Neurocase (2005) **11**, 427–432

ISSN: 1355-4795 print
DOI: 10.1080/13554790500263529

Psychology Press
Taylor & Francis Group

Apraxia of Speech: An overview

JENNIFER OGAR[1,2], HILARY SLAMA[1], NINA DRONKERS[1,2], SERENA AMICI[1] and MARIA LUISA GORNO-TEMPINI[1]

[1]*UCSF Memory and Aging Center, San Francisco, CA, USA*
[2]*VA Northern California Health Care System, Martinez, CA, USA*

Apraxia of speech (AOS) is a motor speech disorder that can occur in the absence of aphasia or dysarthria. AOS has been the subject of some controversy since the disorder was first named and described by Darley and his Mayo Clinic colleagues in the 1960s. A recent revival of interest in AOS is due in part to the fact that it is often the first symptom of neurodegenerative diseases, such as primary progressive aphasia and corticobasal degeneration. This article will provide a brief review of terminology associated with AOS, its clinical hallmarks and neuroanatomical correlates. Current models of motor programming will also be addressed as they relate to AOS and finally, typical treatment strategies used in rehabilitating the articulation and prosody deficits associated with AOS will be summarized.

Introduction

Apraxia of speech (AOS) has emerged as the term to describe a motor speech disorder characterized by an impaired ability to coordinate the sequential, articulatory movements necessary to produce speech sounds (Wertz *et al*., 1984). Confusion in the literature around AOS stems from the fact that terminology associated with this disorder has varied greatly. Also, symptoms associated with AOS often co-occur or overlap with those caused by neuromuscular deficits indicative of the dysarthrias and the linguistic errors associated with aphasia. AOS is, however, a distinct motor speech disorder.

Although vascular lesions are the most common cause of AOS, the disorder may also result from tumors and trauma. Often AOS has also been identified as the first symptom of neurodegenerative diseases such as corticobasal degeneration or non-fluent progressive aphasia (Rosenfield, 1991; Blake *et al*., 2003; Gorno-Tempini *et al*., 2004). In recent years, progressive speech decline has been described as the initial and primary symptom in a number of degenerative cases (Tyrell *et al*., 1991; Broussolle *et al*., 1996; Chapman *et al*., 1997).

In this paper, we will briefly review clinical hallmarks of AOS, the evolution of terminology associated with the disorder and the ongoing controversy regarding lesion sites associated with AOS. We will summarize the cognitive basis of AOS within current models of motor programming and review modern approaches to treatment.

Received 18 March 2005; accepted 17 July 2005

This work was supported by the McBean Family Foundation, The Sandler Foundation, the John Douglas French Alzheimer's Association, the National Institute on Aging (5P01 AG019724–02 and 1 P50 AG-03–006–01), the California Department of Health (DHS 04–35516), the UCSF General Clinical Research Center (M01 RR00079) and the National Institute of Neurological Diseases and Stroke (R01 NS50915).

Address correspondence to Maria Luisa Gorno-Tempini, MD, PhD, UCSF Memory and Aging Center, 350 Parnassus Ave, Suite 706, San Francisco, CA, 94143–1207. E-mail: marilu@memory.ucsf.edu

History of Terminology

Liepmann introduced the general term 'apraxia' and defined it as an inability to perform voluntary acts despite preserved muscle strength (Liepmann, 1908). Liepmann's notation of 'apraxia of the glosso-labio-pharyngeal structures' was refined by Darley, who first coined the term 'apraxia of speech' in the 1960s (Darley, 1969).

The notion of a speech disorder in the presence of preserved language skills and unimpaired muscular function was originally introduced a century earlier by Paul Broca (Broca, 1861). He termed the disorder *aphemia* and observed 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.

Even though Broca distinguished between speech and language disorders early on, it is unclear if his aphemia is synonymous with today's AOS, particularly because his original patients only produced recurrent utterances and not sufficient spontaneous speech to discern specific AOS symptoms.

Marie introduced the term *anarthria* in an attempt to clarify the difference between aphemia and aphasia (Marie, 1906). His term was used to describe a general inability to control complex mechanical movements in speech production. However, Dejerine later redefined this term by classifying 'anarthria' and 'dysarthria' as speech problems caused by muscle weakness, slowness or incoordination (Dejerine,

1914)—a definition that is still accepted today. These symptoms are not characteristic of the articulatory errors apparent in cases of AOS.

