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A Critical Review of Apraxia of Speech and Childhood Apraxia of Speech

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Abstract

The theories and neural bases of Apraxia of Speech (AOS) have long been debated. In 1861, Paul Broca identified two patients with speech/language impairments who became the basis for the theory on aphemia, now known as apraxia. Broca noted of patients with AOS that "there are cases in which the general faculty for language remains unaltered; where the auditory apparatus is intact; where all muscles—including those of speech and articulation—are under voluntary control; and where nevertheless, a cerebral lesion abolishes articulated language" (Broca, 1861/2000). The term "apraxia" was first introduced by Hugo Liepmann in 1908 and was described as "the inability to perform voluntary acts despite preserved muscle strength" (Liepmann, 1908). Darley then coined the term "apraxia of speech" in 1969 (Darley et al., 1969).

The American Speech and Hearing Association (ASHA) defines Apraxia of Speech as a "neurologic speech disorder that reflects an impaired capacity to plan or program sensorimotor commands necessary for directing movements that result in phonetically and prosodically normal speech" (Duffy, 2013). The perceptual characteristics of the disorders are described by ASHA as "(a) phoneme distortions and distorted substitutions or additions (b) reduced overall speech rate (c) syllable segregation with extended intra- and intersegmental durations and (d) equal stress across adjacent syllables" (*Acquired Apraxia of Speech*, n.d.). These characteristics reflect some of the initial clinical features identified by Darley (1969). These symptoms are relatively similar to the current diagnostic criteria of AOS established by (McNeil et al., 2009) except that symptoms are currently described as increased inter and intra segmentation, sound distortions, abnormal prosody, and does not reflect slower rate of speech. McNeil et al. would argue that it is distortions which are the critical diagnostic feature, rather than substitutions and additions.

However, these errors do co-occur as many individuals with AOS or CAS have accompanying phonological impairments.

In 2000, Ballard, Granier, and Robin conducted a critical review of acquired apraxia of speech (AOS) focusing on different theories and supportive research. The review also explored intervention models associated with AOS (Ballard et al., 2000). Since that time, extensive work in AOS has been conducted, existing models of the disorder have been refined, and new models have been proposed. In addition, new information on childhood apraxia of speech (CAS) has emerged and theories related to CAS require critical evaluation. In particular, the relationship between stroke related AOS and CAS is critical to advancing efforts in this area. The purpose of this paper is to update Ballard and colleagues (2000) and expand the information to include CAS. (Ballard et al., 2000) hypothesized that the deficits demonstrated in individuals with apraxia of speech could be due to phonological processing, motor control or both. It is now accepted that AOS is a disorder of motor control. In this paper, the most recent research regarding the theoretical understanding of AOS, as well as neural models will be presented. Treatments for AOS will be reviewed and evaluated for efficacy. This paper covers the history of AOS starting with (Darley, 1975).

Darley (1975) identified the perceptual characteristics of AOS. These characteristics were (a) groping for the correct position to articulate sound (b) consonants distorted more frequently than vowels (c) errors across productions differed (d) errors present caused the word produced to be more articulatorily complex (e) errors approximated the target within one to two features (f) errors represented "anticipation, preservation, and transposition of phonemes" (g) insertion of schwas in consonant clusters and (h) awareness that speech errors are being made (Darley, 1975). These clinical features of the disorder inform the theory of the neural bases of the disorder.

Kent & Rosenbek performed an acoustic analysis on seven participants with a diagnosis of AOS without comorbid symptoms of aphasia. Results of the acoustic analysis showed that individuals with AOS presented with "1. slow speaking rate with prolongations of transitions and steady states as well as intersyllable pauses 2. restricted variation in relative peak intensity across syllables 3. slow and inaccurate movements of the articulators to spatial targets for both consonants and vowels" (Kent & Rosenbek, 1983). Kent & Rosenbeck also identified that individuals with AOS had an increased incidence of all types of perceptual errors the more the syllabic or phonetic complexity increased.

