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A Sub-Acute Case of Resolving Acquired Apraxia of Speech and Aphasia

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Abstract

Apraxia of speech (AOS) is a neurogenic, motor speech disorder that disrupts the planning for speech production. However, there are only a few reports that have described the evolution of stroke-induced AOS symptoms in the acute or sub-acute phase of recovery. The purpose of this report was to provide a data-based description of an individual with sub-acute AOS and aphasia followed from 1 month post-onset a stroke to 8 months post-stroke. Six data collection sessions were conducted at periodic intervals using narrative and procedural discourse tasks and a series of speech and language analyses were completed. The language analyses involved measures of language content and efficiency. The speech production analyses examined the percentage and frequency of errors as well as determining the dominant types of errors produced within and across data collection sessions. For this individual, measures of language content and communication efficiency improved over the six sampling occasions. The number of speech production errors significantly declined after the first data collection session and then gradually over the subsequent sessions. This individual produced five dominant error types within and across sessions. The majority of these error types are behaviors that occur in chronic AOS, but do not distinguish AOS from other acquired neurogenic communication disorders. Due to the lack of research involving acute/sub-acute individuals with AOS additional research is warranted to better understand the evolution of AOS including the speech behaviors that are observed in the acute/sub-acute phase vs. the chronic phase of recovery.

Keywords: Apraxia of speech; Aphasia; Treatment; Neurogenic communication disorders; Speech; Language

Introduction

Stroke-induced acquired apraxia of speech (AOS) is a neurogenic, sensori-motor speech disorder. It is considered to be a disruption of the ability to plan or program the movements necessary for correct articulation [1]. More specifically, AOS is not thought to be a disorder of language (e.g., aphasia); words and the sounds comprising those words are processed accurately. Instead, AOS reflects difficulties in retrieving previously learned movement patterns needed to translate correctly selected sounds/syllables/words to articulated speech [2]. Unlike dysarthria, there are no clinically evident impairments in neuromuscular physiology. Although AOS is a unique clinical entity that differs from aphasia and dysarthria, it can co-occur with both of these other disorders. In particular, AOS rarely occurs without nonfluent aphasia.

AOS may range in severity from a complete inability to produce speech to mild, infrequent sound distortions. The symptoms of AOS that are considered to be necessary for its diagnosis include slow rate of speech production, disrupted prosody, and sound errors that are frequently distortions [3]. Symptoms that often occur with AOS, but that do not differentiate it from other disorders include trial-and-error articulatory groping, sound and syllable repetitions, self-awareness of errors with attempts to correct, speech initiation difficulties, and increased error rates associated with more complex utterances. Controversy continues regarding the relative consistency of error location and error type in AOS [1].

Stroke is the most common etiology for AOS [1]. Damage to cortical and/or subcortical areas of the language dominant hemisphere have been associated with AOS [4]. Debates concerning the specific brain regions implicated in AOS remain unresolved [5-7]. AOS has also been found with neurodegenerative disease (i.e., primary progressive AOS) [8].

Treatment for acquired apraxia of speech (AOS) has taken numerous forms, with positive outcomes reported for most treatments [9]. Following a critical evaluation and synthesis of the AOS treatment literature, AOS treatment guideline developers concluded that "taken as a whole, the AOS treatment literature indicates that individuals with AOS may be expected to make improvements in speech production as a result of treatment, even when AOS is chronic" (p.lxii) [10]. The AOS guidelines developers grouped treatment studies by general focus: articulatory-kinematic, rate/rhythm control, intersystemic reorganization, and alternative/augmentative communication. The majority of evidence supporting AOS treatment has been derived from studies focused on treatments designed to improve articulation (i.e., articulatory-kinematic therapies) [10,11].

The AOS treatment evidence-base has been predominated by studies of persons with *chronic* AOS. Of 146 participants in the AOS guidelines report, approximately one-third were less than 6 months post-stroke [9]. In treatment efficacy reports, the majority of participants in the sub-acute phase have been described as having severe AOS and their response to treatment has been overwhelmingly positive. Approaches to treatment with non chronic AOS speakers have been widely varied (e.g., gestural training, articulatory-kinematic treatment, environmental manipulation). Reports of treatment outcomes have typically been limited to post treatment changes in test scores. That is, treatment efficacy studies have provided relatively little information concerning the progression of change in response to treatment with persons with non chronic AOS.