'Anarthria' and 'aphemia' are two of many terms that have been adopted historically to describe speech disturbances that we now associate with AOS. In much of the current European literature the terms 'anarthria' and 'AOS' are still used synonymously (Broussolle *et al*., 1996; Silveri *et al*., 2003; Dobato *et al*., 2004); however, in the United States, 'anarthria' is reserved primarily to describe severe cases of dysarthria, in which muscle weakness is the cause of the speech disturbance (Duffy, 1995). In severe cases, speechlessness may result, making it difficult to distinguish from speechlessness due to profound AOS. It should be noted that mutism may ultimately result from either progressive AOS or dysarthria.

In a review of the literature, Duffy notes 23 terms that have been used to label the symptoms of AOS (see Table 1). Though these terms have been used previously, particularly before Darley and his collegues clarified distinctions between dysarthria and AOS (Darley *et al*., 1975), 'AOS' is currently the most widely accepted term used in the speech pathology literature.

Clinical Presentation

Darley first described AOS as "a disorder of motor speech programming manifested primarily by errors of articulation" (Darley and Aronson, 1975). He noted that AOS resulted from "an impaired ability to program the positioning of the speech musculature … and the sequencing of speech musculature" (Darley *et al*., 1975).

With a shift from purely descriptive means of study (e.g., using broad phonetic transcription to describe the errors heard in AOS) to a utilization of more objective measures, particularly acoustic analysis of speech errors, came refined definitions of the disorder, ones that emphasized deficient sequencing and timing features of AOS (Itoh *et al*., 1979; Kent and Rosenbek, 1983; Duffy and Gawle, 1984; Square-Storer and Apeldoorn, 1991). For example, Kent and Rosenbek describe AOS as an impairment of motor speech control, which leads to "errors in sequencing, timing, coordination, initiation and vocal tract shaping" (Kent and Rosenbek, 1983).

Articulatory errors and prosodic abnormalities are hallmarks of AOS. Prosodic deficits, however, are thought to be a secondary effect of poor articulation (e.g., patients may speak in a slow, halting manner because they are anticipating difficulty speaking) (Darley and Aronson, 1975). Patients with AOS may present with any or all of the following salient signs: 1) effortful trial and error groping with attempts at self-correction; 2) persistent dysprosody (abnormal rhythm, stress and intonation); 3) articulatory inconsistency on repeated productions of the same utterance and/or 4) obvious difficulty initiating utterances (Wertz *et al*., 1984). These characteristic deficits have traditionally been elicited in clinical settings with the administration of the Motor Speech Evaluation (MSE), which includes a collection of words, phrases and sentences that are particularly sensitive to AOS (Wertz *et al*., 1984). The only normed and standardized test for AOS, the Apraxia Battery for Adults –2 (ABA-2) (Dabul, 2000), includes an inventory of 15 articulation characteristics of the disorder (see Table 2). Many of these are similar to classic AOS speech behaviors noted by Wertz, and discernable on the MSE, but in some cases the ABA-2 is more specific with regard to the types of articulatory errors that may be perceived by a listener (e.g., phonemic anticipatory errors, perseverative errors, transposition errors, etc.).

The most common AOS errors involve place of articulation, with affricates and fricatives most affected (e.g., a word with affricate sounds such as the 'ch' in 'church' will be more difficult to say for apraxic speakers than a word with bilabial phonemes, such as the 'm' in 'mom'). Errors are more common on consonant clusters, rather than singleton consonants (e.g., 'strict' will be more difficult than 'sit') and patients with AOS are more likely to produce errors when asked to repeat nonsense words, as opposed to meaningful words (Duffy, 1995). Patients with AOS often assign equal stress to each word. Pauses between syllables and words are common, as is an overall slowed rate of speech (Duffy, 1995). A speaker with AOS, when attempting to say 'cushion' produced the following: "Oh, uh, uh chookun, uh, uh, uh, dook, I know what it's called, it's c-u, uh, not it's chookun, no …"

Differential Diagnosis

When diagnosing AOS, it is important to distinguish the disorder from Broca's aphasia, conduction aphasia and dysarthria.