Beginning in 1984 Robin began to experimentally study AOS in children and adults in addition to systematically conducting treatment trials using principles of motor learning. In a series of studies across over 20 years, Robin studied motor programming in AOS. Following that, Robin began exploration of treatment approaches. Key studies are presented here. Clark & Robin (1998) studied adults with AOS compared to individuals with conduction aphasia and healthy participants (Clark & Robin, 1998). They identified three aspects of motor programming (1) generalized motor program accuracy (GMP), (2) temporal parameterization accuracy, and (3)

amplitude parameterization accuracy in relation to AOS. GMP refers to the relative timing or amplitude of movements. Parameterization refers to the absolute time or amplitude of a movement. Participants were shown a pattern of movements which they needed to replicate by moving their jaw. Responses were analyzed for how closely they approximated the model. What was found was that the participants with AOS varied in the accuracy of the different aspects of motor programming. Two of the participants demonstrated poorer GMP accuracy but normal parameterization accuracy. The other two participants demonstrated normal GMP but impaired parametrization accuracy. Results demonstrated a trading relationship, as one aspect improved the other became was worse. Participants in this study were compared to individuals with conduction aphasia (CA). The CA group did not exhibit motor programming impairments. This study indicates that either GMP or parameterization impairments can result in AOS and that the deficits noted are due to a disorder of motor programming.

Robin, Bean, & Folkins (1989) investigated whether the velocity of the lower lip and the coordination between the upper and lower lip is impaired in individuals with AOS. Robin, Bean, & Folkins analyzed whether the differences in velocity and temporal coordination resulted in the production of accurate or inaccurate words. Peak articulatory velocity was also measured for syllable production in isolation. Investigators determined that there were no significant differences in velocity or temporal coordination that translated to correct or incorrect word production for the AOS population. This led investigators to hypothesize that individuals with AOS may not have difficulty producing movements with high velocity. Therefore, although the rate of speech in individuals with AOS can be slower, the investigators proposed that this was not due to a decrease in velocity of speech movements and more likely due to segmentation of syllables (Robin et al., 1989).

Hageman et al. (1994) investigated oral motor tracking abilities of individuals with AOS in comparison to typical speakers. Both groups were presented with two tasks. The first was to follow a predictable signal presented on the screen through movement of the jaw and lips as well as voicing. The second task was to follow an unpredictable signal in the same manner. This experiment was designed based on the concept that for typical speakers, following a predictable model involves forming an internal representation of that model and playing the internal model out. This allows the participants to only intermittently check in with the predictable model to ensure accuracy of production. Typical speakers also phase lead the model, meaning that they are slightly ahead of the model as it plays out due to the predictability. Conversely, when presented with a nonpredictable model, speakers have to receive feedback from the model and make adjustments online. This does not allow for the creation of an internal representation of the model, and results in their being a lag between the model playing out and the participant approximation.

The hypothesis of this study for individuals with AOS vs. typical controls was that the disorder is in the ability of individuals with AOS to form an internal representation of a model and program the execution of that motor movement. This would translate to participants with AOS not having a lead time with predictable models, but rather a lag time. Conversely, for the nonpredictable model, the participants with AOS were expected to perform similarly to the typical controls. Results supported this hypothesis and demonstrated that individuals with AOS performed more poorly on predictable tasks in comparison to typical controls. With nonpredictable tasks, participants with AOS and controls performed similarly. This indicates that the disorder is associated with a difficulty forming an internal representation of a motor plan, and not in the ability to execute the movement (Hageman et al., 1994).

Robin et al. (2008) investigated the visuomotor tracking abilities of individuals with AOS. Participants were shown a predictable and nonpredictable model on a screen. The individuals then had to match the models through the movement of their jaw and lips. Results showed that individuals with AOS performed more poorly when the model given was predictable. In contrast, when provided with an unpredictable model, individuals with AOS performed as well as healthy controls and individuals with conduction aphasia. Results indicate that the impairment of visuomotor tracking is correlated with speech motor control. Therefore, the study argues that AOS is a motor programming disorder, rather than a disorder of motor execution (Robin et al., 2008).

