HHS Public Access

Author manuscript

Semin Speech Lang. Author manuscript; available in PMC 2018 March 02.

Published in final edited form as:

Semin Speech Lang. 2018 February ; 39(1): 25–36. doi:10.1055/s-0037-1608853.

Contemporary Approaches to the Management of Post-stroke Apraxia of Speech

Alexandra Basilakos, Ph.D., CCC-SLP[1]

[1]Arnold School of Public Health, Department of Communication Sciences and Disorders, University of South Carolina, Columbia, South Carolina

Abstract

Apraxia of speech (AOS) is a motor speech disorder that disrupts the planning and programming of speech motor movements. In the acute stage of stroke recovery, AOS following unilateral (typically) left hemisphere stroke can occur alongside dysarthria, an impairment in speech execution and control, and/or aphasia, a higher-level impairment in language function. At this time, perceptual evaluation (the systematic, although subjective, description of speech and voice characteristics) is perhaps the only “gold standard” for differential diagnosis when it comes to motor speech disorders. This poses a challenge for speech-language pathologists charged with the evaluation of poststroke communication abilities, as distinguishing production impairments associated with AOS from those that can occur in aphasia and/or dysarthria can be difficult, especially when more than one deficit is present. Given the need for more objective, reliable methods to identify and diagnose AOS, several studies have turned to acoustic evaluation and neuroimaging to supplement clinical assessment. This article focuses on these recent advances. Studies investigating acoustic evaluation of AOS will be reviewed, as well as those that have considered the extent that neuroimaging can guide clinical decision making. Developments in the treatment of AOS will also be discussed. Although more research is needed regarding the use of these methods in everyday clinical practice, the studies reviewed here show promise as emerging tools for the management of AOS.

Keywords

Apraxia of speech; speech production assessment; differential diagnosis; diagnostic imaging

In the acute stage of stroke, several factors can affect a patient’s communicative abilities, including medical status and level of consciousness. These factors aside, damage to the dominant (typically left) hemisphere of the brain, particularly to regions within the territory of the middle cerebral artery, can be especially detrimental to higher-level linguistic functions (i.e., linguistic-symbolic planning) and lower-level planning, programming, and execution of speech motor movements. Damage to the brain’s speech and language regions can result in aphasia (a disorder of language), apraxia of speech (AOS; a disorder of speech

Address for correspondence: Alexandra Basilakos, Ph.D., CCC-SLP, Arnold School of Public Health, Department of Communication Sciences and Disorders, University of South Carolina, Columbia, SC 29208, (basilaka@mailbox.sc.edu).

Acute Care Management of Stroke; Guest Editor, Donna C. Tippett, M.P.H., M.A., CCC-SLP.

motor planning/programming), and/or dysarthria (a disorder of speech execution and control). Among the most important factors underlying the nature and severity of poststroke communication impairment(s) during early recovery and the chronic stage (i.e., >6 months poststroke) include the location of the infarct and the extent of brain damage.[1]

A proportion of stroke survivors recover spontaneously and do not go on to experience chronic deficits.[2] However, in the acute stage, the factors that are the best predictors of recovery are largely unknown, and it is difficult to identify patients who will have chronic speech and/or language impairments. Early assessment and accurate diagnosis of speech and language impairment are important for initiating treatment to maximize communication outcomes and to facilitate an expeditious return to premorbid activities. Unfortunately, however, speech and language assessment in the early stages after stroke can be challenging due to the inherent difficulty of differential diagnosis between AOS, aphasia, and dysarthria, especially if more than one disorder is present.[3] This is particularly true for the assessment of AOS, as it rarely occurs without the presence of concomitant aphasia.

This article focuses on recent developments in the clinical management of poststroke AOS. Many of the studies reviewed here focus on AOS in the chronic stage, or progressive AOS (PAOS)* resulting from a neurodegenerative process, as there is a lack of research with a specific focus on acute poststroke AOS.[6] Despite this critical gap in research, this review includes commentary regarding clinical and research applications for acute AOS. In the sections that follow, an overview of apraxic characteristics will be discussed—those unique to AOS, and those that can also occur in dysarthria and aphasia. This discussion will segue into a description of recent work that has investigated perceptual and acoustic measures that show promise in the differential diagnosis of AOS, as well as studies arguing for the use of neuroimaging to facilitate clinical decision-making.

This article focuses on emerging assessment approaches. Some of the methods discussed here may not be ready, in their current form, for immediate use in a clinical setting. Through discussion of these approaches though, this article addresses issues important to AOS management. Readers are therefore encouraged to stay abreast with research developments pertaining to AOS assessment and treatment, and clinicians are also reminded to refer to the American Speech-Language-Hearing Association's guidelines for evidence-based practice when considering new assessment or treatment approaches.[7]

CHARACTERISTICS OF APRAXIA OF SPEECH: SHARED AND UNIQUE

AOS results from an impairment at the stage of phonetic encoding, when motor codes and muscle commands are formulated from stored sensorimotor programs so that articulatory movements can be handled by the motor system for subsequent speech execution.[8] Speech production in AOS is characterized by articulatory imprecision, reduced speech rate, visible/audible groping for articulatory postures, and dysprosody, which cannot be explained by a peripheral deficit in muscle paralysis or paresis (i.e., dysarthria), or a linguistic impairment

*Over the past decade, AOS that results from a progressive, degenerative process has been identified.[4] Although direct comparisons of progressive and stroke-induced AOS are lacking, there may be subtle differences in AOS presentation based on etiology.[5]

(i.e., aphasia). Stroke-induced AOS rarely occurs in the absence of aphasia,[9,10] and it may occur concomitantly with dysarthria, especially during the acute phase of recovery, as dysarthria often resolves by the chronic phase if caused by a unilateral infarct.[3] Unlike aphasia or dysarthria, AOS is uniquely characterized by articulatory distortions of errors such as sound substitutions and additions.[11] Effortful struggle during speech production, as well as sound/syllable repetitions and prolongations may characterize all three disorders.[11] Despite that each of the three disorders arises from dysfunction at different levels of production, the overlap in perceptual characteristics often confounds perceptual evaluation and differential diagnosis.