Table 1. Historical terms associated with AOS (Duffy, 1995)

Afferent motor aphasia	Anarthria	Aphemia
Apraxic dysarthria	Articulatory dyspraxia	Ataxic aphasia
Broca's aphasia	Little Broca's aphasia	Cortical dysarthria
Efferent motor aphasia	Expressive aphasia	Oral verbal apraxia
Speech apraxia	Peripheral motor aphasia	Phonemic aphasia
Phonetic disintegration	Primary verbal apraxia	Pure motor aphasia
Secondary verbal apraxia	Sensorimotor impairment	Word muteness
Speech sound muteness	Subcortical motor aphasia	

Table 2. Inventory of articulation characteristics of apraxia from the apraxia Battery for Adults – 2 (Dabul, 2000)

Speech Behavior
1. Exhibits phonemic anticipatory errors (gleen glass for green grass)
2. Exhibits phonemic perseverative errors (pep for pet)
3. Exhibits phonemic transposition errors (Arifca for Africa)
4. Exhibits phonemic voicing errors (ben for pen)
5. Exhibits phonemic vowel errors (moan for man)
6. Exhibits visible/audible searching
7. Exhibits numerous off-target attempts at the word
8. Errors are highly inconsistent
9. Errors increase as phonemic sequence increases
10. Exhibits fewer errors with automatic speech than volitional speech
11. Exhibits marked difficulty initiating speech
12. Intrudes schwa sound /IPL/ between syllables or in consonant clusters
13. Exhibits abnormal prosodic features
14. Exhibits awareness of errors and inability to correct them
15. Exhibits expressive-receptive gap

The term 'apraxia of speech' has occasionally been used synonymously with Broca's aphasia. The misconception that the two disorders are one and the same may have arisen from the fact that AOS and Broca's aphasia often occur together (Duffy, 1995). However, the two disorders have been shown to be distinguishable, since AOS has been documented in non-aphasic patients (Square-Storer *et al*., 1990; Square *et al*., 1997), who do not manifest truly linguistic deficits, such as agrammatism and naming deficits.

AOS is often confused with conduction aphasia, perhaps because sound level errors (substitutions, additions, transpositions or omissions) are prominent in both disorders. However, the nature of errors is thought to be different (McNeil *et al*., 2004; Duffy, 1995). The sound errors in conduction aphasia reflect an underlying deficit in the selection of the phonemes for speech, that is, a language deficit. Apraxic speakers, on the other hand, are believed to select the correct phonemes, only to have trouble with their motor execution. Wertz has suggested that patients with conduction aphasia typically speak with near normal prosody, whereas halting, effortful speech with abnormal prosody is considered a hallmark of AOS (Wertz *et al*., 1984). Patients with conduction aphasia may lack awareness of their speech errors and therefore may not always make attempts at self-correction, while the opposite is true in cases of AOS (Square, 1997). Despite this, the differential diagnosis of the speech production errors in AOS and conduction aphasia can be difficult given the similarity in sound level errors.

AOS differs from dysarthria in that dysarthria is caused by impairment of muscle strength, tone, range of motion and/or coordination, while AOS is not caused by these impairments (Darley *et al*., 1975). Dysarthria can affect phonation, resonance, articulation or prosody as the result of damage to the central or peripheral nervous system (Darley *et al*., 1975). In AOS, however, articulation is primarily disrupted, rather than resonance or phonation, due to central nervous system damage. Also, the errors heard in dysarthric speech are typically consistent and predictable, while the speech errors heard in AOS tend to be highly irregular (Darley *et al*., 1975; Yorkston *et al*., 1988; Duffy, 1995). Speakers with AOS may misarticulate a word on one occasion, and accurately articulate the same word on another occasion.

Whether there is a singular speech symptom that is exclusive to AOS remains debatable. Many of the typical speech errors heard in conduction aphasia, such as the transposing of sounds or perseverative errors, can also be heard in cases of AOS. McNeil has suggested that there are three characteristics of pure AOS that do not occur in any other sound-level production disorder (such as conduction aphasia): sound distortions, prolonged segment durations (e.g., prolonged vowels or consonants) and prolonged intersegment durations (e.g., abnormal pauses within sounds, syllables or words) (McNeil *et al*., 2004).

Neuroanatomy of Apraxia of Speech

Pinpointing a singular brain region associated with AOS has been controversial. The disorder has been described in patients with lesions to Broca's area (Alexander *et al*., 1989; Hillis *et al*., 2004), left frontal and temporoparietal cortex (Square, 1997; McNeil *et al*., 2000), the left, superior, anterior region of the insula (Dronkers, 1996), as well as left subcortical structures, particularly within the basal ganglia (Duffy, 1995; Square *et al*., 2001; Peach and Tonkovich, 2004).

