 4. Average arcsine transformed values for the anticipation to exchange error ratio for normal healthy controls (NHC) and persons with aphasia (PWA) across the three speaking rates

Table 1

Demographic information for normal healthy controls (NHC) and persons with aphasia (PWA)

NHC	Age	Education	PWA	Age	Education	Lesion Data
1	63	14	1	51	14	L post. temporal; L inferior parietal
2	58	12	2	52	16	----
3	75	18	3	65	16	----
4	72	12	4	46	18	L MCA; L frontotemporal
5	55	16	5	70	14	Periventricular white matter; chronic ischemic effect
6	67	16	6	70	16	L temporal and frontal
7	73	16	7	51	16	----
8	72	16	8	52	20	----
9	53	16	9	50	18	L frontotemporal extending into BG
10	66	16	10	60	12	----
11	62	18	11	66	16	----
12	56	16	12	53	12	L BG; posterior parietal
13	49	18	13	45	12	L MCA distribution
14	41	18	14	77	16	L post. temporal-parietal
15	53	18	15	36	13	Ant. and post. L MCA; L putamen
16	47	22	16	48	14	----
M	60	16.38		56	15.19	
SD	10.2	2.45		11.08	2.34	

Note. Years of education represents a minimum number of years as some participants had completed additional education that did not result in another degree (i.e., post graduate work).

Ant. = anterior; BG = Basal Ganglia; L = Left; MCA = Middle Cerebral Artery; Post. = posterior

Table 2

Descriptive data for persons with aphasia (PWA) on the Boston Diagnostic Aphasia Examination (BDAAE)

Participant	Word Comp	Commands	Complex Ideational Material	Oral Expressions	Automatized Sequences	Repetition of Single Words	Repetition of Short Sentences	WNT short
1	100	90	67	100	100	100	90	87
2	100	100	100	100	100	100	100	87
3	100	100	100	100	80	50	100	73
4	100	100	83	100	100	100	100	100
5	94	100	83	100	100	100	100	60
6	100	90	67	100	100	100	100	86
7	68	80	67	100	100	100	90	73
8	100	100	100	100	100	100	100	93
9	100	100	100	100	100	100	100	93
10	100	90	100	100	60	50	100	40
11	100	90	100	83	100	100	90	86
12	100	100	100	100	100	100	100	100
13	100	100	100	100	80	100	90	66
14	97	100	100	100	100	50	100	100
15	100	90	100	100	100	100	100	80
16	94	100	100	100	100	100	100	80
M	97.06	95.63	91.69	98.94	95.00	90.63	97.50	81.50
(SD)	8.03	6.29	13.51	4.25	11.55	20.16	4.47	16.26