Although distortion errors occur in dysarthria and to some extent in those with phonemic paraphasias (e.g., Basilakos et al[12] and Cunningham et al[13]), normative data are not currently available to guide clinical decision-making based on the frequency of sound distortions. Instead, the types of distortion errors present in speech may facilitate evaluation. In a study that utilized narrow transcription of apraxic speech, Cunningham and colleagues found that voicing errors, sound prolongations, and lingual distortion errors were more frequent in those with AOS when compared with individuals with phonemic paraphasias but without AOS.[13] When compared with dysarthria, error variability may facilitate the distinction between AOS and dysarthria (although this is not likely the case for the distinction between AOS and aphasia, see Haley et al[14]). For example, when considering just AOS and dysarthria, it is argued that individuals with AOS demonstrate greater variability in sound-level errors,[15] meaning errors are less predictable when compared with dysarthria, where muscle paralysis, paresis, and/or deficits with motor control may be consistently evident across speech production, diadochokinetic rates, and oral-motor speech tasks (e.g., lingual protrusion, lateralization, elevation, etc.; for further discussion, see Haley et al[14]). Moreover, the oral mechanism evaluation for an individual with AOS is generally unremarkable,[†] but the dysarthrias may be associated with facial, labial, and/or lingual weakness and reduced range of motion, especially if there is cranial nerve involvement.

This overlap in production characteristics poses a challenge to the clinical management of AOS, as a misdiagnosis may lead to the selection of inappropriate treatments. The topic of differential diagnosis has been covered extensively in the literature, and readers are referred to several reviews and studies for additional details regarding perceptual evaluation of disordered speech.[17–21] Readers are also encouraged to refer to the Apraxia of Speech Rating Scale for a recent guide for the perceptual evaluation of AOS, aphasia, and dysarthria.[11] This scale has shown promise for the differential diagnosis of all three disorders in the PAOS and aphasia literature, with some preliminary support for its use in poststroke AOS.[9,10,22]

Given the challenges of perceptual evaluation, several acoustic measures have been investigated to facilitate the objective description of speech production, ultimately informing differential diagnosis. The next section focuses on recent studies investigating these

[†]Unless in the case of concomitant nonverbal oral apraxia. Notably, not all with AOS have nonverbal oral apraxia, and not all with nonverbal oral apraxia have a diagnosis of AOS.[16]

approaches. Details of these studies are provided, as well as ways that these measures can be applied to clinical practice.

ACOUSTIC MEASURES TO FACILITATE DIFFERENTIAL DIAGNOSIS

For years following Darley's initial description of AOS,[18] several studies attempted to characterize the disorder using electromyographic/kinematic assessments and acoustic evaluations to supplement perceptually acquired data.[23–27] Although these studies have provided a wealth of information regarding AOS and have laid the ground-work for the first published diagnostic criteria,[28] many of these studies included small sample sizes and were limited in descriptions of participant characteristics. Research over the past decade has continued to investigate objective measures to characterize apraxic speech. Many of these studies have provided more descriptive participant characteristics and have yielded information about measures that capture variability between diagnostic classes. This work is important to the study of AOS for several reasons. First, the identification of acoustic measures that uniquely characterize AOS has the potential to reduce bias in assessment and in the evaluation of response to treatment. Such measures would also allow for more straightforward comparisons across studies by reducing differences in interpretation of diagnostic criteria (for discussion, McNeil et al[20]). Second, scoring criteria for perhaps the most widely used AOS assessment, the Apraxia Battery for Adults,[29,30] are rather outdated and can falsely diagnose an individual with conduction aphasia and frequent phonemic paraphasias as having AOS (for discussion, McNeil et al[19]). Developing more sensitive and specific measures of AOS is therefore important for increasing clinician confidence in diagnosis. Finally, some acoustic measures can be obtained relatively easily in a clinical setting. Although the work discussed in this article is still in rather preliminary stages of investigation, the following studies have shown promise in identifying acoustic measures that can be adapted to clinical practice.

Fine-Grained Acoustic Analysis

Subtle articulatory-distortion errors often go undetected when evaluated perceptually, and due to categorical perception, distortion errors may be falsely attributed to phonemic error.[31] In addition to the clinical challenges this poses, difficulties distinguishing between errors that originate at the level of motor planning from those that are phonological in nature have led to debates surrounding theoretical accounts of AOS. This issue has been investigated through assessment of voice onset time (VOT)[24] and analysis of deletion errors that occur in consonant clusters.[32–34] Collectively, these studies have approached acoustic analysis with the premise that measures of articulatory timing can inform whether perceived substitution and omission errors are subtle articulatory distortions, or if they originate at higher levels of production—at the level of phonological encoding.[24,32–34] For example, VOT studies have shown that individuals with AOS produce VOTs that are outside of normal range (relative to control speakers), but within the range of the target phoneme.[24] In contrast, those with phonemic paraphasias and no AOS produce VOTs within the range of the voiceless cognate, [24] suggesting that perceived phonemic errors are indeed substitution errors for those with predominate phonemic paraphasias.

With regard to omissions, Buchwald et al showed that differences in articulatory timing of singleton productions resulting from/s/omissions in/s/-initial clusters reflects whether the error originated at a phonological or motor planning level, based on the fact that the duration of individual segments in a cluster is shorter than the duration of these same segments as singletons.[32–34] That is, if the omission error was phonologically driven, production of the following consonant should reflect a singleton production (e.g., *small* → *mall*, duration of/m/should not differ between cluster reductions or singleton onset productions). If the timing was driven by an error in motor planning, analysis of the cluster reduction production should reveal some timing parameters that reflect the initial cluster onset (e.g., *small* → *mall*, duration of/m/will be shorter in the omitted cluster attempt when compared with singleton onset production). Although Buchwald and Miozzo found evidence of both types of errors in two individuals with AOS and concomitant aphasia, the individual with more severe AOS demonstrated more frequent cluster reduction errors reflective of a motor planning impairment.[32] Moreover, in a later study, participants who demonstrated more phonetic-level omission errors showed greater gains in a production treatment that incorporated principles of motor learning.[34–36] These studies show that measures reflective of motor production can guide treatment planning (i.e., a phonological versus motor treatment approach) and may also be used to predict the individuals that are likely to show greater gains in a motor-based treatment.