Dronkers compared 25 left hemisphere stroke patients with chronic AOS to 19 patients without AOS and found that all patients with AOS shared a common site of lesion within the precentral gyrus of the left anterior insula (Dronkers, 1996). None of the 19 patients with an infarction of the left MCA without AOS had lesions in this same region. This disassociation

provided strong evidence that lesions to the anterior insula area may result in AOS.

Other studies have argued against a relationship between the insula and AOS. In a study with 80 acute stroke patients, Hillis used diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) in acute patients within the first 24 hours of stroke and found no association between AOS and metabolism in the left insula (Hillis *et al*., 2004). AOS was instead associated with structural damage or low blood flow in the left posterior inferior frontal gyrus. Variations in stage of illness and techniques used may account for these differences.

Accounts of AOS in patients with subcortical damage have also been reported. Kertesz reported 10 cases of patients with AOS and aphasia who had lesions in the basal ganglia and internal capsule (Kertesz, 1984). Closer inspection of CT scans provided with these cases reveals insular involvement as well. Peach and Tonkovich recently described the phonemic characteristics of AOS in a patient with subcortical damage as the result of a hemorrhage (Peach and Tonkovich, 2004).

Functional neuroimaging studies in normal subjects have generally involved similar brain regions as patient studies. However, differences in experimental design and tasks used, that is, overt versus covert speech, cause difficulty in understanding the different contribution of these regions. Several studies have shown left insular recruitment for different speech production tasks, when the task was overt (Kuriki *et al*., 1999; Wise *et al*., 1999). Other studies have reported activation in the frontal operculum as well as the premotor and primary motor cortices for covert articulation (Rueckert *et al*., 1994). Basal ganglia activation has also been described for a covert repetition task of a single syllable (Wildgruber *et al*., 2001).

Though there may be some disagreement as to precise location, accounts of AOS in neurodegenerative cases have demonstrated that patients with progressive nonfluent aphasia and AOS showed focal atrophy in all three of the regions mentioned above, including the inferior frontal gyrus, left insula and subcortical regions (Gorno-Tempini *et al*., 2004).

There have been suggestions that there may be more than one type of AOS—one caused by frontal lobe damage and another, the result of temporo-parietal lesions (Square *et al*., 1997). Using acoustic analysis and neuroimaging data Square-Storer and Apeldoorn (1991) identified a left parietal variant of AOS that differed in presentation from AOS caused by a left frontal lesion. Speech symptoms associated with parietal AOS included: visual and auditory groping on initiation and within utterances, numerous off-target approximations of phonemes and occasional syllable segregation (Square *et al*., 1997). A study by Deutsch of 18 frontal and temporo-parietal patients, all of whom were diagnosed as having AOS, found that the temporo-parietal patients produced a greater percentage of polysyllabic sequencing errors and a smaller percentage of errors on monosyllabic articulation errors than the frontal group (Deutsch, 1984).

Differing results in localization studies of AOS might arise from the fact that diagnostic criteria, localization methods, time post-onset and etiology have varied among studies, making it difficult to draw definitive conclusions about the neurological basis of AOS (Duffy, 1995).

Apraxia of Speech in Current Models of Motor Programming

The cognitive basis of AOS remains a theoretical question. While the dysarthrias are thought to be caused by a deficit at the end-stage execution of articulation, modern motor programming models consider AOS to be impairment at a more intermediate level. A number of models have been put forth that have provided a framework for understanding the complex process that is spoken language. A few early models address phonological encoding in particular (Shattack-Hufnagel, 1979; Shattuck-Hufnagel, 1979a; Garrett, 1980; Garrett, 1984; Shattack-Hufnagel, 1987), while others are more comprehensive, describing speech production from early semantic encoding to end-stage phonological levels of production (Dell, 1988; Levelt, 1989; Levelt *et al*., 1999).

Levelt's influential model of speech production describes a phonological output buffer, which is conceived of as a short-term store for sequences of phonemes to be articulated (Levelt *et al*., 1999). Patients with damage to this buffer have been reported previously (Caramazza *et al*., 1986) and share some speech characteristics of AOS (predominance of phonemic paraphasias and pauses between words, reduced articulatory rate, etc.). However, it is difficult to conclude whether these patients presented with AOS. For a more complete review of modern speech production models see McNeil (McNeil *et al*., 2004) and Ziegler (Ziegler, 2002).