Robin (1992) wrote an opinion piece on CAS, then referred to as developmental apraxia of speech. For many years some researchers had hypothesized that AOS did not exist. In response, Robin argued (a) apraxia exists, (b) apraxia has a developmental form, (c) given the existence of apraxia there must be apraxia of speech (d) apraxia is motoric not phonological in nature. Specifically, Robin also asserted that AOS is a disorder of motor control, and is an impairment seen beyond speech to other effector systems (e.g. limbs). Robin argued that because AOS is a motor programming disorder, the most effective treatment approach would be one which is based in the principles of motor learning (Robin Donald A., 1992). Schmidt (2005) referred to motor learning as "a set of processes associated with practice or experience leading to relatively permanent changes in the capability for movement" (Schmidt, 2005).

Ballard, Robin & Folkins (2003) discuss the integration of speech and non-speech motor systems. They argue that because the systems are closely related, individuals with AOS should demonstrate deficits of both speech and non-speech movements (K.J. Ballard et al., 2003). This

is in contrast to previous literature, (Ziegler, 2003), who argued that the deficits of speech seen in AOS did not correlate with nonspeech deficits.

A two-stage model of motor programming for speech and non-speech movements was developed by Klapp (2003). The two stages of the model reflect two distinct processes that occur when motor information is sent from the brain to the muscles. The first portion of this process is termed INT. INT is responsible for organizing the internal spatiotemporal structure of an individual unit of movement and loading that unit of movement into a motor buffer (short term memory store). The second process is termed SEQ. This part of the motor programming process is responsible for sequencing the units in the motor buffer into their correct serial order, after the initiation of the movement in question. Because the SEQ process begins as the movement is initiated, it cannot be preprogrammed. In contrast, the INT process of motor programming can be completed before the movement begins. This means that it can be preprogrammed. Klapp found through his two-stage model that the INT process is sensitive to the complexity of the motor units being loaded into the motor buffer. The more complex the unit of movement, the longer the INT process was. In contrast, the SEQ process was not sensitive to the complexity of the unit of movement but was longer when there were more units to be ordered in the motor buffer (Klapp, 2003). Later, Klapp examined motor programming in speech production. His findings suggested that words are programmed and loaded into the motor buffer as single units. The complexity of the units is determined by the number of syllables that the word has (Klapp, 2003).

In 2008 Maas et al. utilized the Klapp model to investigate the underlying deficits associated with AOS for both speech and nonspeech movements. Results showed that the deficit for AOS was the ability to organize the internal spatiotemporal structure of a motor unit and load the unit into a working memory buffer (INT). In contrast, the time to sequence and initiate the

motor movements (SEQ) was comparable to typically speaking controls. These results were shown with both speech and nonspeech movements, indicating that AOS is a centralized motor programming disorder (Maas et al., 2008).

Theron et al. (2009) investigated the effects of speaking a first vs. second language on typical speakers vs. individuals with AOS or phonemic paraphasic (PP) speech errors. All participants had Afrikaans as their first language and English as their second language. Vowel duration, utterance duration, utterance onset duration, and voice onset time (VOT) were measured. Results showed that individuals with AOS and PP disorders were more affected by speaking a second language than typical bilingual speakers. Specifically, the participants with AOS and PP struggled with decreasing duration of vowels, utterances, and utterance onset as their rate of speech in the second language increased. It is hypothesized that the reason for the difficulty in durational adjustment is because speaking a second language places a higher processing load on individuals with AOS or PP. This increased processing load then translates to increased difficulty with motor planning and programming of speech movements (Theron et al., 2009).

In 2018, Ballard et al. investigated the effect of auditory perturbations on individuals with AOS. The purpose of this study was to gain a stronger understanding of the neural compensatory abilities of individuals with AOS. Results showed that when presented with the auditory perturbations, participants with AOS demonstrated typical compensatory abilities. When presented with sustained F1perturbations, individuals with AOS were able to adapt, while the comparison and control groups included in the study who were aphasic or typically speaking and age-matched, respectively, could not. This indicates that because there has been damage to the

motor program of an individual with AOS, their ability to adapt that motor program may be greater than those who do not have motor program impairments (Ballard et al., 2018).

The Directions into Velocities of Articulators (DIVA) model is a neurocomputational model which takes into account theories, data on the acoustic, kinematic, and functional magnetic resonance imaging (fMRI) aspects of speech production to create a comprehensive neural model of speech production. Differential equations are used as a means for understanding the cell activity in the simulations of the DIVA model. Cells in the DIVA simulations are mapped onto the Montreal Neurological Institute (MNI) model, which allows for the comparison of fMRI data to anatomical locations on a standardized brain map. The DIVA model provides insight into both the computational and neurophysiological aspects of speech perception and production (Tourville & Guenther, 2011).