Pairwise Variability Index

The pairwise variability index (PVI) is a coefficient that reflects differences in the duration of successive consonant (PVI-C) or vowel (PVI-V) segments. Both PVI-V and PVI-C have been investigated in studies comparing cross-linguistic and dialect differences in speech rhythm and in the dysarthria literature.[37–39] However, only PVI-V has been investigated in AOS (e.g., poststroke AOS and PAOS caused by a neurodegenerative process).[12,40–44]

PVI-V is calculated by obtaining vowel durations from the spectral signal (e.g., in Praat[45]), where vocalic segments are identified based on specific criteria across multisyllabic words,[40–44,46] phrases,[39,47] or connected speech.[12,48] When vowel durations in words (or phrases) are similar, PVI-V coefficients are small, and reduced PVI-V coefficients are arguably reflective of the excess and equal stress that characterizes apraxic speech.[40–44] In typical speech production, vowel durations are more variable, thus resulting in comparatively higher PVI coefficients.

In the AOS literature, studies that have investigated a rate normalized PVI-V (nPVI-V) coefficient, calculated across the first two syllables of multisyllabic words with a weak-strong stress pattern (e.g., potato), have shown that speakers with AOS have significantly reduced nPVI-V coefficients when compared with those with aphasia, but without AOS (e.g., in post-stroke AOS;[40,41] see also Duffy et al[42,43] and Ballard et al[44] for PAOS). In a large sample of participants (n = 72 total individuals, n = 35 with AOS), Ballard et al found that nPVI-V coefficients obtained from as few as five three-syllable words with weak-strong stress patterns, along with a measure of sound errors (obtained from the “words of increasing length” subtest of the Apraxia Battery for Adults-2[30]) provided a clinical classification with > 90% positive predictive value and > 80% negative predictive value

relative to expert diagnosis as the standard for comparison.[40] According to Ballard et al,[40] obtaining nPVI-V measures for a small number of tokens is feasible in a clinical setting.‡ This is important, considering that assessments in acute stroke must consider a wide range of speech, language, and swallowing functions, meaning speech-language pathologists are often limited to brief evaluations to establish diagnosis and plan treatment. Establishing a quick and effective diagnostic measure can have important implications for speech evaluations in acute stroke.

Syllable Duration

Evidence from PAOS and aphasia suggests that the rate and duration of word and sentence productions may also be valuable in diagnosis.[42] Duffy and colleagues showed that word duration for the word *catastrophe* (mean duration after three repetitions) had a high sensitivity (0.90) and specificity (0.91) for PAOS diagnosis. Rate of sentence production also had high predictive value, but sentence production was more difficult for some participants, limiting its clinical utility. Like Ballard et al,[40] this study also showed that clinical classification via acoustic analysis can be based on a small sample of tokens (e.g., three repetitions of the word *catastrophe*, production of two sentences). Future research should consider the extent that measures of syllable duration may also be clinically relevant for poststroke AOS, as well as if syllable duration provides any nonredundant information when compared with PVI.

Envelope Modulation Spectrum

In the speech signal, subtle fluctuations in the amplitude envelope (the intensity contour of a speech signal) correspond to different aspects of production. Slower, rhythmic modulations correspond to speech prosody (i.e., 1 to 2 Hz) and syllabic structure (i.e., 4 to 8 Hz), and faster modulations correspond to rapid articulatory movements (i.e., above 16 Hz).[49–51] Amplitude modulation at 4 Hz may also provide a measure of intelligibility.[52] The amplitude envelope modulation spectrum quantifies these fluctuations, and variables obtained from the envelope modulation spectrum have been explored for their utility in differential diagnosis of the dysarthrias and AOS.[12,52] Specifically, Liss et al found that several variables related to speech rhythm distinguished speakers with dysarthria from typical speakers (95.3% accurate with cross-validation), and that these measures distinguished among the dysarthria types with 84% accuracy (67.4% with cross-validation). In the AOS literature, Basilakos and colleagues found that greater amplitude energy at lower frequencies associated with prosody (1 to 2 Hz) and higher frequencies associated with consonant production (16 to 32 Hz) weighed heavily in clinical classification, with each measure respectively yielding 75% and 80% cross-validated accuracy in discriminating AOS from a group of speakers without AOS, but with aphasia.[12] Moreover, the measure reflective of consonant production (amplitude energy at 16- to 32-Hz modulation) correlated with perceptual evaluations of speech distortions for the individuals with AOS.[12]

The studies reviewed previously are promising with regard to the development of acoustic measures to discriminate between AOS and aphasia. However, there are limited data on how

‡The study by Ballard et al provides a helpful tutorial for calculating nPVI-V from single word productions.[40]

these measures fare in acute post-stroke AOS, as well as when differential diagnosis must also account for the presence of dysarthria. Another potential obstacle for the use of these acoustic analyses is that they may require some amount of specialized training on the part of the clinician. Ongoing efforts to automate these analyses show promise for their clinical implementation in the near future (e.g., see Shahin et al[53]). With further validation at both the acute and chronic stages of stroke, as well as PAOS, these algorithms could become available for more widespread clinical use through applications or other technology.