Darley initially put forth a three-stage model of motor speech programming that involves a central language processor (CLP), a motor speech programmer (MSP) and the motor speech cortex. In this model, the CLP selects meaningful sequences of phonemes for speech and then converts these into neural codes that drive the MSP, which in turn activates the appropriate speech musculature (Darley *et al*., 1975). Based on this model, Darley assumed AOS to be a deficit in speech programming, at the level of the MSP.

Recently, van der Merwe conceived of a model that expanded on the planning and programming stages originally described by Darley (van der Merwe, 1997). van der Merwe's model proceeds in four stages. Initially, basic linguistic units or phonemes are selected. During a second motor planning phase these phonemes are organized into temporospatial codes for speech production. In the third, motor programming phase, muscle-specific motor programs are selected and sequenced before moving forward to the fourth phase, when these sequences are carried out by the speech musculature (van der Merwe, 1997). For Darley, AOS is caused by a disruption at the programming stage, whereas in van der Merwe's model, AOS would result from disruption to the earlier planning stage, between the level of Darley's CLP and MSP (Peach, 2004).

Treatment for Apraxia of Speech

Most available treatments for AOS have limited data to support their efficacy (Wambaugh and Doyle, 1994). Over the last few decades, a variety of treatment approaches have been studied, with no one approach proving to be effective for all patients (Wambaugh, 2002). Given that AOS is generally believed to primarily disturb articulation and prosody, many programs have focused on remediating these specific deficits (Wambaugh, 2002).

Therapy goals are typically designed to improve communicative effectiveness. For the mildly apraxic patients, poor prosody may be the primary speech deficit and, therefore, goals designed to improve intonation and stress may be the most appropriate (Square *et al*., 1997). For the moderately or severely apraxic patient, therapy might focus on relearning oral postures for individual speech sounds (Square *et al*., 1997).

General techniques that have been employed include: traditional articulation therapy (repetitive exercises involving imitation of speech sounds and words) (Rosenbek *et al*., 1973), finger tapping or pacing (using a metronome) (Dworkin *et al*., 1988), singing and electromagnetic (EMG) feedback to reduce tension (McNeil *et al*., 1976; Wambaugh, 2002). Alternative or augmentative communication devices have also been prescribed for patients with severe AOS, as well as the use of compensatory strategies to replace speech (e.g., gesturing, writing, drawing, communication books, etc.) (Wambaugh, 2002).

In a recent summary of treatments for apraxia of speech, Wambaugh noted two particular techniques that have replication data to support initial treatment findings (Wambaugh, 2002). One such approach, PROMPT, developed by Square and colleagues is designed to help patients use rate and rhythm control strategies (Square *et al*., 1985; Square-Storer and Hayden, 1989) to improve their speech. The second program, developed by Wambaugh and collegues, focuses on the remediation of misarticulated consonants through modeling, repetition of minimally contrastive words, graphic cues and phonetic placement cueing (Wambaugh *et al*., 1998).

Despite the fact that there are no large, randomized trials of efficacy of treatment for AOS, single subject studies using rigorous experimental designs have demonstrated the effectiveness of some of the treatments for AOS in individual cases mentioned above. The effectiveness of these treatments has been replicated.

Conclusion

Within the literature on AOS, the site (or sites) of lesion, nature of defining speech characteristics and most effective treatment strategies remain somewhat elusive. Though AOS is now a widely accepted term, there is still debate as to which symptoms are most pathognomonic of this motor speech disorder, as many of the behaviors in AOS are also common symptoms in other speech and language disorders. The development of clear and uniform diagnostic criteria will help clinicians and theorists better describe AOS in relation to its near clinical neighbors.

Consistent diagnostic criteria may also help address the question of whether there are variants of AOS, one associated with frontal damage and another with temporoparietal lesions. These variants might reflect disruption at different stages of the complex process of speech articulation and might, as a result, require different types of intervention. Using modern acoustic, physiologic and neuroimaging techniques may help refine our understanding of motor speech disorders in general and the neuroanatomical and cognitive bases of AOS in particular.

Despite a number of enduring questions, we now understand AOS to be a unique speech disorder that is distinct from other speech and language deficits such as dysarthria, aphasia or stuttering. Because of its importance as an early clinical indicator of progressive neurologic disease, AOS will continue to be a topic of great interest to researchers and clinicians in the coming years.

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