The DIVA model represents both feedforward and feedback models for speech motor control. The feedforward model refers to patterns of articulation which come from motor memory learned through speech production attempts. The feedback model describes a system which regulates and adjusts for differences between speech produced and the intended speech motor program. The inclusion of both of these models in the DIVA model allows for the detection and analysis of speech errors. This ability makes the DIVA model ideal for the representation of speech sounds disorders and analysis of the accompanying brain regions (Tourville & Guenther, 2011).

The Gradient Order DIVA (GODIVA) model allows for the interpretation of both phonology and speech motor control. The model illustrates how the phonological representation is translated to a series of motor movements which are sequenced to produce speech (Golfinopoulos et al., 2010). The GODIVA consists of two loops which go from the cortex to the

basal ganglia, thalamus, and back to the cortex. One of these loops, the motor loop, is the same as the one used in the DIVA model. This loop accounts for the generation of the articulatory movements necessary for speech. These articulatory movements create the motor program for the syllable as it is being played out. The planning loop is where the motor units are stored in the working memory buffer before they are played out. This loop contains information about both the sequence of the motor units and their phonological content (Golfinopoulos et al., 2010).

Miller & Guenther (2020) investigated how the DIVA and GODIVA models could be applied to identify the specific deficit seen with AOS. Miller & Guenther identified that with AOS the deficits may be due to damage to the speech sound map in the left ventral premotor cortex, the phonological content buffer in the left posterior inferior frontal sulcus, and/or the axonal projections between these areas. The speech sound map is responsible for producing articulator movements and the accompanying sensory feedback. Damage to this area would result in impaired articulation accuracy as well as the ability to accurately identify the sensory accuracy of the movements produced in comparison to the target. Damage to this area would explain the difficulty that speakers with AOS have with improving the accuracy of their motor program. This also explains the damage to the feedforward control seen with AOS (i.e. the execution of stored sequences of motor programs for phoneme sequences commonly seen in the speaker's native language). Damage to the phonological content buffer would result in the suprasegmental errors seen in AOS such as the segmentation of syllables and equal lexical stress (Miller & Guenther, 2021).

New et al. (2015) conducted a resting state functional magnetic resonance imaging (rsfMRI) study on individuals with AOS. rsfMRI involves the patient laying in the scanner without performing an activity. Results of this study showed that the connection between the

right and left ventral premotor (PM) cortex is much weaker in individuals with AOS in comparison to healthy controls. When comparing this information to the DIVA model, the damage in AOS would be to the speech sound map. This damage, as stated above, would account for the difficulties with feedforward control as well as interpreting articulatory sensory feedback required to improve motor plans. New et al. (2015) also illustrates how reduced bilateral PM connectivity is negatively correlated with severity of AOS (New et al., 2015).

Civier et al. hypothesized that the neural basis of AOS is rooted in a partial lesion of the anterior portion of the left ventral/lateral precentral sulcus. The hypothesis specifically was that because this region is partially lesioned, the ability to execute motor programs is dampened, resulting in errors and reduced rate of motor program realization. To test this hypothesis, Civier et al. utilized the speech production model GODIVA. Lesions were induced to the left ventral/lateral precentral sulcus. The results of the simulation supported the hypothesis that damage to this region results in prolongation of initial syllables in polysyllabic words. In comparison to resting state fMRI studies of individuals with AOS done previously (New et al., 2015) the brain differences present in the GODIVA model matched those present in the AOS cohort. This supports the hypothesis that the dysprosody present as a perceptual characteristic of AOS is due to damage to the left anterior ventral precentral sulcus (Civier et al., 2021).

While the body literature for AOS and CAS is growing, continued research to support the hypothesis that the two disorders are the same but in different populations must be conducted. Additional literature regarding the neural basis of these disorders would solidify what aspects of the brain differ in these individuals. A literature review of effective treatment approaches as well as further clinical trials to support the efficacy of these approaches would be beneficial to inform best practice when working with this population.

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