USING NEUROIMAGING TO GUIDE CLINICAL DECISION MAKING

As a result of advances in neuroimaging technology, the utility of structural and functional magnetic resonance imaging has been investigated to determine the extent that neuroimaging data can inform clinical decision-making. These studies have focused on identifying patterns of brain damage predictive of speech and language impairment,[54–57] and whether different patterns of brain damage predict response to various treatment approaches.[58] In the aphasia literature, these studies have identified lesion locations that predict language impairments, in both acute and chronic aphasia. However, AOS has been associated with a variety of different lesion locations, resulting in debates surrounding the crucial site (s) of damage that cause(s) AOS (for discussion, see Hillis et al[59] and Richardson et al[60]). Despite remaining inconsistencies in lesion-deficit studies, possibly related to methodological as well as diagnostic differences, recent neuroimaging work has advanced the study of the neuroanatomical bases of AOS. Findings from structural and functional neuroimaging studies support the notion that AOS is indeed a unique clinical entity dissociable from aphasia, and also inform the study of speech production more generally.[9,22,61,62] Continued efforts to identify patterns of damage that predict apraxic features may facilitate differential diagnosis, prognosis, and potentially shed light on regions that may be targets for brain stimulation therapies.[63]

Imaging Evidence from Acute Stroke

Studies that have focused on infarct locations associated with acute AOS are limited in number. Before discussing these studies, a description of Dronkers' seminal study on AOS is in order.[64] In 1996, Dronkers published a study comparing lesion overlap maps obtained from individuals with poststroke AOS with a second group of individuals that had experienced a stroke, but did not have AOS. Dronkers found that all 25 with AOS had damage to the superior precentral gyrus of the insula (SPGI), whereas the group without AOS did not have damage to the SPGI. Additional work from Dronkers' group (e.g., see Baldo et al[65] and Dronkers et al[66]), and others,[67] lends support to the notion of the insula as the primary cortical location that causes AOS, but not all studies converge on this issue.

Hillis et al[59] argued that Dronkers'[64] results may be driven by methodological flaws in lesion overlap methods (for a discussion, see Rordan and Karnath[68]) and a spurious association between damage to the anterior insula and apraxic impairment,[69] better explained by the fact that the insula is commonly damaged in large left hemisphere strokes.[70,71] Hillis et al also argued that lesion-deficit studies should investigate individuals acutely, prior to recovery and possible reorganization of responsible brain regions.[59] To do so, Hillis

et al studied a group of 80 patients within 24 hours of stroke, identifying 31 with AOS and 49 without. Of those with AOS, over half (n = 19) did not have damage (measured as infarct or hypoperfusion; i.e., decreased blood flow to a given region) to the SPGI. The majority (n = 26) had damage to Broca area. A remaining five participants did not have damage to either region; rather, the pre- and postcentral gyri were affected.

The results from Hillis et al's study suggest that damage to Broca area, as well as to the pre/postcentral gyri, are associated with AOS in the acute stage. This has been supported by other acute stroke studies using voxel-based group analyses (e.g., see Itabashi et al[61] and Hickok et al[72]), a lesion overlap study of individuals with "pure AOS" (n = 5) and "equivocal aphasia" (n = 2),[9] and single case studies of pure AOS.[73–75]

Imaging Evidence from Chronic Stroke

Broca area,[65] the pre- and postcentral gyri, and the supramarginal gyrus have been implicated in chronic AOS.[10,22,62] Studies that controlled for aphasic errors either through multivariate analysis methods or through selection of individual cases showed that damage to these areas produces speech production deficits that can be dissociated from the linguistic deficits that occur in aphasia.[9,10,22,75] This suggests that when AOS results from lesions to these areas, individuals may be less likely to resolve over time, resulting in chronic impairment. No published study has evaluated this hypothesis with longitudinal data; further research is needed to identify structures and connections less amenable to early recovery of speech planning and programming processes.

Apraxia of Speech as a Network Impairment?

The studies reviewed previously provide important information pertaining to regional damage that predicts AOS, even though identifying a one-to-one correspondence between a specific infarct location and deficits associated with AOS has remained elusive. Neuroimaging methods that allow for the inspection of regional brain connections have been used to show that compromise to networks of interconnected regions may better explain poststroke deficits.[76] Moreover, the integrity of these networks may influence recovery—spontaneous or treated (e.g., Bonilha et al[77]). It is widely accepted that the brain works as an interconnected network of regions, meaning that damage to one anatomical area can (and typically does) affect processing in regions both structurally and functionally connected to the area of infarct.[77,78] Functional and structural connectome measures have been developed using data acquired from resting state functional magnetic resonance imaging (functional connectome) and diffusion tensor imaging (structural connectome), both of which have been applied to the study of communication deficits in stroke survivors.[54,55,62,77–81]

To date, only one published study has directly evaluated network connectivity in speakers with AOS. This study by New and colleagues found that when compared with a group of stroke survivors without AOS (n = 17), those with AOS (n = 15) displayed (1) functional connections between the left and right premotor cortices that were reduced in strength, and (2) an inverse relationship between left premotor cortex and right anterior insula, where decreased activity in the left premotor cortex was associated with increased activity in the right anterior insula.[62] The reduced connections between the premotor cortices were

correlated with expert-rated AOS severity, indicating that damage to the left premotor region can affect how both hemispheres support speech motor planning and programming. New et al's study emphasizes that connectivity measures are vital to understanding how neural networks are affected in AOS, giving rise to speech impairment.[62]

Specific to the acute phase, future studies should consider how functional connectivity changes through spontaneous and treated recovery, as well as the role of perilesional regions and intra- and interhemispheric connections in plasticity.[82] Based on the studies reviewed here, neuroimaging techniques show promise for informing assessment and prognosis, and with more work, these advances could potentially guide the selection of different treatments.[68,83] At this time, imaging should not be used as the sole means of diagnosis for any communication impairment. However, preliminary work suggests that using sensitive and specific measures, together with diagnostic imaging, may be useful to support diagnosis.[22,55] More research, specifically focusing on the implementation of these methods into everyday clinical practice, is necessary to confirm the utility of neuroimaging to guide clinical decision making in AOS.[63]