In the few reports that have focused on the evolution of stroke-induced AOS symptoms, investigators have noted the presence of mutism in acute or early sub-acute AOS that resolved to less severe AOS [12-14]. In these cases, persisting AOS symptoms included initiation difficulties, articulatory errors, and dysprosody. These reports provided largely anecdotal findings, with limited data.

The purpose of the present report was to provide a data-based description of an individual with sub-acute AOS and aphasia followed from one month post-onset a stroke to eight months post-onset with a course of treatment that primarily focused on AOS.

Method

Participant

A right-handed, 60 year-old Caucasian gentleman suffered a left cerebral vascular accident in April of 2013. He was a native English speaker with 20 years of education. The gentleman was a physician and his daily work duties were comprised of evaluating and treating patients, conducting clinical research trials as well as administrative responsibilities.

He was administered tPA less than two hours after the onset of his stroke symptoms. The symptoms included right handed clumsiness and an inability to produce speech prior to the arrival of emergency medical services.

An MRI revealed evolving patchy areas of thromboembolic infarction involving primarily the posterior aspect of the left middle cerebral artery territory and to a lesser degree the left posterior cerebral artery (PCA) territory. Also observed were tiny foci of suspected coincident embolic lesions involving the left posterior inferior cerebellar artery and right PCA. The participant also had evidence of an old thromboembolic infarct in the right parietal lobe that was asymptomatic.

A speech-language evaluation was conducted after admission to the hospital on the day of the stroke. The participant appeared to have no difficulty with simple comprehension tasks (i.e., identifying pictures, objects, 1-2 step commands, yes/no questions), but exhibited greater impairment on verbal expression tasks. He struggled with automatic speech tasks, repetition tasks (e.g., repeating phonemes, words, phrases), naming, and answering simple questions (e.g., object function, phrase completion). The participant received speech therapy six days a week during his two week hospitalization.

After hospital discharge, the patient had an outpatient speech-language evaluation with subsequent therapy. He was diagnosed with moderate-severe AOS and mild-moderate aphasia. The participant had mild functional level auditory and reading comprehension deficits and moderate deficits in writing (i.e., words/sentences). He continued to exhibit moderate to severe deficits with verbal expression tasks including significant difficulty repeating words, phrases and sentences. The participant required visual/auditory cues to complete these tasks with more cueing required as stimuli length increased. He displayed awareness of his errors, but his attempts at self-correction tended to be unsuccessful. The participant attended outpatient therapy five times a week for the first month, with sessions gradually decreasing in frequency over the next several months.

Evaluation

The participant was referred to the Department of Veterans Affairs, Salt Lake City Health Care System's Aphasia/Apraxia Research Program at one month post stroke. The presence of AOS was determined using the diagnostic criteria described by McNeil et al. [2]. Speech samples were obtained employing the following elicitation tasks: 1) Increasing Word Length, and Repeated Trials subtests of the Apraxia Battery for Adults – 2nd Edition [15]; 2) narrative and procedural discourse tasks [16]; 3) Assessment of Intelligibility of Dysarthric Speech (AIDs) [17]; 4) sentence repetition [18]; and 5) multisyllabic word repetition [19]. The following behaviors deemed necessary for the diagnosis of AOS were demonstrated by the participant: slow rate of speech production (including syllable segregation), sound errors that were relatively consistent in type and location across repeated trials.

The participant received the diagnosis of anomic aphasia on the basis of the Western Aphasia Battery-Revised (WAB-R) [20]. The participants' productive verbal language primarily involved sentences that were broken up by language revisions and struggles with speech production. The participant did not exhibit symptoms of dysarthria as described by Duffy [1].

See Table 1 for assessment results at time of enrollment and subsequent intervals. During the time he was enrolled in the research program, he continued to receive outpatient speech services through the hospital outpatient program. Every effort was made not to duplicate treatment approaches between the hospital outpatient program and the research program.