EMERGING TRENDS IN TREATMENT

Although treatment was not the specific focus of this review, a recent systematic review suggests AOS treatment is effective.[84] The articulatory-kinematic approach is supported by a considerable evidence base that demonstrates its efficacy for improving speech production in AOS, and there is modest support for the rate/rhythm approach. Other approaches have been identified, including compensatory use of augmentative and alternative communication devices, and intersystemic reorganization, such as pairing speech production with gestures. In addition to these approaches, innovative treatment methods that have been studied in aphasia are now being considered for the management of AOS. Two such methods include the use of noninvasive transcranial direct current stimulation (tDCS) and self-administered computer-based therapy.[85–88]

Transcranial Direct Current Stimulation

In two double-blind studies, Marangolo et al showed that using tDCS as an adjuvant to speech production therapy increased production accuracy, beyond the gains made from the therapy with sham stimulation.[85,87] However, both studies were limited in sample size (three and eight individuals) and gains varied across participants. Moreover, these studies tested individuals with chronic AOS. There are no published studies that have investigated the use of tDCS in early phases of AOS recovery, though the few that have considered tDCS for early stages of stroke recovery suggest that its use is promising for improving plasticity at the subacute stage.[89,90]

Self-Administered Computer Treatment

The ubiquitous use of smartphones and tablet devices has resulted in the development of applications that patients can download and use in addition to (or following discharge from) speech-language rehabilitation services. There is some positive evidence for the use of computerized treatment to improve naming and repetition in individuals with AOS and

concomitant aphasia. Using a randomized controlled trial that included 50 participants, Varley et al showed gains in naming and repetition that were directly proportional to the amount of time participants used the program.[88] This study provides support for the use of computerized treatment for AOS; however, at this time, there are no studies that have evaluated whether research-backed approaches for AOS (e.g., articulatory/kinematic) can be successfully delivered via computer or tablet. Further, no studies have assessed the efficacy of computerized treatment for AOS at the acute or subacute stage, when stamina for treatment may be complicated by other factors. Nevertheless, using commercially available apps to deliver speech-language therapy may be a feasible supplement to traditional speech therapy in early phases of recovery.[91]

CONCLUSION

The premise of this article is that reliable and objective evaluation of AOS is crucial for its management. Treatment success hinges on an accurate diagnosis and evaluation of the speech production impairment(s) with which an individual presents. Clinically, adopting objective methods for diagnosis is crucial for the following reasons: first, after an individual experiences a stroke, communication may be impaired for several reasons aside from AOS. The few studies investigating acute AOS report mutism in the early stages, which is a limitation to the assessment of communicative function. Though these studies report such symptoms resolve over time, and patients ultimately present with characteristics of AOS,[6,67] the use of neuroimaging in such complex cases may shed light on the types of impairments a patient may experience as production deficits evolve. Second, using acoustic measures during initial assessments can provide baseline measures with which to track response to AOS treatment, which is especially important considering that perceptual evaluation of speech can become biased as a therapist becomes familiar with a patient's speech.

Theoretically, the acoustic speech analysis and neuroimaging techniques discussed here have the potential to advance the study of AOS not only by improving differential diagnosis, but also by providing more objective means to compare participants across studies. Though neuroimaging data may not always be available in research studies, reporting acoustic measures, in addition to other diagnostic tests, could provide a quantitative means by which to compare participants across studies from different research groups. Of course, more normative information regarding these acoustic measures is required first.

In sum, many new developments in the management of AOS are aimed at increasing reliability of objective assessment and quantification of motor speech impairments. Unfortunately, there is a lack of research investigating AOS in acute stroke, as well as its resolution longitudinally. Future studies should consider applying methods such as those reviewed here to advance our understanding of AOS in acute stroke, as well as its progression into the chronic stage of recovery.

Acknowledgments

The author would like to extend gratitude to Dr. Julius Fridriksson and Dr. Dirk den Ouden for comments on a prior version of this manuscript. Supported by a grant from the National Institutes of Health-National Institute of

Deafness and Other Communication Disorders (NIH/NIDCD) through award T32 DC014435. The content is solely the responsibility of the author and does not necessarily represent the views of the NIH.

References

1. Hope TM, Seghier ML, Leff AP, Price CJ. Predicting outcome and recovery after stroke with lesions extracted from MRI images. Neuroimage Clin. 2013; 2:424–433. [PubMed: 24179796]
2. Flowers HL, Skoretz SA, Silver FL, et al. Poststroke aphasia frequency, recovery, and outcomes: a systematic review and meta-analysis. Arch Phys Med Rehabil. 2016; 97(12):2188–2201.e8. [PubMed: 27063364]
3. Duffy, J. Motor Speech Disorders: Substrates, Differential Diagnosis and Management. St. Louis, MO: Mosby; 2005.
4. Duffy JR. Apraxia of speech in degenerative neurologic disease. Aphasiology. 2006; 20(06):511–527.
5. Duffy JR, Josephs KA. The diagnosis and under-standing of apraxia of speech: why including neurodegenerative etiologies may be important. J Speech Lang Hear Res. 2012; 55(05):S1518–S1522. [PubMed: 23033445]
6. Mauszycki S, Wambaugh J, Wright S. A sub-acute case of resolving acquired apraxia of speech and aphasia. Int J Phys Med Rehabil. 2014; 2:188–194.
7. American Speech-Language-Hearing Association. Acquired Apraxia of Speech. Practice Portal. Available at: http://www.asha.org/Practice-Portal/Clinical-Topics/Acquired-Apraxia-of-Speech/. Accessed September 9, 2017
8. 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. New York, NY: Thieme; 1997. p. 1-25.
9. Graff-Radford J, Jones DT, Strand EA, Rabinstein AA, Duffy JR, Josephs KA. The neuroanatomy of pure apraxia of speech in stroke. Brain Lang. 2014; 129:43–46. [PubMed: 24556336]
10. Moser D, Basilakos A, Fillmore P, Fridriksson J. Brain damage associated with apraxia of speech: evidence from case studies. Neurocase. 2016; 22(04):346–356. [PubMed: 27264534]
11. Strand EA, Duffy JR, Clark HM, Josephs K. The apraxia of speech rating scale: a tool for diagnosis and description of apraxia of speech. J Commun Disord. 2014; 51:43–50. [PubMed: 25092638]
12. Basilakos A, Yourganov G, den Ouden DB, et al. A multivariate analytic approach to the differential diagnosis of apraxia of speech. J Speech Lang Hear Res. 2017; 60(12)doi: 10.1044/2017_JSLHR-S-16-0443
13. Cunningham KT, Haley KL, Jacks A. Speech sound distortions in aphasia and apraxia of speech: reliability and diagnostic significance. Aphasiology. 2016; 304(04):396–413.
14. Haley KL, Jacks A, Cunningham KT. Error variability and the differentiation between apraxia of speech and aphasia with phonemic paraphasia. J Speech Lang Hear Res. 2013; 56(03):891–905. [PubMed: 23275417]
15. Miller N. Variability in speech dyspraxia. Clin Linguist Phon. 1992; 6(1–2):77–85. [PubMed: 20672885]
16. Whiteside SP, Dyson L, Cowell PE, Varley RA. The relationship between apraxia of speech and oral apraxia: association or dissociation? Arch Clin Neuropsychol. 2015; 30(07):670–682. [PubMed: 26275812]
17. Croot K. Diagnosis of AOS: definition and criteria. Semin Speech Lang. 2002; 23(04):267–280. [PubMed: 12461726]
18. Darley, F. The classification of output disturbances in neurogenic communication disorders. Paper presented at: American Speech and Hearing Association Annual Conference; Chicago, IL. 1969.
19. McNeil, MR., Pratt, SR., Fossett, TRD. The differential diagnosis of apraxia of speech. In: Maassen, B.Kent, R.Peters, H.van Lieshout, P., Hulstijn, W., editors. Speech Motor Control in Normal and Disordered Speech. Oxford, UK: Oxford University Press; 2004. p. 389-41.