Treatment

Combined Aphasia and Apraxia of Speech Treatment (CAAST) was selected as the model for treatment. CAAST was modified to accommodate the participant's higher level language and rapidly improving speech production abilities [21]. CAAST is unique in that it combines a language based treatment, CAAST is unique in that it combines a language based treatment, Response Elaboration Training (RET) and Modified RET with Sound Production Treatment (SPT) [22-24]. During treatment, the participant was asked to produce statements based on selected stimuli. In keeping with M-RET, there were no constraints on his production and he was encouraged to express any ideas these stimuli brought to mind. Sound production errors were identified in his response and the SPT hierarchy was incorporated to facilitate correct productions [25]. The SPT hierarchy progresses from sound error identification with clinician modeling of the correct production, production facilitated by integral stimulation and finally articulatory placement cues to assist production. The participant rarely required more than identification of the error in order to facilitate a correct production.

Treatment was provided five days per week by two ASHA certified speech-language pathologists who shared the evaluation/treatment responsibilities. Treatment included three phases, modified by the level of stimuli being used. Each phase was comprised of twenty sessions and each session was approximately sixty minutes in length. The first phase used action pictures/multisyllabic words as stimuli. The second phase used verbal narratives about familiar topics, and the third phase focused on short verbal presentations with varying context (i.e., having another person in the treatment session, or using a different location for treatment). Following completion of the three phases, treatment frequency decreased to three days per week, two

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days per week, and then once per week. These subsequent sessions addressed participant generated concerns as he gradually returned to full time employment. Two follow up visits were conducted seven weeks after the last treatment session.

Data Collection, Dependent Variables, & Data Analyses

Prior to initiating CAAST, after each treatment phase, and 7 weeks post treatment narrative and procedural discourse samples were collected using stimuli and procedures established by Nicholas and Brookshire [16]. These discourse samples were analyzed for content as well as efficiency of language production. A subset (Set B) was used to examine speech production [26]. These data collection sessions were defined by months post onset (MPO) in order to provide a data based description of the participant's speech and language skills from 1 MPO to 8 MPO.

Language Content and Efficiency: Production of words and correct information units (CIUs) in response to narrative and procedural discourse served as the primary dependent variables for language production [16]. Responses are counted as words if they are intelligible in the context in which they are produced. CIUs reflect the appropriateness, relevancy and informativeness of words produced by a speaker in relation to a particular topic [16]. Procedures described by Nicholas and Brookshire for calculating words and CIUs were utilized [16]. All samples were timed and three measures of efficiency were calculated: words per minute, CIUs per minute and CIUs compared to total words (percent CIUs) [16].

Measure	Initial	Interim	Post/Follow-up
Western Aphasia Battery [20] AQ; Type	(1 MPO) 91.4; Anomic	(3 MPO) 98.2; Minimal Aphasia	Did Not Test (DNT)
Porch Index of Communicative Ability [27] Overall Percentile Verbal Percentile Auditory Percentile Reading Percentile	(1 MPO) 88% 64% 72% 89%	(3 MPO) 94% 86% 74/99% 97/99%	(5 MPO) 95% 85% 74/99% 97/99%
Test of Nonverbal Intelligence-4 [30] Percentile	(2 MPO) 70%		DNT
Test of Adolescent/Adult Word Finding [31] Total Raw Score	(1 MPO) 83/107		(8 MPO) 101/107
Assessment of Intelligibility of Dysarthric Speech [17] Word Level *Intelligibility* Sentence Level Intelligibility	(2 MPO) 94% 98%		DNT
Apraxia Battery for Adults-2 [15] Subtest 2A: Words of Increasing Length Subtest 2B: Words of Increasing Length	(1 MPO) 80% 70%		DNT
Aphasia Communication Outcome Measure (38 items) [28,29] Patient - ACOM T-Score Spouse - ACOM T-Score	(2 MPO) 52 62		(6 MPO) 72 72
Speech Production Accuracy [18,19] 3-syllable words 4-syllable words 5-syllable words Phrases/Sentences (5-10 syllables in length/1-3 syllable words)	(1 MPO) 60% 25% 0% 14%		5 (MPO) 80% 90% 75% 87%

Table 1: Assessment results at each interval with month post onset (MPO) reported

Speech Production Errors: Errors in a subset of the narrative and procedural discourse samples served as the primary dependent variable for examining speech production. Segments of speech production that were deemed to be in error based on auditory-perceptual listening tasks completed from high quality recordings of each data collection session were marked on a written transcript. Errors were then analyzed and coded according to predetermined categories created to capture and describe errors observed in data collection sessions. See Appendix A for an outline of types of errors and definitions for each error type. Both treating SLPs listened to each sample and coded auditory-perceptual errors. Any error coding disagreements between the two SLPs were resolved by a non-project SLP that used the same procedures outlined above.