20. McNeil, MR., Robin, DA., Schmidt, RA. Apraxia of speech: definition, differentiation, and treatment. In: McNeil, MR., editor. Clinical Management of Sensorimotor Speech Disorders. New York, NY: Thieme; 1997. p. 311-344.
21. Ogar J, Slama H, Dronkers N, Amici S, Gorno-Tempini ML. Apraxia of speech: an overview. Neurocase. 2005; 11(06):427–432. [PubMed: 16393756]
22. Basilakos A, Rorden C, Bonilha L, Moser D, Fridriksson J. Patterns of poststroke brain damage that predict speech production errors in apraxia of speech and aphasia dissociate. Stroke. 2015; 46(06):1561–1566. [PubMed: 25908457]
23. Duffy, JR., Gawle, CA. Apraxic speakers' vowel duration in consonant-vowel-consonant syllables. In: Rosenbek, J.McNeil, MR., Aronson, AE., editors. Apraxia of Speech: Physiology, Acoustics, Linguistics, Management. San Diego, CA: College-Hill Press; 1984. p. 168-196.
24. Itoh M, Sasanuma S, Tatsumi IF, Murakami S, Fukusako Y, Suzuki T. Voice onset time characteristics in apraxia of speech. Brain Lang. 1982; 17(02):193–210. [PubMed: 7159832]
25. Itoh M, Sasanuma S, Ushijima T. Velar movements during speech in a patient with apraxia of speech. Brain Lang. 1979; 7(02):227–239. [PubMed: 466393]
26. Robin DA, Bean C, Folkins JW. Lip movement in apraxia of speech. J Speech Hear Res. 1989; 32(03):512–523. [PubMed: 2779196]
27. Shankweiler D, Harris KS. An experimental approach to the problem of articulation in aphasia. Cortex. 1966; 2(03):277–292.
28. Rosenbek, J., Kent, R., Lapointe, L. Apraxia of speech: an overview and some perspectives. In: Rosenbek, JC.McNeil, MR., Aronson, AE., editors. Apraxia of Speech: Physiology, Acoustics, Linguistics, Management. San Diego, CA: College-Hill Press; 1984. p. 1-72.
29. Dabul, BL. Apraxia Battery for Adults. 2nd. Dallas, TX: Pro-Ed; 2000.
30. Dabul, BL. Apraxia Battery for Adults. Tigard, OR: C.C. Publications; p. 1979
31. Fromm, D., Abbs, JH., McNeil, MR., Rosenbek, JC. Simultaneous perceptual-physiological method for studying apraxia of speech. In: Brookshire, R., editor. Clinical Aphasiology. Minneapolis, MN: BRK Publications; 1982. p. 65-80.
32. Buchwald A, Miozzo M. Phonological and motor errors in individuals with acquired sound production impairment. J Speech Lang Hear Res. 2012; 55(05):S1573–S1586. [PubMed: 23033450]
33. Buchwald A, Miozzo M. Finding levels of abstraction in speech production: evidence from sound-production impairment. Psychol Sci. 2011; 22(09):1113–1119. [PubMed: 21828349]
34. Buchwald A, Gagnon B, Miozzo M. Identification and remediation of phonolgical and motor errors in acquired sound production impairment. J Speech Lang Hear Res. 2017; 60:1726–1738. DOI: 10.44/2017_JSLHR-S-16-0240 [PubMed: 28655044]
35. Maas E, Robin DA, Wright DL, Ballard KJ. Motor programming in apraxia of speech. Brain Lang. 2008; 106(02):107–118. [PubMed: 18417200]
36. Bislick LP, Weir PC, Spencer K, Kendall D, Yorkston KM. Do principles of motor learning enhance retention and transfer of speech skills? A systematic review. Aphasiology. 2012; 26(05): 709–728.
37. Arvaniti A. The usefulness of metrics in the quantification of speech rhythm. J Phonetics. 2012; 40(03):351–373.
38. Ling LE, Grabe E, Nolan F. Quantitative characterizations of speech rhythm: syllable-timing in Singapore English. Lang Speech. 2000; 43(Pt 4):377–401. [PubMed: 11419223]
39. Liss JM, White L, Mattys SL, et al. Quantifying speech rhythm abnormalities in the dysarthrias. J Speech Lang Hear Res. 2009; 52(05):1334–1352. [PubMed: 19717656]
40. Ballard KJ, Azizi L, Duffy JR, et al. A predictive model for diagnosing stroke-related apraxia of speech. Neuropsychologia. 2016; 81:129–139. [PubMed: 26707715]
41. Vergis MK, Ballard KJ, Duffy JR, McNeil MR, Scholl D, Layfield C. An acoustic measure of lexical stress differentiates aphasia and aphasia plus apraxia of speech after stroke. Aphasiology. 2014; 28(05):554–575.