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For all data collection sessions, the overall percentage of errors, the frequency of errors and dominant types of errors were calculated. The overall percentage of errors was computed by dividing the number of errors by the total of number of words produced at each sampling occasion. The frequency of errors was computed by dividing the number of errors by overall sampling time for each session. Dominant types of errors were errors that occurred across five or more sampling sessions with an error percentage of 9% or greater.

Reliability

To assess inter-rater agreement for language content and efficiency from the narrative and procedural discourse tasks, a non-project SLP calculated both words and CIUs for ten percent of the randomly selected stimuli. Mean point-to-point inter-rater agreement was 99% for words (range 97% to 100%) and 97% for CIUs (range 92% to 100%).

Results

Data representing measures of language content and efficiency are presented in Table 2. The number of CIUs produced initially declined over the first three sampling occasions from 882 to 853, but increased on the subsequent sampling occasions to 1204 CIUs by 8 MPO. A similar trend was observed for number of words produced with a decline after the first three sampling occasions from 2053 to 993. However, the number of words produced gradually increased over the final three sampling occasions with 1359 words produced on the final sampling occasion.

Administration Time	Number of CIUs	Number of Words	Percent CIUs	Total Time	CIUs per minute	Words per minute
1 MPO	882	2053	43%	33:15	26.5	61.7
3 MPO	848	1041	81%	14:30	58.5	71.8
4 MPO	853	993	86%	12:55	66.1	76.9
5 MPO	893	1068	84%	13:06	68.2	81.5
6.5 MPO	1020	1169	87%	14:44	69.3	79.4
8 MPO	1204	1359	89%	15:47	76.3	86.1

Table 2: Language content and efficiency based on narrative and procedural discourse tasks from Nicholas & Brookshire [16]. CIUs = correct information units; MPO = months post onset

Measures of communication efficiency (i.e., percent CIUs, CIUs per minute, words per minute) improved over the six sampling occasions. Percent CIUs was 46% at the initial sampling occasion and increased to 89% at the final sampling occasion. CIUs per minute increased from 26.5 at 1 MPO to 76.3 at 8 MPO. Words per minute increased from 61.7 to 86.1 from the first to the final sampling.

The overall percentage of speech production errors at each sampling occasion is presented in Figure 1. The overall percentage of errors at 1 MPO was 46% and the percentage of errors significantly decline at subsequent sampling occasions with 10% at the final sampling occasion. The frequency of speech production errors per minute are displayed in Figure 2 across sampling times. At the initial sampling occasion, there were almost 29 errors per minute. The number of errors per minute sharply declined on successive sampling occasions with 8.5 errors per minute at 8 MPO.

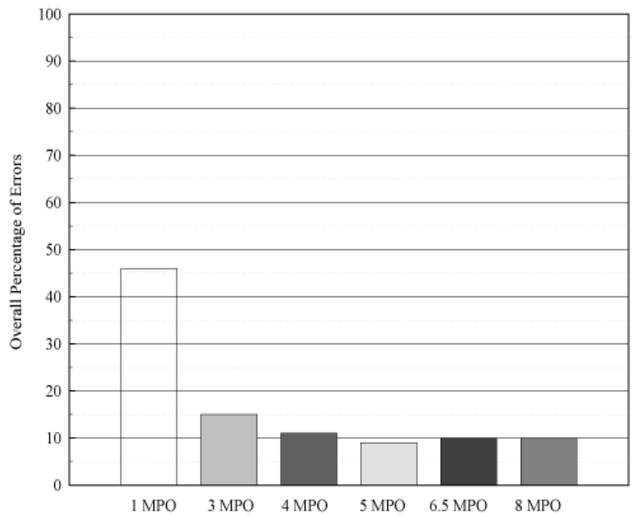

Figure 1: Overall percentage of speech production errors at each sampling time based on months post onset (MPO)

The percentages of types of speech production errors produced at each sampling occasion are displayed in Table 3. There were two dominant types of errors that occurred across all six sampling occasions with a percentage of 10% or greater. The first dominant type of error was intrusive/inaccurate phoneme production with percentages ranging from 13% to 21% across sampling occasions. The second dominant type of error was prolongations of vowels and consonants with percentages varying between 11% and 31%.