42. Duffy JR, Hanley H, Utianski R, et al. Temporal acoustic measures distinguish primary progressive apraxia of speech from primary progressive aphasia. Brain Lang. 2017; 168:84–94. [PubMed: 28187331]
43. Duffy JR, Strand EA, Clark H, Machulda M, Whitwell JL, Josephs KA. Primary progressive apraxia of speech: clinical features and acoustic and neurologic correlates. Am J Speech Lang Pathol. 2015; 24(02):88–100. [PubMed: 25654422]
44. Ballard KJ, Savage S, Leyton CE, Vogel AP, Hornberger M, Hodges JR. Logopenic and non-fluent variants of primary progressive aphasia are differentiated by acoustic measures of speech production. PLoS One. 2014; 9(02):e89864. [PubMed: 24587083]
45. Boersma, P., Weenink, D. Praat, a system for doing phonetics by computer. 2015. [computer program, version 5.4.17]. Available at: http://www.praat.org. Accessed November 15, 2017
46. Peterson GE, Lehiste I. Duration of syllable nuclei in English. J Acoust Soc Am. 1960; 32(06): 693–703.
47. White L, Mattys SL. Calibrating rhythm: first language and second language studies. J Phonetics. 2007; 35(04):501–522.
48. Thomas ER, Carter PM. Prosodic rhythm and African American English. Engl World-Wide. 2006; 27(03):331–355.
49. Ghitza O, Greenberg S. On the possible role of brain rhythms in speech perception: intelligibility of time-compressed speech with periodic and aperiodic insertions of silence. Phonetica. 2009; 66(1–2):113–126. [PubMed: 19390234]
50. MacNeilage PF. The frame/content theory of evolution of speech production. Behav Brain Sci. 1998; 21(04):499–511. discussion 511–546. [PubMed: 10097020]
51. Giraud A-L, Kleinschmidt A, Poeppel D, Lund TE, Frackowiak RS, Laufs H. Endogenous cortical rhythms determine cerebral specialization for speech perception and production. Neuron. 2007; 56(06):1127–1134. [PubMed: 18093532]
52. Liss JM, LeGendre S, Lotto AJ. Discriminating dysarthria type from envelope modulation spectra. J Speech Lang Hear Res. 2010; 53(05):1246–1255. [PubMed: 20643800]
53. Shahin, MA., Ahmed, B., Ballard, KJ. Automatic classification of unequal lexical stress patterns using machine learning algorithms. Paper presented at: Spoken Language Technology Workshop (SLT); Miami, FL. 2012. IEEE2012
54. Bates E, Wilson SM, Saygin AP, et al. Voxel-based lesion-symptom mapping. Nat Neurosci. 2003; 6(05):448–450. [PubMed: 12704393]
55. Yourganov G, Smith KG, Fridriksson J, Rorden C. Predicting aphasia type from brain damage measured with structural MRI. Cortex. 2015; 73:203–215. [PubMed: 26465238]
56. Price CJ, Seghier ML, Leff AP. Predicting language outcome and recovery after stroke: the PLORAS system. Nat Rev Neurol. 2010; 6(04):202–210. [PubMed: 20212513]
57. Hillis AE, Wityk RJ, Tuffiash E, et al. Hypoperfusion of Wernicke's area predicts severity of semantic deficit in acute stroke. Ann Neurol. 2001; 50(05):561–566. [PubMed: 11706960]
58. Fridriksson J, Morrow-Odom L, Moser D, Fridriksson A, Baylis G. Neural recruitment associated with anomia treatment in aphasia. Neuroimage. 2006; 32(03):1403–1412. [PubMed: 16766207]
59. Hillis AE, Work M, Barker PB, Jacobs MA, Breese EL, Maurer K. Re-examining the brain regions crucial for orchestrating speech articulation. Brain. 2004; 127(Pt 7):1479–1487. [PubMed: 15090478]
60. Richardson JD, Fillmore P, Rorden C, Lapointe LL, Fridriksson J. Re-establishing Broca's initial findings. Brain Lang. 2012; 123(02):125–130. [PubMed: 23058844]
61. Itabashi R, Nishio Y, Kataoka Y, et al. Damage to the left precentral gyrus is associated with apraxia of speech in acute stroke. Stroke. 2016; 47(01):31–36. [PubMed: 26645260]
62. New AB, Robin DA, Parkinson AL, et al. Altered resting-state network connectivity in stroke patients with and without apraxia of speech. Neuroimage Clin. 2015; 8:429–439. [PubMed: 26106568]
63. Ballard KJ, Tourville JA, Robin DA. Behavioral, computational, and neuroimaging studies of acquired apraxia of speech. Front Hum Neurosci. 2014; 8:892. [PubMed: 25404911]

64. Dronkers NF. A new brain region for coordinating speech articulation. Nature. 1996; 384(6605): 159–161. [PubMed: 8906789]
65. Baldo JV, Wilkins DP, Ogar J, Willock S, Dronkers NF. Role of the precentral gyrus of the insula in complex articulation. Cortex. 2011; 47(07):800–807. [PubMed: 20691968]
66. Dronkers N, Ogar J, Willock S, Wilkins DP. Confirming the role of the insula in coordinating complex but not simple articulatory movements. Brain Lang. 2004; 91(01):23–24.
67. Nagao M, Takeda K, Komori T, Isozaki E, Hirai S. Apraxia of speech associated with an infarct in the precentral gyrus of the insula. Neuroradiology. 1999; 41(05):356–357. [PubMed: 10379594]
68. Rorden C, Karnath HO. Using human brain lesions to infer function: a relic from a past era in the fMRI age? Nat Rev Neurosci. 2004; 5(10):813–819. [PubMed: 15378041]
69. Trupe LA, Varma DD, Gomez Y, et al. Chronic apraxia of speech and Broca's area. Stroke. 2013; 44(03):740–744. [PubMed: 23362082]
70. Finley A, Saver J, Alger J, et al. Diffusion weighted imaging assessment of insular vulnerability in acute middle cerebral artery infarctions. Stroke. 2003; 34(01):259–259.
71. Kodumuri N, Sebastian R, Davis C, et al. The association of insular stroke with lesion volume. Neuroimage Clin. 2016; 11:41–45. [PubMed: 26909326]
72. Hickok G, Rogalsky C, Chen R, Herskovits EH, Townsley S, Hillis AE. Partially overlapping sensorimotor networks underlie speech praxis and verbal short-term memory: evidence from apraxia of speech following acute stroke. Front Hum Neurosci. 2014; 8:649. [PubMed: 25202255]
73. Fox RJ, Kasner SE, Chatterjee A, Chalela JA. Aphemia: an isolated disorder of articulation. Clin Neurol Neurosurg. 2001; 103(02):123–126. [PubMed: 11516558]
74. Kasahata N. Speech disturbances due to left precentral cortical lesions. Neurocase. 2014; 20(03): 328–337. [PubMed: 23548114]
75. Ojha PK, Nandavar S, Pearson DM, Demchuk AM. Aphemia as a presenting symptom in acute stroke. Neurol India. 2011; 59(03):432–434. [PubMed: 21743177]
76. Carey LM, Seitz RJ, Parsons M, et al. Beyond the lesion: neuroimaging foundations for post-stroke recovery. Future Neurol. 2013; 8(05):507–527.
77. Bonilha L, Nesland T, Rorden C, Fillmore P, Ratnayake RP, Fridriksson J. Mapping remote subcortical ramifications of injury after ischemic strokes. Behav Neurol. 2014; 2014:215380. [PubMed: 24868120]
78. Bonilha L, Rorden C, Fridriksson J. Assessing the clinical effect of residual cortical disconnection after ischemic strokes. Stroke. 2014; 45(04):988–993. [PubMed: 24619391]
79. Yourganov G, Fridriksson J, Rorden C, Gleichgerrcht E, Bonilha L. Multivariate connectome-based symptom mapping in post-stroke patients: networks supporting language and speech. J Neurosci. 2016; 36(25):6668–6679. [PubMed: 27335399]
80. Corbetta M, Ramsey L, Callejas A, et al. Common behavioral clusters and subcortical anatomy in stroke. Neuron. 2015; 85(05):927–941. [PubMed: 25741721]
81. Ivanova MV, Isaev DY, Dragoy OV, et al. Diffusion-tensor imaging of major white matter tracts and their role in language processing in aphasia. Cortex. 2016; 85:165–181. [PubMed: 27289586]
82. Fridriksson J. Preservation and modulation of specific left hemisphere regions is vital for treated recovery from anomia in stroke. J Neurosci. 2010; 30(35):11558–11564. [PubMed: 20810877]
83. Fridriksson J, Bonilha L, Baker JM, Moser D, Rorden C. Activity in preserved left hemisphere regions predicts anomia severity in aphasia. Cereb Cortex. 2010; 20(05):1013–1019. [PubMed: 19687294]
84. Ballard KJ, Wambaugh JL, Duffy JR, et al. Treatment for acquired apraxia of speech: a systematic review of intervention research between 2004 and 2012. Am J Speech Lang Pathol. 2015; 24(02): 316–337. [PubMed: 25815778]
85. Marangolo P, Fiori V, Cipollari S, et al. Bihemispheric stimulation over left and right inferior frontal region enhances recovery from apraxia of speech in chronic aphasia. Eur J Neurosci. 2013; 38(09):3370–3377. [PubMed: 23930827]
86. Marangolo P, Fiori V, Sabatini U, et al. Bilateral transcranial direct current stimulation language treatment enhances functional connectivity in the left hemisphere: preliminary data from aphasia. J Cogn Neurosci. 2016; 28(05):724–738. [PubMed: 26807842]

87. Marangolo P, Marinelli CV, Bonifazi S, et al. Electrical stimulation over the left inferior frontal gyrus (IFG) determines long-term effects in the recovery of speech apraxia in three chronic aphasics. Behav Brain Res. 2011; 225(02):498–504. [PubMed: 21856336]

88. Varley R, Cowell PE, Dyson L, Inglis L, Roper A, Whiteside SP. Self-administered computer therapy for apraxia of speech: two-period randomized control trial with crossover. Stroke. 2016; 47(03):822–828. [PubMed: 26797664]

89. Polanowska KE, Leśniak MM, Seniów JB, Czepiel W, Członkowska A. Anodal transcranial direct current stimulation in early rehabilitation of patients with post-stroke non-fluent aphasia: a randomized, double-blind, sham-controlled pilot study. Restor Neurol Neurosci. 2013; 31(06): 761–771. [PubMed: 24047756]

90. You DS, Kim D-Y, Chun MH, Jung SE, Park SJ. Cathodal transcranial direct current stimulation of the right Wernicke's area improves comprehension in subacute stroke patients. Brain Lang. 2011; 119(01):1–5. [PubMed: 21641021]

91. Mallet KH, Shamloul RM, Corbett D, et al. Recover Now: feasibility of a mobile tablet-based rehabilitation intervention to treat post-stroke communication deficits in the acute care setting. PLoS One. 2016; 11(12):e0167950. [PubMed: 28002479]

Learning Outcomes

As a result of this activity, the reader will be able to (1) describe shared and unique speech production deficits in apraxia of speech (AOS), aphasia, and dysarthria; (2) identify brain regions that have been implicated in AOS; and (3) discuss how acoustic measures can improve the differential diagnosis of AOS from aphasia.