There were three dominant types of errors that occurred across five sampling occasions with a percentage of 9% or greater. One dominant type of error was the intrusive/inaccurate syllable production with percentages ranging from 14% to 27%. The next dominant type of error was audible groping with percentages varying between 10% and 36%. The final dominant type of error was a repetition of a phrase or revision of language with percentages ranging from 9% to 14%.

Discussion

The purpose of this report was to provide a data-based description of an individual with sub-acute AOS and aphasia followed from 1 MPO a stroke to 8 MPO with a course of treatment that primarily focused on AOS.

For this individual, measures of communication efficiency (i.e., percent CIUs, CIUs per minute, words per minute) improved considerably from the initial session at 1 MPO and the final session at 8 MPO suggesting an overall improvement in his language functioning. However, the language content measures, number of CIUs and words per minute did not reach the normal non-brain damaged range reported by Nicholas and Brookshire [16]. This is likely due to the individual's slow rate of speech which is a salient feature of AOS. The overall gains observed in language functioning was further evidenced by the improvement on the subsequent administrations of the WAB-R and the Porch Index of Communicative Ability (PICA) both standardized aphasia tests used

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to evaluate language functioning [20,27]. At 1 MPO, he had an aphasia quotient of 91.4 on the WAB-R classifying him as having anomic aphasia and at 5 MPO he scored 98.2 indicating minimal aphasia [20]. This same pattern was observed with administrations of the PICA with a score of 88% at 1 MPO and 95% at 5 MPO indicating mild aphasia initially and minimal aphasia at the final administration [27].

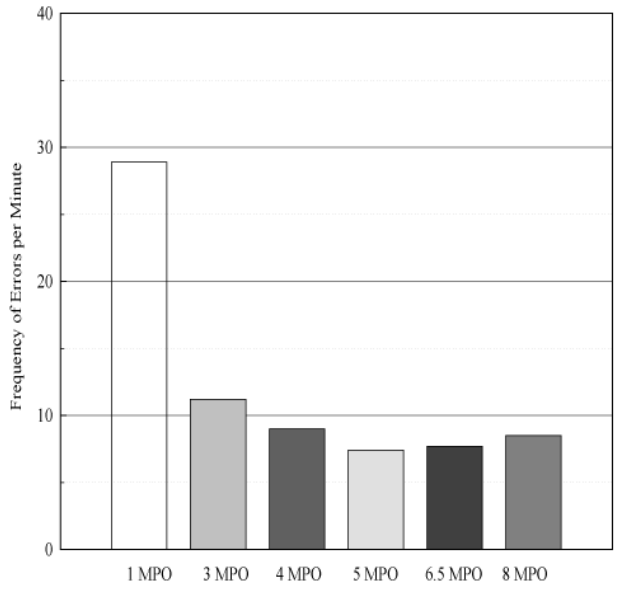

Figure 2: Frequency of speech production errors per minute at each sampling time based on months post onset (MPO)

Speech production errors declined over the six sampling occasions with a sharp decline after the initial session at 1 MPO and then a gradual decline in the number of errors over the subsequent sessions. The same trend was observed when error frequency was examined based on errors per minute. The dominant types of errors produced by this speaker were consonant and vowel prolongations, intrusive/inaccurate phonemes, intrusive/inaccurate syllables, audible groping, and repetition of phrase/revisions of language. These five error types were more frequently produced within and across sampling occasions. Consonant and vowel prolongations are frequently observed behaviors in individuals with chronic AOS and reflect increased movement durations for articulation (e.g., transitioning among individual speech segments/phonemes) [3]. Interestingly, three of these errors types are behaviors that are found to occur in individuals with chronic AOS, but do not differentiate AOS from other communication disorders [3]. These behaviors include intrusive/inaccurate phonemes, syllable repetitions, and audible groping. Due to the lack of data and limited description of patients with acute/sub-acute AOS it remains unknown if these are behaviors that are frequently observed in patients diagnosed with AOS in this phase of recovery.

The other dominant type of error was repetition of phrase/revisions of language. This type of error typically is not a behavior associated with chronic AOS, but it is a behavior that occurred fairly frequently (i.e., comprised 9% to 14% of errors across five sampling occasions) for the individual described in this report. It is possible that this behavior could reflect motor planning difficulties in terms of retrieving a motor plan or initiating a motor plan, hence repeating a previous phrase or revising language until a motor plan could be accessed or initiated. Or it is possible this behavior reflects higher level language deficits. The language measures carried out as part of this report revealed an overall improvement in language functioning (i.e., minimal aphasia) by 5 MPO. However, the tests/tools used to examine language functioning many not have been sensitive to higher level language deficits this individual was exhibiting and consequently the repetition of a phrase/revisions of language that were found to be a dominant type of error when examining this individual's speech production.

Type of Errors	1 MPO	3 MPO	4 MPO	5 MPO	6.5 MPO	8 MPO
Intrusive/ inaccurate production of a word	21%	6%	5%	4%	7%	6%
Intrusive/ inaccurate production of a phoneme	13%	16%	21%	18%	20%	15%
Intrusive/ inaccurate production of a syllable	27%	18%	21%	4%	18%	14%
Prolongation of vowel or consonant	17%	31%	14%	16%	18%	11%
Partial voicing or devoicing	1%	3%	2%			
Excessive Aspiration			2%		2%	1%
Audible Groping	8%	10%	11%	43%	12%	36%
Vowel error or distortion	1%	3%	7%	2%	7%	3%
Reduced cluster/blend			3%			
Substitution	1%		5%		2%	
Insertion of a phoneme	1%		3%			
Errors of prosody/stress		3%	2%	4%	2%	
Repetition of a phrase/ revision of language	10%	10%	4%	9%	12%	14%

Table 3: Speech production errors: Percentage of types of errors produced at each sampling time

The Aphasia Communication Outcome Measure (ACOM) [28] was administered to the patient and his spouse at 2 MPO and 6 MPO. The ACOM is a patient-reported and surrogate-reported assessment of communicative functioning. The version of the ACOM used in this report was a 38-item version that included content related to everyday speaking, listening, reading and writing activities [29]. In rating his communication skills, the patient had a 20 point gain from the first to the subsequent administration of the ACOM and the patient's spouse had a 10 point gain. The increase in communication function ratings via the ACOM, also serve to support the overall improvement in language and speech that was observed with other measures conducted over the eight months of following this individual in the sub-acute phase of recovery (See Table 1 for further details).

The other dominant types of errors of repetition of syllables, phonemes and audible groping identified in this individual's speech production are behaviors that occur in chronic AOS, but do not distinguish it from other acquired neurogenic communication disorders. Due to the lack of research involving acute/sub-acute individuals with AOS it remains unclear how speech behaviors may vary over the course of recovery and how these behaviors may evolve over time as these patients recover and enter what is considered a chronic phase of AOS.

This report only provided data and description of one individual with sub-acute AOS and aphasia. This report is only one of a few reports to examine individuals with AOS in the acute/sub-acute phase. Additional AOS research involving individuals in the acute/sub-acute phase of recovery is warranted to better understand the evolution of the disorder including the speech behaviors that are observed in the acute/sub-acute phase vs. the chronic phase of recovery. Ideally, it would be most beneficial to follow a number of patients with AOS from the acute/sub-acute phase through to the chronic phase of recovery completing a series of measures at regular intervals in order to gain a better insight into the evolution of AOS.

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References

1. Duffy JR (2013) Motor speech disorders. Substrates, differential diagnosis, and management (3rdedn), Elsevier Mosby.
2. McNeil MR (2009) Clinical management of sensorimotor speech disorders (2ndedn), Thieme.
3. McNeil MR (1997) Clinical management of sensorimotor speech disorders. New York.
4. Chapey R (2008) Language intervention strategies in aphasia and related neurogenic communication disorders (5thedn), Lippincott, Williams, & Wilkins.
5. Dronkers N (1996) A new brain region for coordinating speech articulation. Nature 384: 159-161.
6. Hillis AE, Work M, Barker PB, Jacobs MA, Breese EL, et al. (2004) Re-examining the brain regions crucial for orchestrating speech articulation. Brain 127: 1479-1487.
7. Trupe LA, Varma DD, Gomez Y, Race D, Leigh R, et al. (2013) Chronic apraxia of speech and Broca's area. Stroke 44: 740-744.
8. Josephs KA, Duffy JR, Strand EA, Machulda MM, Senjem JL, et al. (2012) Characterizing a neurodegenerative syndrome: primary progressive apraxia of speech. Brain 135: 1522-1536.
9. Wambaugh J, Duffy J, McNeil M, Robin D, Rogers M (2006a) Treatment guidelines for acquired apraxia of speech: A synthesis and evaluation of the evidence. Journal of Medical Speech Language Pathology 14: xv-xxxiii.
10. Wambaugh J, Duffy J, McNeil M, Robin D, Rogers M (2006b) Treatment guidelines for acquired apraxia of speech: Treatment descriptions and recommendations. Journal of Medical Speech Language Pathology 14: xxxv-ixvii.
11. Ballard K, Wambaugh J, Duffy J, Layfield C, Maas E, et al. (2013) Treatment guidelines for acquired apraxia of speech: Systematic review of studies 2004 to 2012. American Speech-Language-Hearing Convention.
12. Ojha PK, Nandavar S, Pearson DM, Demchuk AM (2011) Aphemia as a presenting symptom in acute stroke. Neurol India 59: 432-434.
13. Patidar Y, Gupta M, Khwaja GA, Chowdhury D, Batra A, et al. (2013) A case of crossed aphasia with apraxia of speech. Ann Indian Acad Neurol 16: 428-431.
14. Helm-Estabrooks N, Holland A (1998) Approaches to the treatment of aphasia. Singular Publishing Group, Inc.
15. Dabul BL (2000) Apraxia battery for adults (2ndedn), Pro-ed.
16. Nicholas LW, Brookshire RH (1993) A system for quantifying the informativeness and efficiency of the connected speech of adults with aphasia. J Speech Hear Res 36: 338-350.
17. Yorkston K, Beukelman D (1981) Assessment of intelligibility of dysarthric speech. Pro-Ed.
18. Wambaugh J, West J, Doyle P (1998) Treatment for apraxia of speech: Effects of targeting sound groups. Aphasiology 12: 731-743.
19. Mauszycki S,Wambaugh J (2008) The effects of rate control treatment on consonant production accuracy in mild apraxia of speech. Aphasiology 22: 906-920.
20. Kertesz A (2007) The Western Aphasia Battery-Revised. Pearson.
21. Wambaugh J, Wright S, Nessler C, Delong C, Mauszycki S (2013) Effects of a Novel Treatment for Aphasia & Acquired Apraxia of Speech. American Speech-Language-Hearing Convention.
22. Kearns K (1985) Response elaboration training for patient initiated utterances. Clinical Aphasiology: 196-204.
23. Wambaugh J, Martinez A (2000) Effects of modified response elaboration training with apraxic and aphasic speakers. Aphasiology 20: 603-617.
24. Wambaugh J, Kalinyak-Fliszar M, West J, Doyle P (1998) Effects of treatment for sound errors in apraxia of speech. Journal of Speech, Language, and Hearing Research 41: 725-743.
25. Wambaugh J, Mauszycki S (2010) Sound production treatment: Application with severe apraxia of speech. Aphasiology 24: 814-825.
26. Brookshire R, Nicholas L (1994) Test-retest stability of measures of connected speech in aphasia. Clinical Aphasiology 22: 119-133.
27. Porch B (1981) The reference title should be: Porch Index of communicative ability [vol. 2]: Administration, scoring, and interpretation (3rdedn), Pro-Ed Inc.g.
28. Doyle P, Hula W, Hula S, Stone C, Wambaugh J, et al. (2012) Self-and surrogate-reported communication functioning in aphasia. Quality of Life Research 22: 1-11.
29. Winans-Mitrik R, Hula, W, Dickey M, Schumacher J, Swoyer B, et al. (2014) Description of an Intensive Residential Aphasia Treatment Program: Rationale, Clinical Processes and Outcomes. American Journal of Speech-Language Pathology.
30. Brown L, Serbenou RJ, Johnsen SK (2010) Test of Nonverbal Intelligence – 4. Pro-ed.
31. German DJ (1990) Test of Adolescent/Adult Word Finding. DLM.

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